EM Resident
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Defeating de Winter

Managing STEMI Equivalents

Pediatric Airway

Kratom Concerns

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TOGETHER, WE HEAL

Dear Intern...

PHOTO BY RENEE FERNANDES, WITH PERMISSION FROM PARKLAND HEALTH & HOSPITAL SYSTEM

Joan Noelker, MD, MACM
Washington University in St. Louis

Dear Intern,

Today you looked at me with a face that begged the question, "Am I terrible?" Yes intern, yes — you are.

Intern, you have been a doctor for only a few hours. You expect to confidently manage multiple patients and only struggle slightly with our unknown hospital system, not to mention our new EMR, and all the ED staff whom you have just met, and all the consultants you've only spoken to on the phone, whom you have also never met. You expect that you can do all of these things for your entire shift without being too terrible. **This is not a realistic expectation.** You cannot do these things yet. You may not believe me, but this is normal.

You are trying very hard, dear intern. You are also very smart. The fact that you are struggling right now does not mean your imposter syndrome was correct, that in fact you are stupid, and have been stupid for all of these years, through high school, college, medical school, and whatever other degrees you probably also possess. You are not a stupid person, but that does not mean you might not be failing right now, and that you will not continue to fail for an indeterminate period of time, and that even when you succeed, that you might not fail again in the future.

Dear intern, when you work in a new system, when you have to document in a new EMR that seems more focused on making you write notes in 80 different locations while also putting in orders that disappear into the ether, **it is normal to struggle**. It is normal to suddenly become worse at the things you used to be good at, like taking histories, and interacting with patients, and not accidentally cursing in the middle of the care area when you get startled. We *all* get worse before we get better.

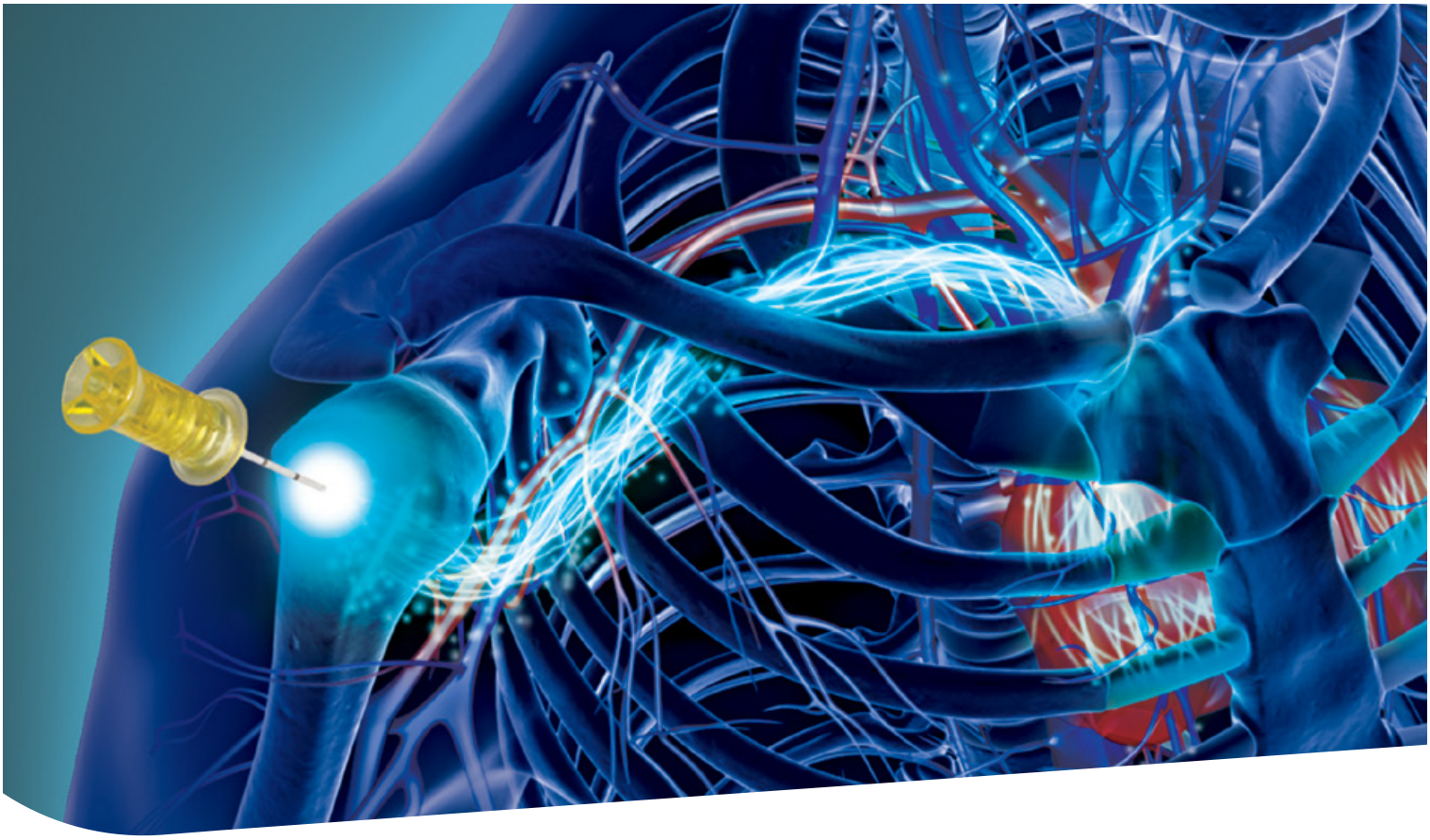
Dear intern, if you feel like you are a liar when you tell patients you are Dr. So-and-so, don't worry; you're not lying, even though you certainly do not feel like a doctor for these first few days, or weeks, or even months. It seems like a doctor should know quite a bit more than you know, that they should be confident, ooze knowledge and intelligence, and understand how to get lidocaine out of a bottle without exploding it in their face, thus filling their eyeballs with lidocaine and having numb eyeballs for an entire shift (*not to say that ever happened, of course*). And I certainly have never dropped, at least once, every single piece of a central line kit on the floor, or accidentally shot the wire across the room when I was trying to look smooth by removing the wire cap with a flick of my thumb. Of course I have never done these things, dear intern, because probably I was never an intern. Probably I magically achieved my attending-level of knowledge and confidence and skill through no hard work of any sort but through wizardry; really, I did not need residency at all.

But you must know this cannot be true. You may have detected sarcasm, dear intern, if you looked hard with your tired, lidocaine-filled eyeballs. We have all struggled. We are all smart people who have occasionally looked stupid. We have *all* done things that we think prove we don't know anything, and that we are unfixable, or unteachable, or that we will never actually be good doctors. It is normal to feel like this. But do not worry, this is why residency exists: so that you can continue to be terrible while we help you and while your patients teach you so much more than we ever could. Because one day, many months from now, you will realize you are in fact not terrible, that all of your hard work is paying off, that **you actually know what to do for your patient** with chest pain, and how to explain the CT results to your patient with new cancer, all without having to check with me.

And then one day, even farther along than that, you will not need me at all. You will do all of these things by yourself. And while you are growing, and while you are failing, and while you are slowly learning to not be terrible, I will be learning with you. You will not know it, but you will actually be teaching me many more things than I will be teaching you.

And so, dear intern, to answer your question: are you terrible? Maybe a little bit, but you are certainly less terrible than I was in July of *my* intern year, and if you keep working hard, and if I also keep working hard, I have no doubt you will one day be a much better doctor than I.

Yours with love,
Dr. Attending ✶



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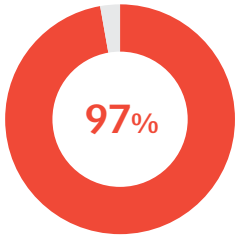

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"Remember That Patient..." and Other Feedback

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During residency you refine the habits that will underpin the quality of your care, job satisfaction, and career progression. Consider some advice based on 36 years of emergency medicine practice in both academic centers and busy community hospitals. These pearls are excerpted from 9 years of end-of-shift feedback to residents under my supervision.

Caveat: These are solely my opinions. Your mileage may vary.
Editor's note: Please read online for extended advice.

DAY-TO-DAY RISK MANAGEMENT AND PATIENT SAFETY

- **"Remember that patient . . ."** Whenever you send an email or leave messages containing any variation on "remember that patient," **start with** something like *"nothing bad happened, but..."* or *"no problem, but..."* (if appropriate)
- **Take a conservative approach to evaluation and disposition**. "Worst case scenario" is our area of expertise, and pursuing that concern should be balanced only against the risks, costs, and complications *for the patient* of the tests and measures we employ to rule out "badness." It *is* important to think twice before ordering tests for which false positives (think D-dimer) have potentially harmful consequences in time, dollars, or risk from precipitating more invasive diagnostic measures.
- **Repeat vital signs and physical exams at discharge.** Take note and explain: "Ambulatory without symptoms at discharge, nontender, taking PO, no questions, return precautions discussed."
- **Examine every patient before discharge (and document it).** A succinct note provides a picture of the patient's course. It also effectively answers a common complaint from the dissatisfied, that they "never saw the doctor again, no one told me ..."
- **Beware your gut feelings.** I can be comfortable with trainee decisions based on "gut" when (tempered with a risk/benefit analysis) they lead to *more* conservative management. I am rarely comfortable relying on your "gut" or mine in doing less than the objective H&P dictates.
- **Check your biases.** Reigning in your "gut" goes double for the inevitable **unlikeable patients or those who return frequently to the ED.** Following one's visceral reaction is a frequent source of preventable error. Read the chart, and then ask, *with the benefit of hindsight*, what would someone else looking at this chart conclude I should have done?
- **Avoid minimizing.** Minimizing is common in settings (like the military) where the majority of patients are young and healthy or where lack of primary care access drives "minor" chief complaints to the ED.
- **Rely on evidence.** Review the clinical decision rules, algorithms, and protocols in use in your ED *every time* you wander into the clinical territory they cover. Failing to follow guidelines (or justify why not) can result in a prima facie negligence determination, regardless of the other facts. Even in the absence of bad outcomes, regulators (from department chairs to employers, hospital committees, and federal agencies) find that auditing compliance with such guidelines is an easy metric that can blow back on providers in many ways.
- **Exercise caution with "hand off" patients.** Turnover patients are a well-known patient safety issue.[1] When any turnover patient's course becomes at all complicated, do a complete H&P and test review, forming your own impression and plan. Document carefully; inadequate handoffs were a factor in 24% of closed ED malpractice claims.[2]
- **Don't just "move the meat."** Few experienced docs can see above-average numbers of patients per hour safely. Sometimes you must do so, but only AFTER your clinical skills have matured.

MAXIMIZING YOUR EDUCATION

- **Question everything.** Before your opinions and practice patterns become "locked in" to what someone told you in med school or residency, adopt the habit of validating/challenging/questioning.
- **Look stuff up.** Similarly, you need a plan for when you are given conflicting information from staff and consultants. When advice differs, you have the right (and obligation) to ask for the evidence behind the suggestions. Then do your own research.

YOUR PRACTICE PATTERN: CARING FOR THE INDIVIDUAL PATIENT

- **Avoid cognitive biases.** **Anchoring** is the tendency to rely too heavily on information obtained early in the diagnostic process, with subsequent failures to adjust as new information becomes available. Accepting the patient's explanation for their symptoms is one form of anchoring. Start with a non-directive history ("describe your symptoms"), look at the objective data, and consider the full differential before acting.
- **Confirmation bias in history-taking:** Emergency physicians structure history-taking to elicit answers that point toward (or away) from the serious diagnoses. Err on the side of caution. These and other cognitive biases represent some of the biggest threats to optimizing our diagnostic performance.[3]
- **Talk to prehospital providers and witnesses early.** Identify useful witnesses early and get their story. Don't let them leave without learning what they know.
- **Complete a physical examination.** Although we correctly emphasize the importance of history, one thing I've seen fall off during residency is performing an appropriately thorough and focused physical exam.
- **A "negative" neurological exam does not mean no need for CT.** Nearly all patients who qualify for a scan using any of the accepted clinical decision rules predicting statistically increased risk for an intracranial process will lack focal findings. As a corollary, no one should be considered to have a negative neuro exam until they've gone through a series of maneuvers while standing and walking.
- **Pelvic exams are important.** Think of the pelvic exam as just another part of your initial exam in the female patient with abdominal pain. While the value of the "routine" pelvic has been questioned in

pregnant patients with an IUP on ultrasound[4] or in young women with strong suspicion of sexually transmitted infections,[5] it remains an essential part of the evaluation of women with lower abdominal pain.

- **Test critically.**
  Work on narrowing your differential diagnosis by history and physical exam as opposed to testing. Think twice before ordering "basic" labs, which are costly, delay throughput, can lead to morbidity from chasing false positives, and frequently fail to improve decision-making. Test ordering is a measure of your understanding of the case at the time and a reason that "resource utilization" is scored on the oral board examination. Over a career, the costs of a reflexive habit of "routine" testing are considerable.
- **Don't forget stuff.**
  Develop a nuts-and-bolts *process* of providing care. We all need a system to "close the loop" on tests and treatments either ordered or forgotten. Slowing down is often not an option, so having a system you apply EVERY TIME will save you (and your patients) delay and risk.
- **Do a slit lamp examination.**
  Don't bring me an eye problem without having done a slit lamp exam.[6-7] The Wood's lamp is not a tool for EM physicians who aspire to board certification.
- **Ask for help.**
  This can be a "critical action" on the oral boards and is a step in a number of algorithms. Medicolegally it can be considered indefensible if help was available but you didn't call. It would also feel really bad.
- **When you're stuck for a diagnosis and/or disposition:**
  1. *Repeat the H&P* and review your differential — go back to the beginning.
  2. Satisfy yourself that you have eliminated a need for intervention, further testing, or admission.
  3. If the patient is too sick or uncomfortable to go home, or if significant life-threats remain on your differential, refer for admission or observation.
  4. If stable for discharge: Explain to your patient what you have done, and the diagnoses you have considered and have or have not eliminated. Ask: "Do you feel well enough to go home?" If not, what will it take to get them that way? (See #3)
- **Weigh risk, pretest probability, and admission vs. discharge.**
  Our job is "worst case scenario." We are looking for the rare but life-threatening event with a common presenting complaint. When studied, emergency physicians commonly consider admission for patients whose risk for a serious outcome is in the 1-2% range.[8] Use Bayes theorem: *Finding* uncommon serious conditions *first* requires a high index of suspicion and *sensitive* tests.
- **Call consultants.**
  Consultants are here to teach us and help care for our (often also their) patients. At 2 am, we are our patient's best and only advocate. "Call your doctor in the morning" is easy, but not always the best option. It is our equivalent of "if it's not better, go to the ER."
- **Arrange follow-up care.**
  Many of our patients lack access to quality ambulatory care. On occasion, we should take responsibility for (and do the work of) closing the follow-up loop. By extending the ED safety net, such a practice can improve outcomes and reduce return visits for a vulnerable subset of our discharged patients. If you know what the patient needs, try to help them get it.
- **Write thorough discharge instructions.**
  Discharge instructions demonstrate that we have considered potential complications, done what we can to detect or prevent them, and advised patients of what to look for and when to return. EHR-generated generic instruction sheets do not relieve us of this responsibility.

SHIFT MANAGEMENT: AS EMPLOYEE, CO-WORKER, MANAGER

- **Stay ahead of the interns/ students.**
  A student H&P can take a while – during which time patients, frequently in pain and potentially unstable, are being "seen" but not treated. There is no downside to getting in ahead of the intern/student.
- **Manage the department.**
  Leading requires a combination of situational awareness and assuming responsibility. Learn and follow the environmental cues that should drive you to the bedside: staff members hurrying, raised voices, more than one nurse at the bedside, any question brought to you about a patient you haven't seen, etc. Look at a crowded board and ask yourself: "What could I do now if I were solely responsible for this ED?"
- **Come in early.**
  Habits you form in training will influence your practice long-term. In any setting it's always appreciated when someone comes in a little early for their shift.
- **Ignore co-worker behavior.**
  One way I've successfully practiced for 36+ years without burnout is by studiously avoiding comparing myself to coworkers. I strongly recommend that you focus on caring for your patients. Optimize your efficiency, and limit the ability of any third party to critique your practice. ✶

Dr. Frumkin currently serves as volunteer faculty, Emergency Medicine Residency, Naval Medical Center, Portsmouth, Virginia.

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

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1975 Residents joined this new organization for $15. By the end of the decade, EMRA boasted 269 resident and 14 medical student members.

1982 EMRA officers manned a booth at Scientific Assembly that featured a bulletin board with job opportunities.

1988 Hurricane Gilbert hit Jamaica. EMRA members secured and transported medical supplies to Montego Bay.

1994 EMRA hosted the first Medical Student Forum. Membership soared to 2,581.

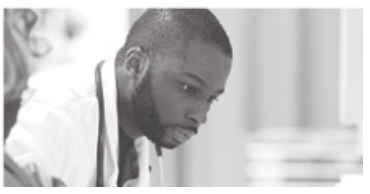
2000 Membership explodes to 4,320. By the end of the decade, another 2,145 were added to the EMRA membership roster.

2005 Hurricane Katrina strikes the U.S. Gulf Coast. EMRA collected textbooks for residents and medical students to replace those lost at LSU and Tulane University.

2013 EMRA debuted the landmark documentary, *24/7/365 – The Evolution of Emergency Medicine*. The film tells the story of the specialty's maverick founders.

2016-PRESENT Multiple on-shift guides and publications are published including EMRA's *EM Fundamentals*, *EKG Guide*, and *EMRA and ACMT Medical Toxicology Guide*. Membership exceeds 15,000 and EMRA now funds 111 leadership positions.





STEMI Equivalents

Can't-Miss Patterns

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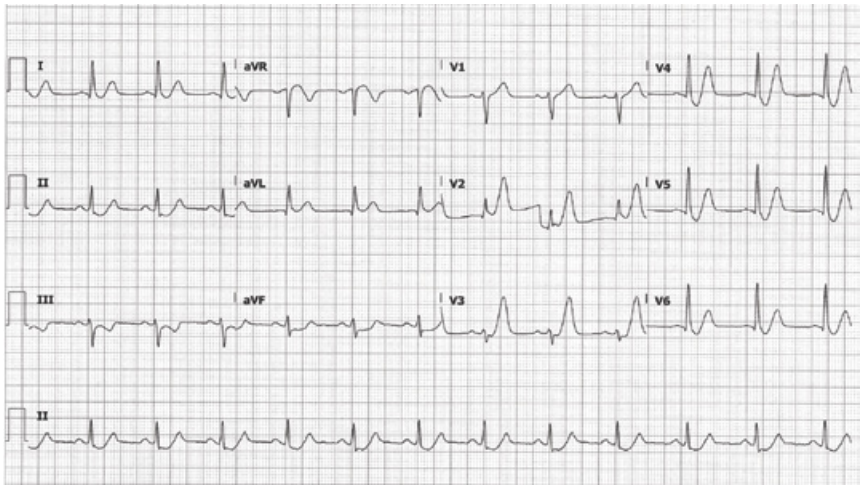
Case Introduction

A 50-year-old male with a past medical history of hyperlipidemia presented to the ED after an out of hospital cardiac arrest. 911 was activated after the patient had a syncopal episode while riding his bicycle. EMS reports that the patient was diaphoretic and reported substernal chest pain and nausea.

En route to the ED, the patient lost pulses, and ROSC was obtained after 1 round of CPR and a single defibrillation. Upon presentation to the ED, the patient was alert and oriented with ongoing chest pain. Initial vitals were stable. Initial ECG is shown at right.

What would be your next step in management?



STEMI Criteria

Acute Coronary Syndrome is "bread and butter" emergency medicine, and emergency physicians must excel at early recognition of cardiac ischemia, as time is myocardium. Recognizing ischemic patterns on ECG is essential for identifying coronary occlusion early and facilitating emergent catheterization. STEMIs represent myocardial ischemia caused by complete occlusion of an epicardial coronary artery, often because of atherosclerotic plaque rupture and subsequent thrombosis formation. STEMI criteria have been developed to identify the patient population that benefits from emergent catheterization and revascularization.[1,2] ACCF/AHA guidelines recommend first medical contact to PCI device time (commonly referred to as door to balloon time) of less than 90 minutes. If time to PCI will exceed 120 minutes, fibrinolytic therapy is recommended with 30 minutes of hospital arrival in the absence of contraindications.[2]

Per the 2018 AHA "Fourth Universal Definition of Myocardial Infarction," ECG manifestations suggestive of MI (in the absence of LVH and BBB) include:

- ≥ 2.5 mm STE in V2-V3 for males < 40 years*
- ≥ 2 mm STE in V2- V3 for males ≥ 40 years*
- ≥ 1.5 mm STE in V2-V3 for females regardless of age*
- ≥ 1 mm STE all other leads

*New J-point elevation ≥ 1 mm from prior ECG should be considered ischemic

The J-point is defined as the junction between the QRS termination and the ST-segment onset, and the ST-segment should be measured against the isoelectric TP segment (assuming a stable baseline).[1]

STEMI Equivalents

STEMI equivalents represent coronary occlusion without meeting the traditional STE criteria and are equally important to recognize in a timely fashion. Emergency physicians must know to involve interventional cardiologists for patients with dynamic ECG changes, persistent ischemic chest pain, hemodynamic instability, and STEMI equivalent patterns that require emergent PCI to minimize morbidity and mortality.

De Winter T-waves

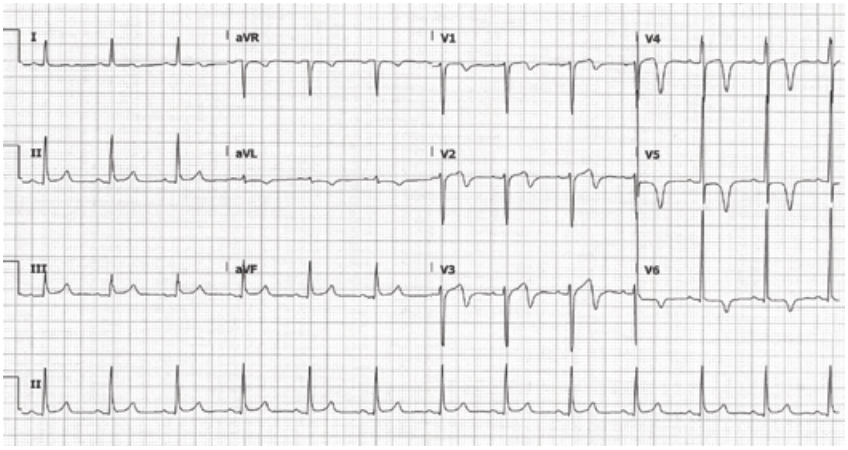
This ECG pattern was described in 2008 by de Winter RJ, et al. in 2008 with a case series that described this pattern occurring in approximately 2% of LAD occlusions.[3] This pattern, as seen on ECG #1, demonstrates > 1 mm of upsloping STD and tall symmetric T-waves, most commonly in the precordial leads. There may be 0.5-1 mm STE in aVR, but there should be no STE in the precordial leads. Patients with this ECG pattern and a presentation concerning for ACS warrant immediate revascularization.[4]

Suggested occlusion: LAD

Wellen's Syndrome

In 1979 Gerson MC, et al. described exercise-induced U-wave inversions during exercise that were highly predictive of proximal LAD disease.[5] The term Wellen's syndrome is used describe these ECG changes, which represent a reperfusion pattern in the setting of severe proximal LAD stenosis, when seen in an asymptomatic patient who was recently symptomatic. There are 2 patterns seen with Wellen's syndrome: Type A has biphasic T-waves, and Type B has deep, symmetric T-wave inversions. Diagnosis requires preserved R-wave progression, no precordial Q-waves, minimal STE, and the characteristic T-waves in the precordial leads (typically V2-V3). Treatment is coronary catheterization; stress testing is contraindicated because of the high risk of precipitating infarction.[6]

Suggested occlusion: LAD

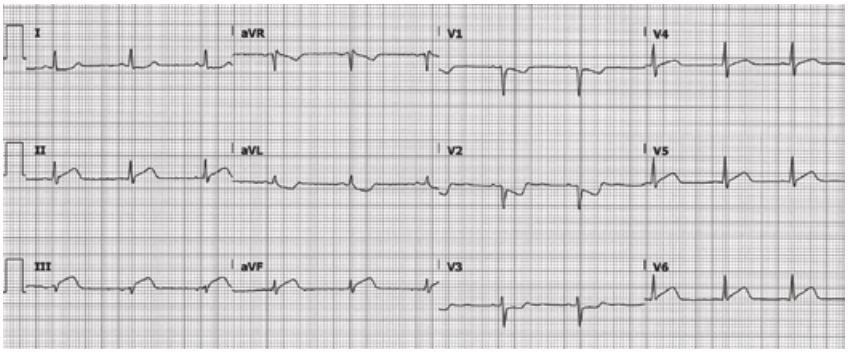


Biphasic T waves in anterior precordial leads suggesting Wellen's Syndrome

Posterior MI

Posterior MIs are easily missed because of the absence of any STE. Posterior involvement is estimated to occur in 15-21% of all acute myocardial infarctions[8] and in isolation ~3% of the time, typically due to occlusion of the left circumflex or right coronary arteries. Since there are no leads that look directly at the posterior wall, the associated ECG changes are reciprocal changes seen in the anterior leads V1-V3.[7] Typical ECG changes include STD (reciprocal STE), tall R-waves (reciprocal Q-waves), and prominent positive T-waves (reciprocal terminal T-wave inversions). A posterior ECG should be obtained, and STE ≥ 0.5 mm (≥ 1 mm in men < 40 years) in V7, V8, or V9 is diagnostic of a posterior MI. Note that the absence of elevations in the posterior leads does not exclude a posterior MI.

Suggested occlusion: LCx or RCA

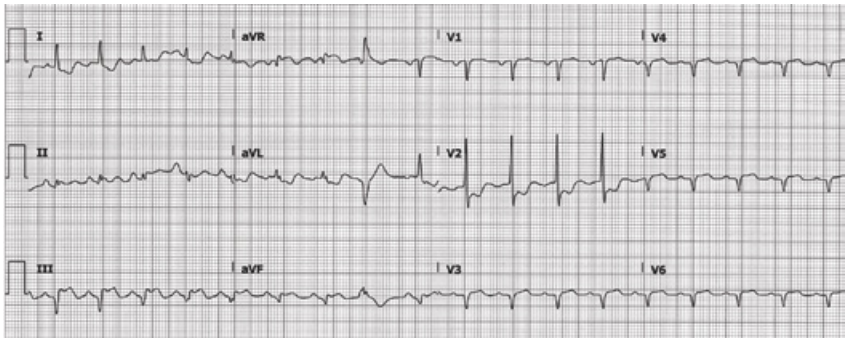


Inferior MI with STD in V1-V3 suggesting concurrent posterior MI

Right Ventricular MI

While isolated RV MIs are uncommon, ~1/3 of inferior MIs have RV involvement.[7] The importance of recognizing RV involvement is that these patients are preload dependent. Treatment is often IV fluids and inotropes. Medications that decrease preload (eg, nitroglycerin, beta blockers, morphine) can precipitate hypotension and should be avoided or used judiciously. RV MI should be suspected when there is concurrent STE in V1 (which can be masked by a concurrent posterior MI) but should be considered in any inferior MI. A right-sided ECG should be obtained, and STE ≥ 0.5 mm (≥ 1 mm in men < 30 years) in V3R or V4R is diagnostic of a RV infarction.[1] Note that the absence of elevations in the right-sided leads does not exclude a RV MI.

Suggested occlusion: RCA



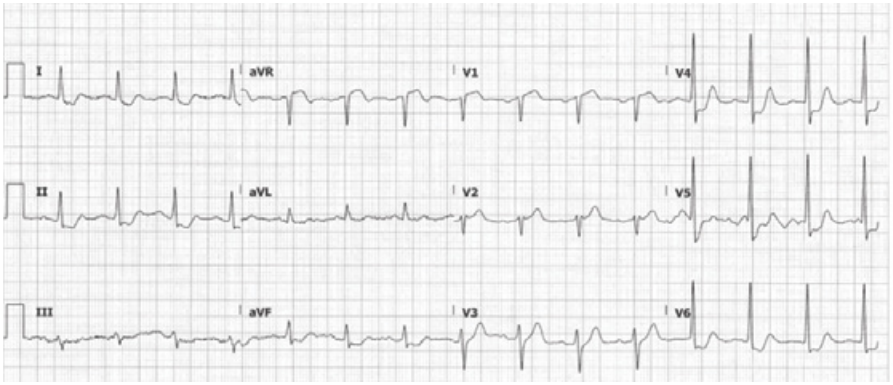
Inferior MI with STE in the right-sided leads (V3-V6 = V3R-V6R) indicating RV involvement

STE aVR with diffuse STD

STE ≥ 1 mm in aVR or V1 with STD ≥ 1 mm in ≥ 6 leads can suggest left main coronary artery insufficiency, proximal LAD insufficiency, or triple vessel disease, especially if accompanied by pathologic Q-waves, hemodynamic compromise, and/or refractory symptoms. STD are most prominent in the

inferior and lateral leads and thought to represent subendocardial ischemia. This ECG pattern is not specific to LMCA/proximal LAD insufficiency and can be seen in other conditions (eg, pulmonary embolism, aortic dissection, LVH with strain pattern). The 2013 ACCF/AHA Guidelines for the Management of STEMI recommend invasive angiography for patients with this ECG pattern and a presentation concerning for ACS.

Suggested insufficiency: LMCA, proximal LAD, triple vessel



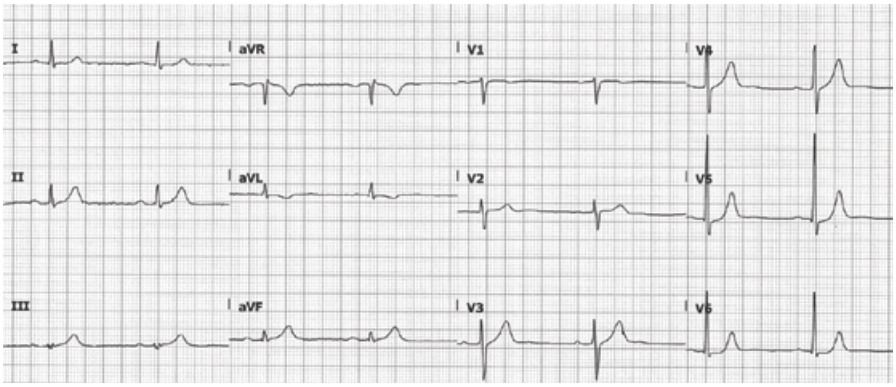
STE in aVR and V1 with diffuse STD suggesting LMCA/proximal LAD insufficiency or triple vessel disease

TWI in aVL

The isolated TWI in aVL is associated with both impending inferior wall MI and mid-LAD lesions.

Birnbaum Y et al. described reciprocal changes in aVL occurring in most patients with inferior wall MIs, and in some cases it was the only early indicator of an impending MI.[13] Hassen et al. describes TWI in aVL as also predictive of a mid-LAD lesion.[14] It is important to look for reciprocal changes when evaluating ECGs for signs of ischemia and to have a low threshold for obtaining serial ECGs, especially if there are non-diagnostic but concerning findings.

Suggested occlusion: RCA, LCx, LAD



TWI in aVL and hyperacute T-waves in the inferior leads seen in an early inferior MI

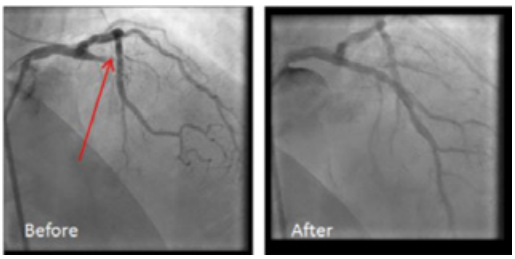
Hyperacute T-waves

Hyperacute T-waves in ≥ 2 contiguous leads may be the first signs of a developing infarct, often preceding any STE.[1] Hyperacute T-waves appear broad based, often asymmetric with a more gradual upstroke than downstroke, and tall relative to the associated QRS complex. An upright T-wave in V1 > V6, described as loss of precordial T-wave balance, is considered hyperacute and concerning for ischemia especially if this is a new finding.[15] A new upright T-wave in lead V1 in the absence of a LBBB or LVH is abnormal and may represent ischemia. Manno et al. described an upright T-wave in V1 as often associated with left circumflex disease.[16] These findings warrant close observation, serial ECGs, and ruling out hyperkalemia as the cause of the ECG changes.

Suggested occlusion: Left circumflex

Case Conclusion

The patient's ECG was notable for diffuse STD and tall prominent T-waves in the anterior precordial leads, concerning for De Winter T-waves. The patient was placed on telemetry, defibrillator pads applied, IV access obtained, and aspirin was administered. Interventional cardiology was called stat and immediately took the patient to the catheterization lab. Coronary angiography revealed a 100% occlusion of the proximal LAD, which was successfully stented (see figures 1 and 2). The patient was discharged 2 days later on appropriate medical therapy.



FIGURES 1 AND 2. Pre- and post-stenting angiography

TAKE-HOME POINTS

- It is important to remember that an ECG is not 100% sensitive for coronary occlusion and that the absence of STE does not rule out an MI. This case is an excellent example of a patient requiring PCI even though his ECG didn't meet STEMI criteria.
- Always remember that you are treating the patient in front of you (not their ECG), serial ECGs are your friend, and the 2014 AHA/ACC Guidelines for Management of Patients with Non-ST-Elevation ACS recommend early (within 2 hours) invasive strategies for patients with hemodynamic instability, signs of heart failure, ventricular dysrhythmias, or angina refractory to medical management. ✶

AN ATYPICAL PRESENTATION
Cardiac Tamponade and the Value of Point-Of-Care Ultrasound

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End-stage renal disease is increasing in the United States. These patients demonstrate an increased risk for all-cause mortality, most commonly from cardiovascular etiologies.[1] Dyspnea is one of the most commonly reported symptoms by patients with chronic kidney disease (CKD). Observational studies have demonstrated that its prevalence in conservatively managed patients with ESRD is as high as 60%.[2,3] Pulmonary edema and volume overload are the most common causes of dyspnea in this population.[1] However, patients are also at risk for other causes of dyspnea, including acute coronary syndrome (ACS), unrecognized chronic lung disease, pulmonary hypertension, air embolism, hyperkalemia, anemia, pericarditis, and pericardial effusion.[1,2]

The incidence of pericardial disease in ESRD patients is < 20%, ranging from pericarditis to pericardial effusion to cardiac tamponade.[4] Pericardial effusion is usually associated with fluid overload in this population.[1] We present a case of cardiac tamponade in an ESRD patient, diagnosed with POCUS that prevented a potentially fatal outcome if the patient underwent hemodialysis.

Case

A 47-year-old male with a past medical history including CKD (recently started hemodialysis), coronary artery disease, and chronic obstructive pulmonary disease (normally on 2 L of oxygen) presented to the ED from hemodialysis with a 1-day history of moderately worsening shortness of breath. While at hemodialysis his oxygen saturations were 86-87% on 4 L of oxygen. The patient reported a cough and orthopnea over the past day, but he denied fever, chest pain, hemoptysis, and abdominal pain. He was hospitalized numerous times over the past few months and displayed poor compliance.

Upon arrival to the ED, his initial vital signs were temperature of 98.0 degrees Fahrenheit, heart rate of 121 bpm, respiratory rate of 24, blood pressure of 140/84, oxygen saturation of 89% on 4 L nasal cannula. The physical exam was notable for an ill-appearing male with labored respirations, conversational dyspnea, coarse bilateral inspiratory and expiratory wheezing, and diminished bibasilar breath sounds. He was tachycardic, had bilateral lower extremity swelling, and jugular venous distention. The patient was given bronchodilators, steroids, and his oxygen was increased to 5 L nasal cannula with improvement of oxygen saturation to 92%.

The initial plan consisted of an ECG, lab evaluation, portable chest radiograph, and computed tomography angiography (CTA) of chest. The ECG demonstrated sinus tachycardia. Laboratory evaluation demonstrated a white blood cell count of 18.9 K/mL, sodium of 140 mmol/L, potassium of 5.1 mmol/L, and creatinine of 5.5 mg/dL. His troponin was 0.03 ng/mL. A chest radiograph (Image 1) demonstrated diffuse bilateral interstitial and airspace disease most likely representing pulmonary edema with the possibility of a right-sided pneumonia. Cardiomegaly and pulmonary venous congestion were also demonstrated. Prior to ordering the CTA of chest, POCUS was performed.

POCUS demonstrated bilateral pulmonary edema and a moderate pericardial effusion with early tamponade physiology. The CTA of chest order was discontinued. Cardiology and nephrology were both consulted and felt the patient was too unstable to tolerate dialysis. The patient was admitted to the ICU, and thoracic surgery performed a pericardial window, removing 750 mL of serous fluid; findings were consistent with fibrinous uremic pericarditis. After stabilization, patient underwent dialysis and improved significantly.

Discussion

Cardiac tamponade is often thought of as a clinical diagnosis based on Beck's Triad: hypotension, dilated neck veins (JVD), muffled heart sounds.[5] However, these findings are only seen in a minority of patients, and none of the findings alone are highly specific or sensitive.[6] Although our patient had a moderate pericardial effusion and sonographic evidence of cardiac tamponade, he was normotensive. Studies have demonstrated that most patients with non-traumatic cardiac tamponade are normotensive (50%) or hypertensive (35%), with only 15% of patients being hypotensive.[7] The physical exam is subjective and can vary among providers.[8] Key physical exam findings, such as JVD and muffled heart sounds, may be variable among providers.

The workup of ECG, laboratory evaluation, and chest radiograph did not provide much usefulness in establishing this patient's diagnosis. In fact, the workup may have been misleading and committed the patient to an intervention that would have been harmful (hemodialysis). Sinus tachycardia is very common in cardiac tamponade, but is not nearly as specific as ECG findings of electrical alternans or low QRS voltage.[9,10] Chest radiographs are neither specific nor sensitive for cardiac tamponade. Findings of cardiomegaly, as in this patient, are non-specific for cardiac tamponade and can be attributed to other disease processes, such as congestive heart failure.[6,9] Furthermore, the chest radiograph in the case demonstrated findings consistent with pulmonary edema which could have easily, but incorrectly, led the provider to attribute the patient's

dyspnea to volume overload. Lastly, POCUS also prevented the patient from undergoing a CTA of chest. Although a CTA of chest can provide useful information for the diagnosis of cardiac tamponade, it would have exposed the patient to unnecessary radiation and delayed the appropriate therapy.[11]

POCUS has been proven useful in the ED for managing critically ill patients and is highly recommended for patients with suspected pericardial disease.[6,12] POCUS diagnosis of cardiac tamponade is suggested by:[13,14]

1. Diastolic chamber collapse
2. Increased respiratory variations of transmitral and transtricuspid doppler inflow velocities
3. Inferior vena cava (IVC) plethora

Diastolic collapse of the right heart is highly suggestive of cardiac tamponade.[7] Diastolic collapse of the right atrium is more specific and sensitive compared to diastolic collapse of right ventricle, especially if the right atrium is collapsed for greater than a third of the cardiac cycle.[15,16] Respiratory variation of flow velocities across mitral and tricuspid valves are indicative of cardiac tamponade. When referenced to expiration, patients with cardiac tamponade will typically have a flow of variation of > 30% and 60% across the mitral valve and tricuspid valve respectively.[17] Lastly, IVC plethora (defined as IVC dilatation plus < 50% IVC diameter reduction during inspiration) is highly sensitive, but not specific for cardiac tamponade.[18] The patient in our case had, right atrial collapse for > 50% of cardiac cycle, transmitral flow velocity variation of 30.4%, and IVC plethora.

This case demonstrates an ESRD patient with dyspnea secondary to a non-classic presentation of cardiac tamponade. Despite chest radiograph evidence suggesting volume overload and pulmonary edema, ESRD patients are a unique population that can have dyspnea for non-volume related complications. Without POCUS, this patient could have received suboptimal management by undergoing hemodialysis instead of receiving the more urgently needed pericardial window. Based on the broad pathology associated with ESRD, the authors will have an increased index of suspicion for pericardial disease in dyspneic patients.✶

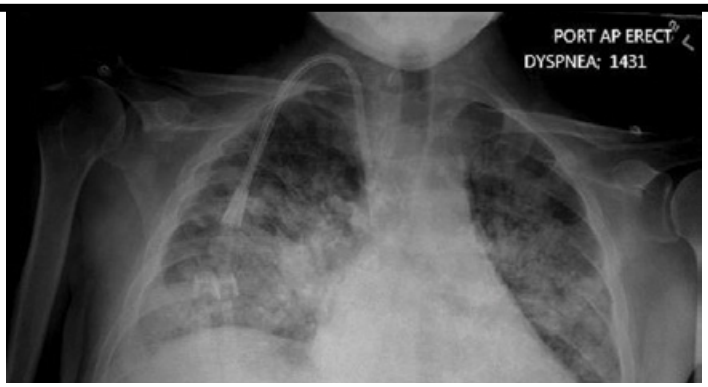

Image 1. Anterior-posterior chest radiograph demonstrating diffuse bilateral interstitial and airspace disease with cardiomegaly.

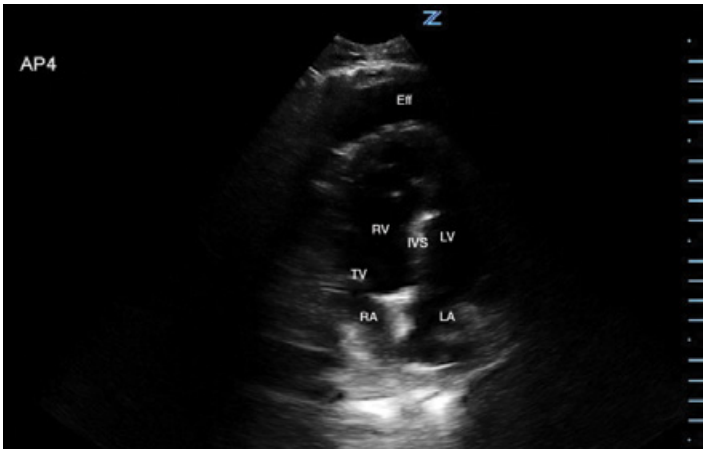

Image 2. Abnormal POCUS (apical 4 chamber) view in 2D-mode) demonstrating a large pericardial effusion with right atrial collapse during diastole. *Eff,* effusion; *RV*, right ventricle; *IVS*, intraventricular septum; *LV*, left ventricle; *TV*, tricuspid valve; *RA*, right atrium; *LA*, left atrium.

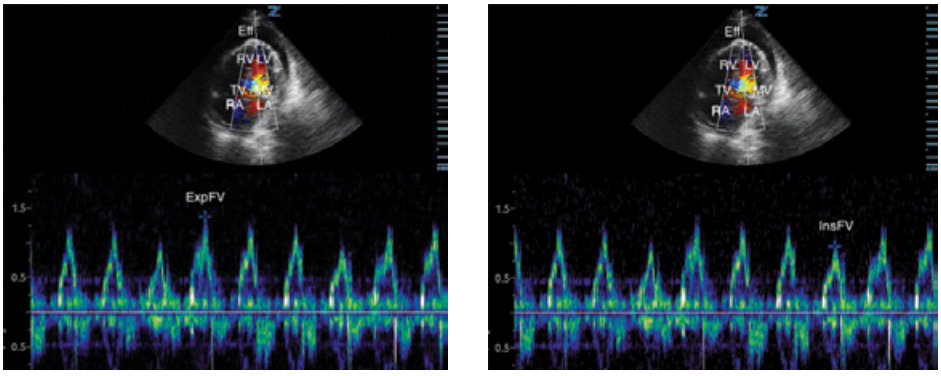

Image 3. Abnormal POCUS (apical 4 chamber) view in 2D-mode with color flow doppler demonstrating abnormal flow velocity across mitral valve during respiration (30.4%). *Eff*, effusion; *RV*, right ventricle; *LV*, left ventricle; *TV*, tricuspid valve; *MV*, mitral valve; *RA*, right atrium; *LA*, left atrium; *ExpFV*, expiratory flow velocity; *InsFV*, inspiratory flow velocity.

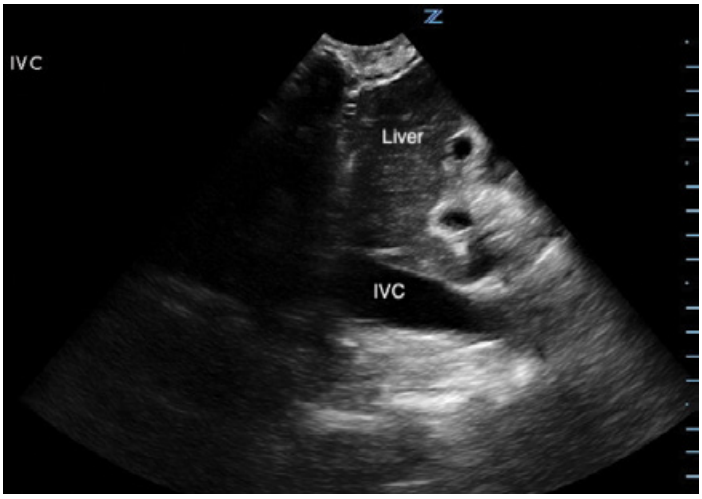

Image 4. Abnormal POCUS (longitudinal subcostal view in 2D-mode) demonstrating inferior vena cava plethora. *IVC*, inferior vena cava.

References available online

KRATOM
A Blurb on the Herb

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A 33-year-old male with history of anxiety, depression, and opioid use disorder arrives to the emergency department after witnessed seizure-like activity. He is post-ictal on arrival. Initial workup, including bloodwork and imaging, does not reveal any source of the seizure. As the patient returns to baseline mental status, he states that he has been self-treating his anxiety and opioid use disorder with kratom, a new herbal supplement he bought at a local shop.

What is Kratom?

Kratom derives from *Mitragyna speciosa*, a plant native to Southeast Asia. Historically it has been used in Thailand and Malaysia for its stimulant properties and purported medicinal benefits.[1] Recently, kratom has become popular in the United States for self-treating pain or opioid use disorder. Because of its availability as an herbal supplement, patients perceive kratom as a "safe" alternative to opioids.[2]

There are more than 25 active alkaloids in kratom. Mitragynine, the primary alkaloid, is less potent than morphine and accounts for about 66% of the total alkaloids in kratom. 7-hydroxymitragynine exists in small concentrations, but is about 46 times more potent than mitragynine.[1] Both mitragynine and 7-hydroxymitragynine act on mu-opioid receptors in the same way as heroin or oxycodone. Kratom additionally acts on many other receptors, including alpha-2 receptors, GABA receptors, serotonin receptors, and noradrenergic receptors.[2-3] The clinical implications of these interactions are poorly understood.

Why are People Using Kratom?

Kratom has been used throughout Southeast Asia for centuries as a stimulant for workers trying to combat fatigue associated with manual labor. It is also used medicinally to help with diarrhea, pain relief, morphine withdrawal, and a variety of other ailments.[1-4]

Kratom's recent popularity in the United States stems primarily from its use as an herbal supplement for self-treating pain or opioid use disorder.[4-5] Patients with opioid use disorder face significant barriers that prevent them from accessing evidence-based treatments. This lack of access has caused patients to seek alternatives to medication assisted treatment (methadone or buprenorphine). Kratom is perceived as a "safe" alternative. Not only do patients substitute kratom for their opioids, they also substitute kratom in for antidepressants as well.[2]

Kratom is readily accessible in local tea shops, smoke shops, gas stations, and on the Internet. Patients can either buy kratom leaves, capsules, or extracts to chew, smoke, or ingest.[1-2]

What are the Reported Effects of Kratom?

The effects of kratom are dose dependent, and people generally take a dose tailored to their desired response. White et al.[1] found that anecdotally, a low dose, roughly 1-5 g, is reported to produce a mild stimulant effect while a moderate dose, 5-15 g, will produce an opioid-like effect. Large doses, > 15 g, may be very sedating and pose risks similar to heroin or oxycodone overdose. They also report that, due to lack of a scientifically established dose-response curve or quality control measures on kratom packaging, accurate dosing of kratom is difficult and dosing errors are likely.

Kratom is also reported to impact pain, mood, and energy. A survey conducted in 2017 found that almost two-thirds of people stated they use kratom "to relieve negative moods or mental states, including anxiety, depression, and posttraumatic stress".[2]

The reported effects of kratom have also been described in the mainstream media. In March 2018, *Rolling Stone* published an article criticizing the United States Drug Enforcement Administration's decision to classify kratom as a schedule I drug and highlighting the anecdotal positive effects reported by people using kratom. The DEA has since removed the schedule I classification and instead classified it as a "drug of concern".[1, 4-5]

What are the Adverse and Toxic Effects of Kratom Ingestion?

As kratom use becomes more ubiquitous, a growing body of literature has called into question the safety of this herbal supplement. A recent study of kratom exposures reported to United States Poison Control Centers showed that nearly two-thirds of reported exposures spanning 2011-2017 occurred within the last year of the study window,

highlighting its growing popularity.[6] Reported adverse effects of kratom include nausea, vomiting, tachycardia, and hypertension. Some of the more serious reported toxicities include seizure, hallucinations, profound sedation, coma, respiratory depression, and neonatal abstinence syndrome.[1-2,4,6] Patients using kratom chronically may also experience symptoms of opioid withdrawal when they discontinue usage.

Mortality associated with kratom use has been a point of controversy. Until recently, proponents of kratom have advocated for its use as a safe alternative with minimal overdose risk. However, multiple publications have since reported a growing number of kratom associated fatalities.[6] These prompted the FDA to release a report in 2018 detailing 44 deaths associated with kratom use, one of which mitragynine was the only substance detected in post mortem analysis.[7] The FDA's report, coupled with growing concerns about kratom toxicities, prompted the Department of Health and Human Services to recommend that the DEA ban kratom use in the United States.

How Do We Manage the Toxic Effects of Kratom?

Toxic effects of kratom should be treated based on the patient's clinical presentation. Swogger et al.[2] recommend a symptom-based approach to managing these patients. If opioid-like effects (respiratory depression) are present, naloxone should be administered. Benzodiazepines are recommended for seizures, tachycardia, and hypertension associated with suspected kratom toxicity. Patients using kratom may also experience symptoms of opioid withdrawal. It is recommended to treat these symptoms similarly to any other type of opioid withdrawal, with evaluation for opioid use disorder, initiation of medication assisted therapy, and referral to treatment as appropriate.[8]

Why Does Kratom Matter?

Kratom is a widely available herbal supplement that has gained popularity among people looking to self-treat opioid use disorder or pain.[1,6] As the opioid epidemic continues to impact millions of people in the United states, it is inevitable that emergency physicians will encounter patients using kratom. It is important that we recognize these clinical effects and treatment strategies so we can appropriately treat and educate this patient population when they present to our EDs. There is a need for additional research on kratom to determine its safety as well as potential effectiveness.

KEY POINTS

- Kratom contains mitragynine, which acts on opioid receptors in the same manner as other prescription or illicit opioids.
- Kratom's popularity in the United States is on the rise, with patients using it as alternative treatment for pain, opioid use disorder, and mood disorders. Kratom-associated calls to poison control centers are on the rise, even as kratom-associated adverse effects and toxicities are being reported in EDs across the country.
- Kratom's effects vary widely, and use of this herbal product may lead to significant patient morbidity and mortality. ✶

References available online

Iliopsoas Rupture and Hematoma

A Rare Cause of Inability to Ambulate

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Spontaneous iliopsoas muscle or tendon injury is a rare cause of atraumatic hip pain. Given its mechanism, injuries tend to be found in athletes who suffer trauma during flexion activities. Nonetheless, spontaneous rupture with subsequent hematoma formation should be on the differential for acute, atraumatic hip or low back pain in the elderly, especially when the patient is unable to ambulate.

Case Report

A 91-year-old Caucasian female presented to the ED for right hip pain that started 7 hours prior to arrival. She stated she was attempting to put her right foot up onto a footstool when she felt a sudden pain in her right groin and hip. She had been previously able to walk on her own but has not been able to ambulate since the pain started. She had a catheter in place for chronic urinary retention, but denied any paresthesias in her groin, changes in her bowels, or pain radiating into her legs. She denied any recent traumas or falls. She described the pain as sharp, non-radiating, worse with attempting ambulation, and better at rest. She did not take anything at home for this pain. Her past medical history was significant for osteoarthritis, hypertension, hyperlipidemia, vocal cord dysfunction, and GERD.

Physical exam revealed a well-appearing elderly female in no acute distress. Her abdomen was soft. She had 5/5 strength with dorsiflexion and plantarflexion in the lower extremities bilaterally. Hip flexion was 2/5 in the right lower extremity and 5/5 in the left lower extremity. Sensation was intact throughout. Patellar reflexes 2/4 bilaterally. When the patient attempted ambulation, she was unable to bear weight on the right leg.

Plain films of the right hip and pelvis showed arthritic changes in all joints with some soft tissue swelling overlying the right hip. US duplex of the right leg showed no evidence of DVT. Basic labs, including CBC and BMP, were within normal limits. Non-contrast CT of the pelvis and lumbar spine showed a moderate sized right iliopsoas hematoma and a small amount of retroperitoneal bleeding, consistent with iliopsoas muscle rupture. Orthopedic surgery recommended admission and a type and screen and PT/INR, which was within normal limits.

After admission, the patient was evaluated for surgical intervention. With pain control, she was able to ambulate, so surgery was deferred. Hemoglobin dropped from 11.3 to 9.9 but stabilized at that point. The patient received physical and occupational therapy and was discharged 2 days later.

Discussion

Spontaneous iliopsoas rupture is an extremely rare cause of atraumatic hip pain seen in the elderly. Iliopsoas injury is usually seen in the setting of athletic trauma. The prevalence of atraumatic and traumatic iliopsoas tendon injury is 0.66%. The most common risk factors for atraumatic tendon injury are age, female sex, chronic steroid use, osteopenia, fluoroquinolone use, metastatic cancer, and chronic inflammatory diseases.[1] Risk factors for rupture and hematoma formation include uncontrolled hypertension, underlying bleeding disorder, or anticoagulant use.[2,3,4,5]

Iliopsoas tendon injuries can be complete or incomplete tears. Complete tears tend to be more painful and present with greater disability, depending on the patient's baseline functional status and concomitant injuries. Decision to admit mostly relies on ability to ambulate and pain control. It would not be unreasonable to discharge an ambulatory patient with pain medication and follow up to primary care for physical therapy or orthopedic surgery referral. Activity restrictions would include weight lifting and fast movements. Most patients will likely be best served with admission, pain control, and orthopedic surgery consult.

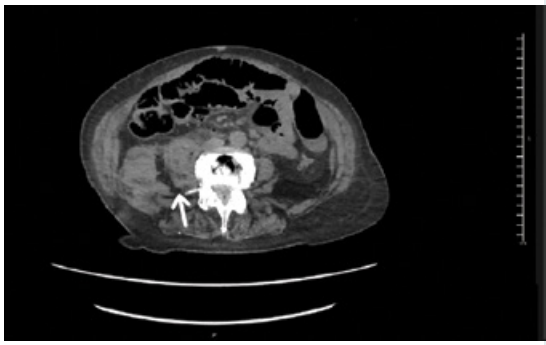

IMAGE 1. Hematoma and retroperitoneal bleed

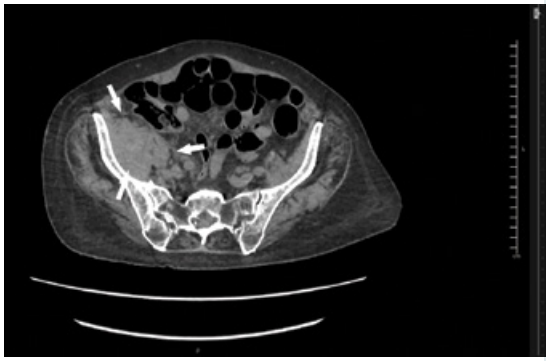

IMAGE 2. Iliopsoas with edema

CT is often used to diagnose rupture or hematoma. The most sensitive imaging is MRI; however, it is not routinely done because of cost and the lack of added value in management.[6] The management is usually conservative, including physical rehabilitation and pain management, though more invasive procedure might be necessary in the setting of continued bleeding or expanding hematoma compressing on vital structures. Overall, prognosis is quite good, though life-threatening anemia and hypovolemic shock can arise. Anticoagulants should be reversed, and underlying bleeding disorders should be treated as otherwise appropriate.[7] ✶

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You Can't Spell Fluoroquinolones without N-O

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Fluoroquinolone antibiotics (ciprofloxacin, levofloxacin, moxifloxacin, among others) are an important class of antibiotics used in a variety of settings. Until fairly recently, ciprofloxacin was a primary treatment option for cystitis. It has been used routinely for inpatient treatment of pneumonia, and many still consider it part of the first-line treatment for diverticulitis. However, in the past few years its role has come into question as concerns abound regarding its safety profile. The quinolone class of antibiotics is associated with tendinitis and Achilles tendon rupture, and potentially significant adverse effects don't stop there: other deleterious muscle and connective tissue effects may be seen.

In 2008, the FDA added its first black box warning to fluoroquinolones, indicating the risk for tendon injuries. Despite this, in 2010, levofloxacin was the best-selling antibiotic in the U.S., with sales exceeding $1.5 billion. By 2012, it was the subject of more than 3,000 lawsuits following severe reactions.

The first successful lawsuit involved an 82-year-old male who was prescribed levofloxacin and a corticosteroid for an upper respiratory infection. He suffered bilateral Achilles tendon ruptures and was ultimately awarded $1.8 million. In a large population-based case control analysis, patients treated with fluoroquinolones had a 4.1-fold increase in risk of Achilles tendon rupture and a 46 times greater risk if there was concomitant use of corticosteroids.[1] Risk factors include elderly males (over the age of 60), patients with chronic renal disease, and those taking corticosteroids. Symptoms of tendinopathy typically begin about 6 days after the onset of treatment, but the risk of tendon rupture persists for up to 90 days. More than 50% of patients experience symptoms that began after their treatment was completed.[2]

Oral fluoroquinolones have been shown to interfere with collagen synthesis. They also have a relatively high volume of distribution and bioavailability: this makes for a dangerous combination. A 2012 case-control study published in *JAMA* concluded that oral fluoroquinolones were associated with an increased risk of developing retinal detachment. Current users of oral fluoroquinolones were nearly 5 times more likely to be diagnosed with retinal detachment than non-users, although the risk did not translate to patients who had already completed treatment.[3] Another case-control study attempted to quantify the risk of acute kidney injury. Researchers found a two-fold increase in the risk of acute kidney injury in patients currently taking fluoroquinolones.[4]

A study in *JAMA Surgery* found that patients who received fluoroquinolones had a higher risk for aneurysms, ruptures, or dissections than those who did not receive the antibiotics. The study showed that normal, unstressed mice who took the antibiotic did not show significant negative effects on the aorta. Mice with moderately stressed aortas who were given fluoroquinolones developed aortic aneurysm and dissection 79% of the time, compared to 45% of those moderately stressed mice who did not receive the antibiotic[5]. This led the FDA to issue the following statement in December 2018: *"A U.S. Food and Drug Administration (FDA) review found that fluoroquinolone antibiotics can increase the occurrence of rare but serious events of ruptures or tears in the main artery of the body, called the aorta....Fluoroquinolones should not be used in patients at increased risk unless there are no other treatment options available. People at increased risk include those with...high blood pressure, certain genetic disorders that involve blood vessel changes, and the elderly..."*

The manufacturer is reportedly facing an $800 million lawsuit, alleging that the company hid vital information about side effects of levofloxacin. The lawsuit argues, among other things, that in 2015 at an Advisory Committee meeting, the manufacturers were made aware of and chose to ignore a potential link between levofloxacin and "Fluoroquinolone-Associated Disability" (FQAD). Long-term FQAD may manifest as chronic fatigue, neuropathies, and memory and concentration problems.

There are 3 conditions for which fluoroquinolones are still considered first-line treatment:

1. **Prostatitis**
2. **Anthrax**
3. **Plague**

In the absence of any of these conditions, strongly consider an alternate class of antibiotic.

For instance, for years, the go-to outpatient treatment for patients diagnosed with diverticulitis has been 10 days of ciprofloxacin and metronidazole. An acceptable alternative is amoxicillin-clavulanate 875 mg every 8 hours (or 1 g every 12 hours). Trimethoprim-sulfamethoxazole (1 double-strength tablet every 12 hours) can also replace ciprofloxacin and be used in concert with metronidazole. A 2017 study questioned the use of antibiotics altogether in a first episode of CT-proven uncomplicated acute diverticulitis. Approximately 260 patients were randomized to observation and 260 to antibiotics; there were no significant differences between the groups for complications, ongoing diverticulitis, recurrence, sigmoid resection, readmission, or mortality.[6] Antibiotics remain the standard of care and should be prescribed, but this study does provide food for thought.

When prescribing fluoroquinolones, consider any reasonable alternative. If there is none, explain the potential risks and benefits to your patient. Document this conversation to minimize risk in the event of a bad outcome. ✶

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Don't Get Burned by Hydrofluoric Acid Toxicity

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Hydrofluoric acid (HF) is used in a variety of industries, including plastic, dye, semiconductor, and fertilizer manufacturing, beer fermentation, and in petroleum production. It is also found in rust remover, various pesticides, refrigerants, car wash cleaning products, and detergents.[1-4]

Most HF burns occur in the occupational setting, although toxicity from household products is also reported. Cutaneous exposures are most common, especially to the fingers and hands. Hydrofluoric acid toxicity may also result from ingestion or inhalation.[2]

Dilute aqueous HF is a weak acid, but at concentrations greater than 20%, severe and potentially lethal burns can occur. Hydrofluoric acid behaves as a strong acid at concentrations > 50%. **Burns covering even a small surface area can be fatal.**[2] Unlike strong acids, HF is highly penetrative due to primarily being in the non-dissociated state, causing deep tissue destruction via liquefactive necrosis and allowing for easy entry into the systemic circulation.[1,2,5] Once within the tissues, the fluoride ion complexes with calcium and magnesium, causing their depletion. Hyperkalemia results from increased cellular permeability and inhibition of the Na+/K+ pump. Neuron depolarization and severe pain result from these electrolyte abnormalities.[1] Fluoride also causes myocardial irritability, predisposing to dysrhythmia.[2]

In 2017, 676 HF exposures were reported to U.S. poison centers. There was 1 fatality and an additional 6 that resulted in life-threatening signs and symptoms.[6] In the past, there have been several mass-casualty incidents involving HF. In 2012, 8 tons of HF leaked from a chemical plant in South Korea, killing several and affected thousands. There were 2 separate incidents in China in 2014. A rust remover leak injured 48 people, and a tanker truck collision resulted in several deaths and more than 250 injuries.[7]

Clinical Presentation

The classic finding of HF toxicity is pain out of proportion to the exam.[1] The discomfort is often described as severe throbbing.[8] The National Institutes of Health classifies HF burns based on the concentration of the product. In exposures to concentrations > 50%, there will be immediate pain and tissue damage, which may include erythema, blistering, ulceration, underlying bone damage, and tenosynovitis. Systemic toxicity develops more slowly and can manifest as nausea, vomiting, abdominal pain, renal failure, hepatic failure, seizure, hypotension, dysrhythmia, or heart failure. Electrolyte abnormalities (hypocalcemia, hypomagnesemia, and hyperkalemia) are common.[2] Coagulopathy may develop secondary to hypocalcemia. The combination of coagulopathy and chemical burns may lead to hemorrhage.[2] Necrotic lesions of the kidneys and myocardium have been observed on autopsy.[9]

Systemic toxicity is also likely following ingestion or inhalation.[1,10] Gastrointestinal (GI) and pulmonary toxicity, including bronchospasm and pulmonary edema, may develop.[2]

Tissue destruction and pain usually occur within 1-8 hours after exposure to HF concentrations of 21-50%. Some systemic toxicity is possible, but typically less severe than what is observed at higher concentrations. At concentrations < 20%, signs and symptoms may be delayed up to 24 hours. The toxic effects of lower-concentration exposures are generally confined to the affected areas; systemic toxicity is unlikely.[2]

Diagnostic Testing

Patients with significant HF exposures require continuous cardiac monitoring. A 12-lead EKG should also be obtained to evaluate for dysrhythmia and interval abnormalities. Prolongation of the QTc interval is one of the best indicators of systemic HF toxicity; symptoms of hypocalcemia (eg, tetany) are often absent.[10] Serial electrolyte levels should be measured, and more significant exposures warrant more frequent laboratory testing, as often as hourly in large or concentrated exposures.[1,2]

Management

Pain relief is the primary indicator of treatment success. Multiple treatment algorithms have been proposed, but there is no consensus on management.[2] However, there are steps that are universally recommended.

Irrigation: As with any chemical burn, the first step is to immediately irrigate with water for 15-30 minutes to remove and dilute the acid.[1] Irrigation will not effectively remove HF that has already penetrated into deeper tissue.[2]

Calcium gluconate gel (topical): Application of topical calcium gluconate gel turns the fluoride into an insoluble salt, preventing further absorption. This reduces the amount of tissue destruction and systemic toxicity. It is reasonable to initially apply the gel to

affected areas every 30 minutes. Once pain is controlled, the frequency can be reduced to every 4 hours. Consider filling an examination glove with gel to treat hand burns.[9] Commercially-prepared calcium gluconate gels are available. Alternatively, homemade gel can be prepared by combining 100 mL of water-based lubricant with 2.5 g of calcium gluconate.[1,10] The majority of HF burns can be effectively treated with topical calcium gluconate, despite its relatively poor skin penetration.[7]

Calcium gluconate infiltration: Local infiltration allows for much greater skin penetration than topical application can provide, and its use should be considered for significant burns. Infiltration is generally unnecessary for exposures of <20% HF. Infiltration is performed with a small gauge needle, e.g. 25- or 27-gauge. Inject approximately 0.5 mL/cm^2 of 5% calcium gluconate into the affected skin and subcutaneous tissue.[1,2] For finger injuries, do not exceed 0.5 mL per phalanx to prevent an excessive rise in compartment pressure.[1]

Calcium gluconate, intravenous: Calcium gluconate should be administered intravenously following significant exposures and in cases of hypocalcemia. Intravenous calcium gluconate decreases pain and prevents extension of the burn to deeper tissues.[2] For extremity burns, regional intravenous calcium gluconate may be administered via Bier block. In this technique, a tourniquet is placed proximal to the burn, and calcium gluconate is administered intravenously distal to the tourniquet. One approach is to inject 10 mL of 10% calcium gluconate diluted in 30–40 mL of normal saline and maintain for 20–25 minutes.[11] This regional technique may be complicated by electrolyte abnormalities and dysrhythmia once the tourniquet is released, so cardiac monitoring and serial laboratory tests are essential. Fortunately, because most victims are young and otherwise healthy, significant systemic complications are rare.[8]

Calcium gluconate, arterial: Arterial injection of calcium gluconate has a high incidence of complications, e.g. arterial spasm, necrosis, dysrhythmia, and vasculitis. Some experts recommend that this procedure only be done via angiography, making it less practical for ED use. There is no demonstrable benefit when compared to intravenous calcium gluconate for most HF exposures. Intra-arterial calcium is theoretically advantageous in severe burns affecting tissues with clear arterial distribution and in small spaces that cannot accommodate large volumes of locally- infiltrated calcium. Cardiac monitoring is essential when administering calcium intra-arterially.[2]

Techniques for elimination of fluoride: Hemodialysis should be considered in patients with refractory hypocalcemia and/or hyperkalemia. Diuresis and urine alkalinization may also enhance the elimination of fluoride.[1-2]

Non-Skin/Dermal Exposures

- **GASTROINTESTINAL**
  This form of exposure is very rare. If it suspected, concurrent inhalational exposure should also be considered. Hydrofluoric acid is rapidly absorbed by the GI tract, leading to vomiting, abdominal pain, hemorrhage, perforation, and systemic effects.[2]
- **INHALATIONAL**
  Anhydrous HF boils at room temperature, and aqueous HF releases fumes that can lead to inhalational exposure.[2] Consider the possibility of inhalational exposure in any patient with HF burns to the face, head, or neck, as well as in burns sustained in confined spaces. Inhalational injuries may also occur following exposures involving greater than 5% of the body surface area, in burns from concentrated (ie, > 50%) HF solutions, and in patients who are not properly decontaminated in a timely fashion.[10] Inhalation may cause fever, chills, pulmonary edema, hemorrhage, chest discomfort, cyanosis, and wheezing. Obtain chest radiography and provide supplemental oxygen in these patients. Consider nebulized calcium gluconate (2-3%), positive pressure ventilation, and intubation in more severe cases.[2]
- **OCULAR**
  Ocular exposure is an ophthalmologic emergency. As with any ocular chemical burn, irrigation is the immediate priority. Remove contact lenses, if present, after brief irrigation, and then resume irrigation.[10] Consider administration of calcium gluconate (1-10%) eye drops.[1] Instill 1-2 drops every 2-3 hours into the affected eye(s). Hydrofluoric acid rapidly penetrates into the anterior chamber; immediate aqueous humor removal and replacement is commonly required despite aggressive irrigation. Ophthalmology must be consulted for ocular exposures.[2]
- **FINGERNAIL**
  HF easily penetrates the nail, and removal is generally required to apply calcium gluconate gel to the affected nailbed.[1]

Provider Safety

Health care professionals must take precautions to avoid contaminating themselves while caring for HF victims. The use of "double gloving" is recommended in case a glove has microscopic pinholes. Providers should also consider the use of additional personal protective equipment, including eye protection and gowns.[8]

TAKE-HOME POINTS

- ✓ HF is found in a variety of products and used in multiple industries.
- ✓ Burns to the upper extremity and hand are most common and may cause severe pain.
- ✓ HF is lipophilic, enabling it to cause deep burns and systemic toxicity.
- ✓ Systemic toxicity is mostly due to electrolyte abnormalities, including hypocalcemia, hypomagnesemia, and hyperkalemia.
- ✓ Important diagnostic tests for HF burn victims include EKG and serial electrolyte measurements.
- ✓ Most HF burns can be successfully treated with topical calcium gluconate.
- ✓ Resolution of pain is a good indicator of treatment efficacy.
- ✓ If topical application of calcium gluconate is not effective, consider local infiltration, intravenous injection (including regional injection using Bier block), and arterial injection.
- ✓ There are specific methods used to manage non-skin exposures to HF (gastrointestinal, inhalation, ocular, etc.). ✶

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Difficult to Ventilate and Oxygenate
The Pediatric Airway Overview

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Case 1

A 2-year-old boy from Ecuador is brought into your community ED. The nurse quickly brings the child into the critical care bay because of respiratory distress and begins placing the child on a monitor. You note the child to have inspiratory stridor and to be tripoding. Oxygen saturation is 88% on room air, heart rate is 165, respiratory rate is 55, and auricular temperature is 39.0 C. You are unable to immediately obtain a history from the mother because of a language barrier. You ask the nurse to administer nebulized epinephrine while you grab the pediatric airway cart and contact the "on-call" anesthesiologist.

Case 2

A 4-year-old, fully vaccinated girl is brought in by her father because of an acute onset of cough and wheeze since this morning. In triage the patient is given albuterol and placed on a monitor. When you walk into the room, you observe a mildly tachypneic patient, with inspiratory stridor at rest and clear nasal discharge. On auscultation you hear diffuse wheeze. There are no other pertinent exam findings, but the dad tells you the patient gets frequent upper respiratory infections for which she sometimes requires albuterol. Your differential includes croup, early epiglottitis, foreign body, reactive airway disease and early retropharyngeal abscess. You order nebulized epinephrine along with a portable chest and neck X-ray. After albuterol and nebulized epinephrine, no change is noted on exam. The X-ray tech comes by to show you the image, and while you notice a possible foreign body the nurse calls you over for help.

Case 3

It is mid-December and a mother brings in her 6-month-old boy because of worsening respiratory distress and decreased energy and appetite over the past 3 days. His past medical history is significant for 34 weeks gestation, and a 2 day NICU stay. He has since been medically stable. The infant is placed on a monitor and his respiratory rate is 70, with an oxygen saturation of 86%, a heart rate of 180, and a temperature of 102.9 F. On exam, you note upper airway congestion, nasal flaring, subcostal retractions, abdominal breathing, and bilateral wheezing. Nasal suctioning only improves oxygen (O2) saturation to 88% with no change in work of breathing (WOB). You have the nurse start blow-by with no improvement in WOB or O2 saturation. The patient is administered nebulized albuterol, while IV access, blood cultures, and a portable chest X-ray are obtained. You instruct the nurse to give 20 cc/kg of fluids.

The Pediatric Airway

As emergency physicians-in-training, we can feel uncomfortable when caring for critically ill pediatric patients. This anxiety can be heightened when facing a pediatric patient in respiratory distress and a potentially difficult airway. This article will review some of the basics of the management of the pediatric airway (including anatomical differences, initial interventions, definitive airways, medication selection, ventilator settings, and difficult airways) in the hopes of somewhat lessening that anxiety.

The steps to the pediatric airway parallel that of an adult. Just as in adults, managing the pediatric airway requires a familiarization with the equipment and a stepwise approach that begins with assessment of airway patency followed by identifying an oxygenation or ventilation issue.

For the child in respiratory distress, initial interventions include low-flow oxygen delivery systems (<10 L/min), such as a nasal cannula or simple oxygen mask. If the patient fails to respond, high-flow oxygen delivery systems, such as non-rebreathing mask with reservoir or high flow nasal cannula can be used.[18] These modalities deliver oxygen but do not help with pressure support. If these interventions fail, initiation of non-invasive positive pressure ventilation via high-flow nasal cannula (HFNC), continuous positive airway pressure (CPAP), or bi-level positive airway pressure (BIPAP) should be attempted.

Finally, in patients with persistent respiratory distress despite these efforts, a definitive airway should be established. While supplies are gathered for the definitive airway, a bag-valve mask (BVM) can be used to bridge the patient. There are several options for invasive airways in the pediatric patient, including supraglottic airways (King tube, LMA, etc), endotracheal, or in some cases, nasopharyngeal intubation. As a last resort, needle-cric and jet ventilation in pediatrics under 8 years old or traditional cricothyroidotomy for those patients above 8 years old can be attempted.[2]

When attempting intubation in children, several anatomic factors can result in airway obstruction or can complicate the visualization of the vocal cords. Children younger than 2 years of age have large occiputs, causing neck flexion in the supine position, leading to tracheal obstruction and malalignment of the oropharynx with the larynx and vocal cords.[1,10] The pediatric airway is also shorter, floppier, and narrower than in adults, making it more susceptible to kinking and obstruction.[1] Children also have larger tongues relative to the size of their oropharynx, which increases the risk of obstruction and difficulty visualizing the cords.[1,10] Furthermore, the epiglottis is floppier, making it difficult to manipulate in order to visualize vocal cords.[1,10] There is also increased risk of right mainstem intubation due to the

short tracheal distance leading to the right main bronchus.[1] Even after successful maneuvering of the endotracheal tube (ETT) past the vocal cords, there may be difficulty passing the ETT further. This is because the narrowest portion of the pediatric airway is the cricoid cartilage, rather than the vocal cords in adults.[1,10] If the ETT is difficult to pass a smaller ETT must be attempted to avoid mucosal injury and airway edema, which can be exchanged via a bougie/gum elastic bougie, for example.

Besides anatomic differences, there are physiologic differences that impact the management of the pediatric airway. Pediatric patients have a higher oxygen demand and oxygen consumption. Combined with smaller airway volumes, the higher oxygen demand and consumption results in lower functional residual capacity and a smaller oxygen reserve.[4] Additionally, abdominal distension, most commonly due to too much BVM ventilation prior to intubation, can lead to decreased functional residual capacity and O2 desaturation.[4]

There are a number of techniques providers can utilize in order to optimize first-pass intubation rates. Placing a towel underneath the shoulders and using the head tilt-chin lift maneuver can overcome the neck flexion caused by the larger occiput, resulting in better oropharyngeal and laryngeal alignment, and reduce airway obstruction.[1] It is important to avoid overextension, which may also result in airway obstruction due to the flexible trachea. In order to avoid airway obstruction when utilizing non-invasive airway adjuncts like BVM, as a result of the large pediatric tongue, it is important to open the mouth widely via the jaw thrust maneuver or by placing an oral or nasal airway.[10] The jaw thrust maneuver can also be utilized to prevent the large pediatric tongue from obscuring your view of the airway. Additionally, a miller blade should preferentially be utilized in children < 2 years old to allow for better control of the floppy epiglottis and better view of the anterior pediatric airway.[10]

Set up for the pediatric airway is no different from the adult airway, except for the sizing of equipment. The Broselow tape is a useful adjunct as it can be used to determine the size of equipment needed and the medication dosing. The Broselow tape is placed at the patients head and is used to measure the length of the child, which correlates to the dosing and sizing of medication and equipment. Remember the mnemonic, RED to HEAD, which means to place the red color of the Broselow to the patients head, and then measure to the patient's heel.

LMA and Laryngoscope Sizing[12,13]

Age	Preemie	Term	1-6mo	6mo-2yr	2-5yr	5-8	9-13
Weight	< 3 kg	> 3 kg	4-6 kg	6-12 kg	12-20 kg	20-30 kg	30-45 kg
Blade	Miller 0	Miller 0-1	Miller 1	Miller 1	Miller 1-2 Mac 2	Miller 2 Mac 2	Miller 2 Mac 2-3
Mask	Neonate	Infant	Infant	Toddler	Child	Child/Small Adult	Small Adult

Induction Agents[14]

Induction Agent	Dose	Onset	Duration
Etomidate	0.3-0.5 mg/kg IV push	30-60 sec	3-5 min
Ketamine*	2 mg/kg IV push over 30 sec	~ 30 sec	5-10 min
Propofol	2-4 mg/kg IV push	~ 30 sec	3-10 min

***Pearl:** Ketamine is not recommended for children < 3 months

Paralytics[15]

Paralytic*	Dose	Onset	Duration
Cisatracurium	0.2 mg/kg IV	3-5 min	45-60 min
Pancuronium	0.05-0.1mg/kg	3-5 min	90-120 min
Vecuronium	0.1-0.2 mg/kg	2-3 min	60-90 min
Rocuronium	1 mg/kg	1-2 min	40-60 min
Succinylcholine	1- 2 mg/kg	~ 60 sec	5-10 min

***Pearl:** Do not use a paralytic unless you are confident you can oxygenate and ventilate using a BVM

PEDIATRIC INTUBATION PEARLS

- **RED** TO HEAD
- Cuffed ETT = (Age/4) + 3.5
- Uncuffed ETT= (Age/4) + 4
- Oral Airway/Nasopharyngeal Airway
  - Oral airway sizing: Measure from angle of mouth to angle of mandible.
    - Do not use in awake patients!
  - NPA sizing: Measure from tip of nose to ear lobe.
- Intubate to a depth of 3x ETT size

Difficult Pediatric Airways

Management of the airway should always include preparation for possible difficulties. Familiarizing yourself with the location of the necessary equipment and a step-wise assessment of your equipment beforehand is essential. You should have yankauer suction; BVM attached to oxygen, a monitor that can measure end tidal CO2, multiple laryngoscope blade sizes with working lights, and multiple sizes of ETT with stylets. If using video laryngoscopy, the video should be tested prior to use. There should also be airway adjuncts and supplies for difficult airways including bougie, OPA, NPA, and scalpel. However, standard issue bougies do not fit most pediatric sized ET tubes, rather a gum elastic bougie (GEB) should be used.[18] Finally, the patient should have a working IV or IO line and the nurses should have appropriate medications, doses. If there is time, consent should be obtained and the ventilator set up.

If you attempt to intubate and fail then the most experienced provider should be given the next attempt. After 1-2 failed attempts at securing an airway either via direct or video laryngoscopy, after you BVM to re-oxygenate, your next step can include utilization of a bougie/GEB or lighted stylet.[18] However, an experienced provider should make the attempt as GEB and lighted stylets are not routinely used in pediatrics.[18] If that fails, you may be able to temporize with a supraglottic device until you can get the patient to a pediatric ENT or anesthesiologist. Care should be taken as supraglottic devices are contraindicated in epiglottitis. The last line in a critically ill pediatric patient who has respiratory failure is cricothyroidotomy. Depending on the age, either needle cricothyroidotomy with jet ventilation in

children < 8 years old (due to the smaller size of the cricoid membrane) or traditional cricothyroidotomy in children > 8 years old should be implemented.[2]

When attempting a needle cricothyroidotomy be wary of the compressibility of the cricoid, as there is a larger risk of puncturing through to the esophagus compared to adults.[1] To perform needle cricothyroidotomy you identify the cricothyroid membrane, found in-between thyroid and cricoid cartilage, and prepare a sterile field. Connect a 14 gauge needle to a syringe with 3cc-5cc of sterile saline. Insert the needle and advance at a caudal angle while withdrawing until you see air bubbling, confirming placement.[1] A 14 gauge to 3.0 ET tube adapter is then connected and the patient can be oxygenated but very poorly ventilated, with approximately 40-60 minutes max until a more definitive airway is necessary.

Post Intubation

Post-intubation steps are similar between pediatric and adult patients. Tube placement should be confirmed through auscultation of bilateral breath sounds, fogging of the ETT, and CO2 capnography. A gastric or nasogastric tube should be placed to decompress the stomach contents in order to reduce aspiration risk while improving expansion of the diaphragm and therefore, functional residual capacity. It is important to obtain post-intubation chest X-ray for tube confirmation; similar to adults, the tip of the tube should be 2 cm above the carina.[8]

After the tube placement is confirmed, the patient should be placed on a ventilator and sedation started. Below are some general guidelines for initial settings in the emergency department which can be modified based on patient needs.[6,7]

An important contrast between pediatric and adult basic ventilator settings is that pediatric patients are most often placed on pressure control setting due to the higher risk of ventilator associated barotrauma.[6,7] After intubation and placement on a ventilator, then, based on the pathophysiology of the patient, a sedative and/or long acting paralytic may need to be initiated. Benzodiazepines, like midazolam are commonly used in pediatric patients, as well as fentanyl or propofol. These can be used as either pushes or continuous infusions.[9]

Case 1 Continued

The on-call anesthesiologist is 10 minutes out. You are worried about epiglottitis and anticipate a difficult airway. You set up for emergent intubation with suction, BVM, 1.0 miller blade, and a cuffed 3.5/4.0 ETTs. As backup you have video laryngoscope, GEB and a 14 gauge needle with a 3.0 ET tube adapter for possible needle cricothyroidotomy. Suddenly the patient becomes unconscious and bradycardic to the 50s. CPR is initiated and oxygenation with a BVM is started. The O2 saturation remains in the 80s despite repositioning of the airway, verifying a tight face-mask seal and continued BVM use. When you attempt to visualize the cords during direct laryngoscopy, they are obstructed by a markedly swollen epiglottis. You attempt to intubate via video laryngoscope and GEB, but are unsuccessful. Given the situation, you perform a needle cricothyroidotomy and start jet-ventilation with successful improvement in O2 saturation. Anesthesia arrives and you aid in the successful nasal intubation of this patient via a flexible bronchoscope and 3.0 uncuffed ETT. You start a midazolam drip, send labs, start IV antibiotics and arrange for transfer to the nearest Pediatric Intensive Care Unit.

General Pediatric Ventilator Settings[6,7,11]

Parameters	General Pediatric Settings
FiO2	100% (quickly trend down)
Respiratory Rate	Parallels Normal Respiratory Rate or Pathophysiology
Inspiratory Time	0.4 (Infants)–1.0 (Adolescents)
PEEP	3-5 cm H_2O
Pressure Regulated-Volume Control Ventilation	
Tidal Volume	5-8 mL/kg ideal body weight
Pressure Support	4-6 cm H_2O
Pressure Control Ventilation	
Peak Inspiratory Pressure	<28

Case 2 Continued

You enter the exam room and see that the patient is grabbing at her neck and has no stridor. You start abdominal thrusts as she is unable to make sounds and is still responsive. Simultaneously, the nurse grabs the airway cart, Magill forceps, a 2.0 Mac blade, a 4.0/4.5 cuffed ET tube, a child BVM and the cricothyroidotomy tray. After placing the patient supine on the bed, she becomes unresponsive and you start CPR, while the nurse calls for help and contacts the on-call ENT resident. You visualize the airway and see a small round object in the trachea right below the vocal cords, which you cannot grab with the Magill forceps. You quickly decide to intubate, pushing the foreign body down into the right main bronchus, and then selectively intubate the left lung with improvement in ventilation and oxygenation via BVM. The patient is placed on a pressure regulated low tidal volume ventilator setting and transitioned with ENT to the operating room for rigid bronchoscopy with successful removal of the foreign body.

Case 3 Continued

The portable chest X-ray was unremarkable. You begin HFNC at 2 L/kg/min, which brings the oxygen saturation to 88% and the patient continues to be tachypneic to 75.[16] Given the severity of disease, you administer nebulized epinephrine with minimal improvement in WOB. The patient starts to fatigue and the O2 saturation falls back to 80% with a RR of 80 and HR of 175. Due to impending respiratory failure you intubate the patient with etomidate and rocuronium, using a Miller 1 and 3.5 cuffed ET tube. You start a midazolam drip and place the patient on a pressure control settings with FiO2 of 100% while awaiting transfer to PICU. ✶

(Disclaimer: Cases are all fictional)

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Management of Simple and Systemic Asphyxiant Injury

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Introduction

Asphyxiants are dangerous substances that deprive the body of oxygen. They are separated into two categories, simple and systemic asphyxiants, based on their mechanism of action (Figure 1).

Simple asphyxiants include any gas that dilutes oxygen in ambient air and decreases the fraction of inspired oxygen (FiO_2) below 21%.[1] Examples include carbon dioxide, nitrogen, fuels like propane and methane, and noble gases like argon. Systemic asphyxiants on the other hand, work by interfering with oxygen transport or with intracellular utilization of oxygen. These include carbon monoxide (CO), cyanide (CN), and hydrogen sulfide (HS). Unlike simple asphyxiants, systemic asphyxiants are not limited to gases, and include non-gaseous substances that can be ingested.[2] If untreated, asphyxiant exposure can lead to myocardial damage, permanent neurological deficits, and death.

The most common source of asphyxiant poisoning is CO, with more than 50,000 cases annually in the United States primarily related to fires, motor exhaust, and gas leak.[3] CN has been associated with occupational exposure in mining and manufacturing, and up to 35% of fire-related inhalational injuries are linked to CN toxicity.[4,5] HS is a byproduct formed by the decay of organic material and the exposure risk is usually occupational, particularly for those working in oil drilling or with manure.[1,6,7] Exposure to non-CO simple asphyxiants is less common, with the American Association of Poison Control Centers reporting about 2,500 cases in 2016. These are mostly related to industrial exposure while working in confined spaces.[2,8]

FIGURE 1. Common Asphyxiants

Simple Asphyxiants: Gases that displace oxygen in ambient air and impair oxygen exchange in the lungs when present at high enough concentrations. Includes:

- Carbon Dioxide
- Propane, Methane
- Nitrogen
- Noble Gases (Argon)

Systemic Asphyxiants: Agents that impair the body's ability to properly utilize oxygen, either by interfering with hemoglobin oxygen transport or cellular respiration. Includes:

- Carbon Monoxide
- Cyanide
- Hydrogen Sulfide

General Approach to Management

When encountering a patient who potentially suffered asphyxiant poisoning, several steps in management should be taken regardless of exposure. First, a good history is essential in directing treatment. These patients may be found unconscious, and if brought in by ambulance, questions to ask EMS should include the circumstances in which the patient was found. If they were brought in after a fire, found in a confined space, or found in any industrial-type setting, asphyxiant toxicity should be included in the differential. For conscious patients, it can be helpful to ask if they recall any distinct odors prior to symptom onset. Many odorless simple asphyxiants such as propane are used as fuels and are injected with a substance with a "rotten egg scent" during manufacturing. HS has also been said to have this smell, whereas CN has been described to have a "bitter burning taste."[2,9]

History should focus on affected organ systems, including the nervous, cardiac, and respiratory systems. Neurologic presentations vary, ranging from headache and dizziness to seizure and coma. Likewise, cardiorespiratory symptoms including chest pain, dysrhythmias, shortness of breath, cough, and respiratory distress may be present.[10] It is especially important to include asphyxiant exposure on your differential diagnosis when patients present with flu-like symptoms in the winter time. During this time of year the flu is common but so is CO exposure from patients bringing sources of heat such as stoves or generators indoors, and both diagnoses present with similar symptoms.

Combining physical exam with pertinent history findings is critical in guiding physicians towards the diagnosis of asphyxiant exposure (Figure 2). Non-specific physical exam findings include altered mental status, respiratory distress, bradycardia, hypotension, and cardiac arrhythmia. Some classic signs include "cherry red" appearance of the skin and lips in CN and CO poisoning.[3,10] Additionally, some asphyxiants like HS may also act as mucous membrane irritants, causing eye redness or signs of pulmonary edema.[2] If a patient quickly and significantly improved with oxygen therapy alone, they are more likely to be suffering from simple asphyxiant exposure rather than systemic asphyxiants, the latter of which act in more complicated mechanisms than decreasing FiO_2.[1]

All patients treated for asphyxiant injury must be given supplemental oxygen and must be evaluated in a systematic manner. Assess major organ systems by beginning with the ABCs of evaluating Airway, Breathing, and Circulation. Once ABCs are secured, general management of systemic asphyxiant exposure must also include consultation of a poison control center and decontamination via removal of the clothes and low-pressure water irrigation.[10] Systemic asphyxiant patients should receive

100% oxygen via non-rebreather mask or mechanical ventilation, as well as cardiac monitoring and serial EKG's due to a risk of myocardial damage and dysrhythmia. Other relevant diagnostics include arterial blood gas, serum lactate, and co-oximetry. Imaging should be guided by clinical exam and history, but may include a chest X-ray, as well as advanced brain imaging.[2,9]

Simple Asphyxiants

For simple asphyxiants, treatment focuses on removal from the causative agent, securing the ABCs, and providing supplemental oxygen. Patients suffering from simple asphyxiant exposure often receive oxygen therapy prior to arrival and can be significantly improved by the time they arrive to the ED. The patient should still be closely observed and monitored for several hours, depending on the exposure, and after discharge should be referred to outpatient follow-up for any potential delayed neurologic sequelae.[1,2,11]

Systemic Asphyxiants

Carbon Monoxide

Carbon monoxide works by binding hemoglobin with higher affinity than oxygen, significantly impairing oxygen carrying capacity and limiting its delivery to the tissues.[3] Clinical management focuses on delivery of 100% oxygen via non-rebreather mask or mechanical ventilation. Untreated, carboxyhemoglobin has a half-life of about 4 to 5.5 hours.[18] Importantly, in CO poisoning, oxygen saturation via standard pulse oximetry will appear normal despite hypoxia because this oxygen measurement method cannot distinguish between carboxyhemoglobin and oxyhemoglobin. Co-oximetry is required to do so, with laboratory methods being recommended over pulse co-oximetry.[2] Nonsmokers have carboxyhemoglobin levels of about 3% or less, whereas smokers can have carboxyhemoglobin levels up to 10-15%.

Highflow oxygen can reduce the half-life of CO to 90 minutes and should be administered until the carboxyhemoglobin level is less than 5%.[18] Additional care will focus on managing ABCs and potential complications, including metabolic acidosis, cardiovascular or neurological damage, and shock.[1,2,3]

Another treatment considered for CO poisoning is hyperbaric oxygen therapy (HBOT), which reduces the half-life of carboxyhemoglobin. The use of HBOT has been shown to decrease long-term neurologic dysfunction, but its use is not currently universally recommended for CO toxicity or for systemic asphyxiant exposure.[3] If available, HBOT is generally accepted in patients with coma, signs of myocardial ischemia, neurological deficits, or a CO level greater than 25% in non-pregnant patients or greater than 15% in pregnant patients.[3,12,13]

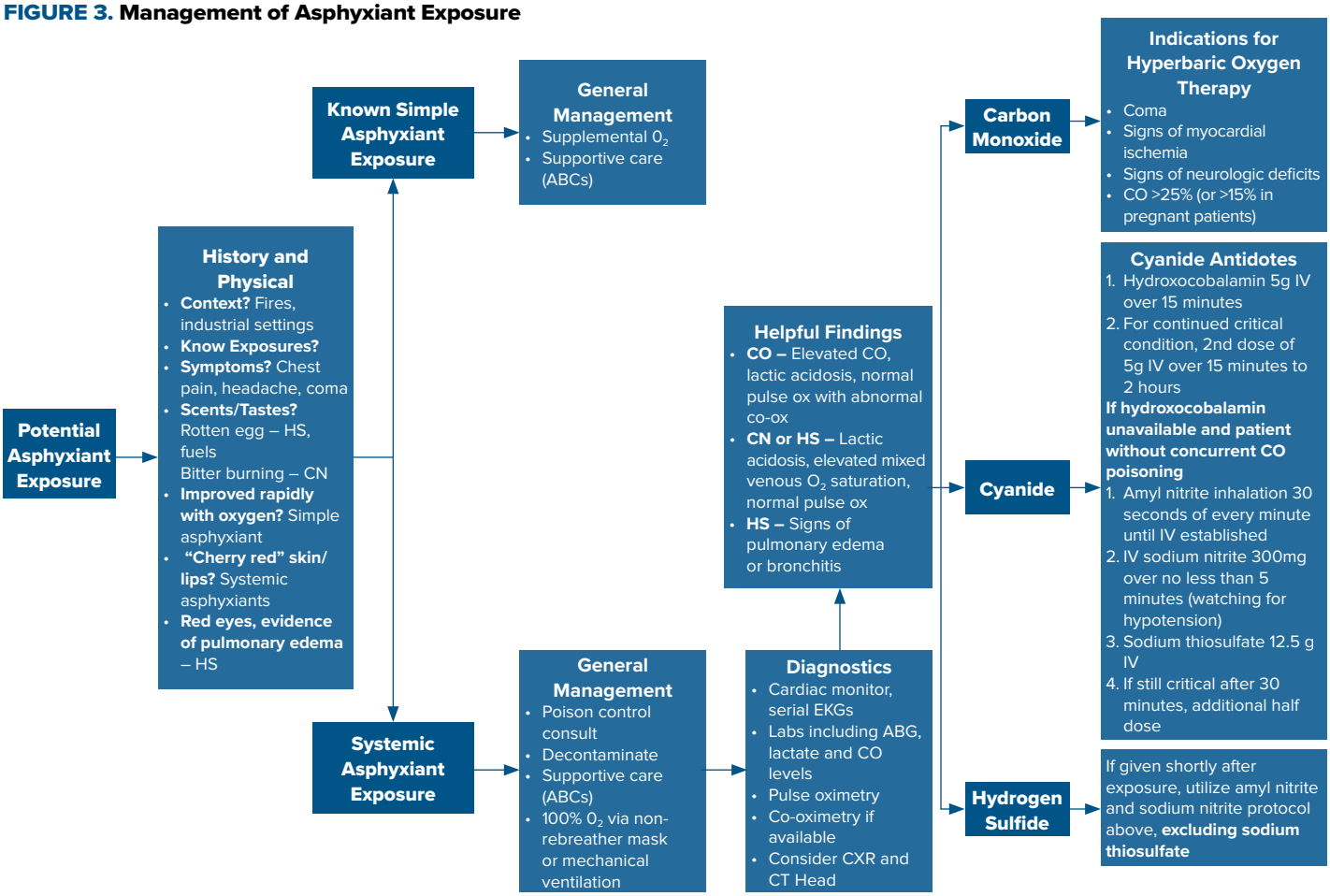
Cyanide

Cyanide acts by impairing cytochrome oxidase of the mitochondrial electron transport chain and thereby inhibiting aerobic respiration.[1] It is important to note that there are many cyanogenic agents including laetrile, acetonitrile, and even peach seeds that can be ingested and metabolized to CN, causing a delayed

FIGURE 2. Clinical Characteristics of Asphyxiant Exposures

	Sources of Exposure	Mechanism of Toxicity	History and Physical Findings	Laboratory Findings	Treatment
Simple Asphyxiants	• Work in confined spaces (manholes, pipelines) • Propane/methane used for heating	Gas replaces oxygen in ambient air, reducing FiO2below 21%	• "Rotton egg" scent (propane, methane) • Improved significantly with oxygen therapy alone	CO2 can cause respiratory acidosis	• Supplemental oxygen • Supportive care and observation (ABCs)
Carbon Monoxide (CO)	• Fire • Motor vehicle exhaust • Poorly ventilated fuel-burning devices	CO binds hemoglobin with greater affinity than oxygen and displaces it	• No notable scent prior to symptom onset • "Cherry red" skin and lips	• Metabolic acidosis • Elevated lactate • Elevated CO levels	• 100% oxygen via non-rebreather mask or mechanical ventilation • Supportive care and observation (ABCs) • May include hyperbaric oxygen
Cyanide (CN)	• Fire • Occupational eg, mining; plastics and rubber production • Sodium nitroprusside • Laetrile, acetonitrile • Peach seeds	CN impairs oxidative phosphorylation by inhibiting cytochrome oxidase	• "Bitter burning taste" • "Cherry red" skin and lips	• Metabolic acidosis • Elevated lactate • Elevated mixed venous oxygen saturation	• 100% oxygen via non-rebreather mask or mechanical ventilation • Supportive care and observation (ABCs) • Hydroxocobalamin or nitrite antidote
Hydrogen Sulfide (HS)	• Occupational eg, oil workers • Decomposing animal products eg, manure • Caves with sulfur springs	HS impairs oxidative phosphorylation	• "Rotton egg" scent • "Cherry red" skin and lips • Eye redness • Signs of pulmonary edema	• Metabolic acidosis • Elevated lactate • Elevated mixed venous oxygen saturation	• 100% oxygen via non-rebreather mask or mechanical ventilation • Supportive care and observation (ABC's) • May include nitrite antidote

FIGURE 3. Management of Asphyxiant Exposure



presentation.[2] Because no rapid laboratory confirmatory test exists for CN poisoning, empiric treatment will likely be required. In addition to the history and physical discussed above, findings that can aid this diagnosis include severe lactic acidosis greater than 10 mmol/L, anion gap metabolic acidosis, elevated mixed venous oxygen saturation, and a normal pulse oximeter reading.[10]

Two antidotes are available for CN poisoning in the United States, the Cyanide Antidote Kit (CAK) and hydroxocobalamin. Numerous studies have demonstrated the efficacy and safety of hydroxocobalamin relative to CAK, and it has been recommended as the antidote of choice if available.[10, 14, 15, 16] The standard dose is 5g IV over 15 minutes and if the patient remains in critical condition, a second 5g IV dose can be given over 15 minutes to 2 hours.[9] The CAK contains amyl nitrite, sodium nitrite, and sodium thiosulfate. Amyl nitrite perles are broken and given via inhalation for 30 seconds of every minute until an IV is established. Then, 300mg of the sodium nitrite is infused over no less than 5 minutes. Finally, 12.5g sodium thiosulfate is given IV over 10-20 minutes. If the patient remains in critical condition thirty minutes after the first dose, another half-dose can be given. [1,2,9] A major drawback to the CAK is formation of a methemoglobin intermediate, which can be dangerous in smoke inhalation patients because they may have concurrent CO toxicity already reducing hemoglobin oxygen carrying capacity.[2] Additionally, the components of the CAK can cause serious side effects including hypotension and psychosis. [1,2]

Hydrogen Sulfide

Hydrogen sulfide works as a systemic asphyxiant via inhibition of cellular respiration in a similar manner to CN.[7] HS exposure does not benefit from the same body of treatment data as other asphyxiants and does not currently have any proven antidote. Nitrite therapy via the CAK has been recommended if it can be given shortly after exposure, following the same procedure above, excluding sodium thiosulfate.[2,7,17]

Conclusion

Although asphyxiant exposure is a relatively uncommon phenomenon, it is important to keep them in mind when evaluating an altered patient or a patient who has been found down, particularly in industrial or fire related incidents. It is also important to consider these diagnoses in patients who present with vague symptoms, as the history may be the key to narrowing in on the diagnosis. Exposure can leave people unconscious and unable to provide a history, and because of how rapidly these agents can kill, confirmatory testing is often unable to provide a definitive diagnosis in a clinically relevant time frame. Providers must be ready to move quickly if asphyxiant poisoning is suspected and remembering the basics of presentation and treatment can save critical time in the management of these conditions (Figure 3). ✶

References available online

Ketamine's Therapeutic Value in Suicidal Patients

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The management of an acute mental health crisis is often a daily occurrence in the practice of emergency medicine, and yet many of us can feel out of our element when confronted with a suicidal patient. We are comfortable running through our ABCs, looking for acute, life-threatening medical pathology. If a patient presents with an acute abdomen, a gunshot wound, or respiratory failure, we can expect our consultants to expedite the patient's care after our evaluation and stabilization.

But when it comes to first-line treatments for suicidality, our options for acute intervention are limited.

The patient often spends hours in the ED awaiting an evaluation by a mental health provider. Some patients may then be referred to inpatient care, whereas others are merely encouraged to connect with an outpatient psychiatrist. If they are prescribed a selective serotonin reuptake inhibitor (SSRI), many will have to wait several weeks before deriving any benefit — and even then, only 40-60% will respond significantly to their first antidepressant trial.[1,2] Side effects from SSRIs are nearly universal, with studies showing 90% of patients reporting at least 1 side effect and half of patients reporting a moderate to severe side effect. Amongst those who discontinue their SSRI (up to 72% by day 90 of treatment), 36-62% report doing so because of side effects.[3] **Thus the unsettling reality is that the dearth of acute interventions means we discharge many of our depressed patients home without first being able to significantly reduce their symptoms.** This is particularly worrisome in light of data suggesting that the risk of suicide increases immediately after discharge.[4]

But it doesn't have to be this way. An anesthetic that the World Health Organization has listed as an essential medicine since 1985, in part due to

its "high level of safety," has been quietly revolutionizing the treatment of depression for the past 15 years. In 2014 Dr. Thomas Insel, former director of NIMH, declared that this drug "might be the most important breakthrough in antidepressant treatment in decades."[5]

That drug is ketamine.

It turns out that ketamine isn't unique just in its ability to induce anesthesia without significantly impairing the respiratory drive. It also has a robust antidepressant effect, with as many as 70% or more of patients experiencing clinical relief.[6] But what makes it so revolutionary, and of particular interest to emergency physicians, is that it works fast — within 4 hours fast for most patients. And effects can persist, with studies demonstrating the benefit of a single infusion often enduring for 7-14 days.[7]

In the late 1990s, researchers began to realize that the story behind depression is more complicated than simple deficiencies in monoamines. Investigators noticed that the glutamate system also appears to be dysregulated, with *hypo*functioning NMDA activity in cortical regions and *hyper*functioning NMDA activity in subcortical regions.[7] Relying on one of the most widely used NMDA modulators, ketamine, in 2000 Berman et al. conducted a small, double-blind RCT where they gave an infusion of 0.5 mg/kg IV over 40 minutes to 7 patients. **Scores on a depression scale started to decrease within hours of the infusion and continued to fall over the next 3 days.**[8]

Since then, many studies have been conducted throughout the world with consistent results. Ketamine works for the majority of depressed patients, it works fast, and the effects of a single dose persist long after the drug has been completely metabolized.[9] It appears that ketamine causes a release of brain-derived neurotrophic factor, causing synaptogenesis and other significant neuroplastic changes in a number of important brain regions, including the amygdala, hippocampus, dorsal anterior cingulate cortex, prefrontal cortex, and supplementary motor area.[10-12] And in 2017 a meta-analysis by Wilkinson et al. helped identify ketamine as belonging to an exclusive club. Alongside lithium, clozapine, cognitive behavioral therapy, and dialectical behavioral therapy, **ketamine has been shown to reduce suicidality, independent of its effect of improving depression**.[13]

Research is ongoing, looking for the ideal dosing, route of administration, and frequency of administration. The dosage that is used most commonly in these studies, 0.5 mg/kg, is far below the anesthetic threshold (often 1.0-4.5 mg/kg IV) and recreational amounts (often 0.45-1.45 mg/kg), yet can still induce transient intoxication and feeling of disassociation.[7,14] Studies show that giving a bolus is as effective as infusing the dose over an hour.[15] In addition to the intoxicating effects, ketamine has been documented to impact other bodily systems, including cardiovascular (elevated BP and heart rate), neurologic (headache, dizziness, unsteadiness), cognitive (memory loss, confusion, poor concentration), and gastrointestinal (nausea and vomiting).[16] Despite these effects, researchers have found that when a patient is safely seated during an infusion, occasionally observing the patient and monitoring vitals is sufficient for keeping them safe.[15]

Many pharmaceutical companies are currently investigating molecules similar to ketamine, hoping to find one that retains ketamine's rapid antidepressant effects but without the inebriation. The FDA very recently granted approval to a groundbreaking Johnson & Johnson nasal spray of esketamine, the S(+) enantiomer of ketamine. But with an estimated cost of $800 per dose, or $4,720-$6,785 per month, and physician unfamiliarity with the drug, it is possible that many of the patients who need it most will not receive prompt access.[17] And while drug companies continue searching for the best way to harness the antidepressant effects of ketamine, people continue to experience immense suffering from inadequately treated depression and suicidality. This has led to the creation of ketamine clinics in many large cities across the country, where providers are giving depressed patients off-label infusions of ketamine. While these clinics have improved access to this novel treatment for refractory depression, their high cost places it out of reach for many. And it might not be long before ketamine is approved for the treatment of other disorders, as preliminary data suggests that it improves suffering in those struggling with PTSD, bipolar disorder, social anxiety, chronic pain.[18-21]

The field of emergency medicine has much to gain by the development of a fast-acting antidepressant. If we were able to provide infusions in the acute setting for depressed and suicidal patients, as has been done in research studies around the world,[22-24] we could be more confident that our patients can stay safe upon discharge. Or perhaps we can administer a dose of the newly approved esketamine to our suicidal patients, increasing access to a drug that might otherwise be out of reach for many. Imagine if, instead of telling our patients that they need to survive incredible distress and pervasive thoughts of death for a month or more before our medications start to work, we could give them a solution that provides immediate relief. What if we had a drug that could serve as a bridge, helping patients stay safe in the short term as they wait for their SSRIs and/or therapy to start to ameliorate their suffering? And better yet, what if this drug had a proven track record with decades of use and minimal side effects? We have all of this in ketamine. And who is better suited to advocate for making it accessible — whether through the newly approved albeit expensive nasal spray or the more traditional, and cheaper, infusion — for patients in the midst of a mental health crisis than emergency physicians, the first providers these patients often encounter following an admission of suicidality or an attempt at taking their life. ✶

It turns out that ketamine isn't unique just in its ability to induce anesthesia without significantly impairing the respiratory drive. It also has a robust antidepressant effect, with as many as 70% or more of patients experiencing clinical relief. Moreover, it works fast — within 4 hours for most patients.

References available online

Intentional Flecainide Overdose

–A CASE REPORT–

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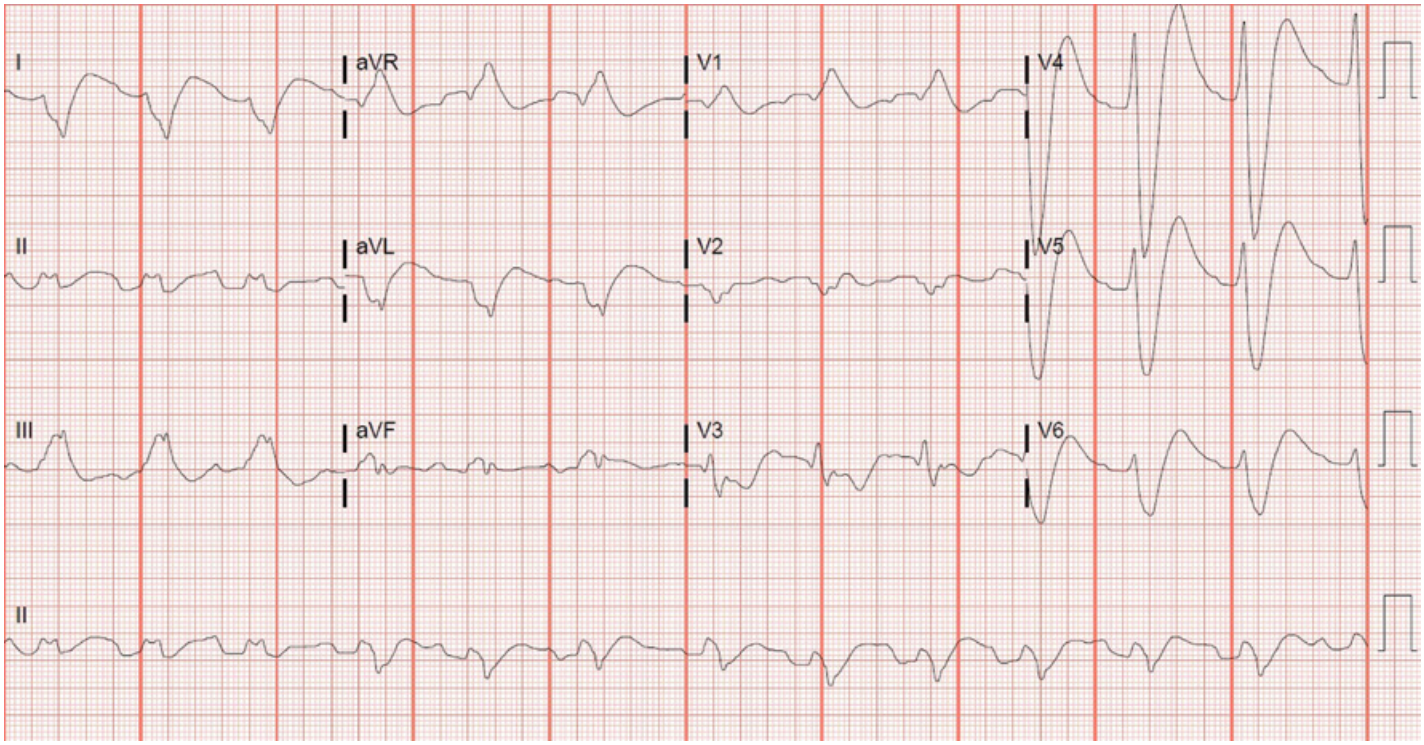
You are working a busy overnight with your senior resident and attending when a broken patch comes through on the radio:

We are en route with a 30-year old female in cardiac arrest…history of heart problems…ET tube secure, 5 rounds of epi…See you in 4 minutes.

Realizing the gravity of cardiac arrest in such a young patient, your attending readies the room for a prolonged and vigorous resuscitation.

On arrival, the patient is being actively coded with chest compressions and endotracheal ventilation. A history is given by emergency medical services (EMS) personnel that the patient was potentially down for approximately 20 minutes before being found by family. As you review the electronic medical record, a handful of pill bottles are given to you that were found on scene, including flecainide and ondansetron. A quick review of the chart shows a previous history of peripartum cardiomyopathy, history of pulmonary embolism (PE) no longer on anticoagulation, and bipolar disorder. With that information in mind, you continue the resuscitation.

IMAGE 1. EKG Performed in the ED



Clinical Course

Over the next 2 hours, the patient goes in and out of wide complex arrhythmias, including torsades de pointes and monomorphic ventricular tachycardia. Throughout this time you have maintained concern for massive PE given her history, but also electrolyte derangements and prolonged qT syndrome given the additional information of the anti-emetic. Overdose is also a major concern. Point-of-care labs during the resuscitation were significant for hypokalemia and acidosis. Return of spontaneous circulation (ROSC) is achieved and lost repeatedly amidst multiple rounds of epinephrine, magnesium, potassium, and defibrillation. Bedside ultrasound is essentially unremarkable save an enlarged heart, with no evidence of right ventricular strain, tamponade, or aneurysm. The family is insistent she was not suicidal, but given her heart's unresponsiveness to every medication in the code cart, you assume the worst and begin treatment for either accidental or intentional overdose. At this point you are able to achieve ROSC long enough to obtain an electrocardiogram.

About Flecainide Poisoning

Flecainide is a Vaughan Williams Class 1C antiarrhythmic. It works as a sodium channel blocker to prolong depolarization of myocytes and thus inhibit ventricular ectopy.[1] As a sodium channel blocker, overdose can produce a characteristic EKG with a prolonged QRS, and a rightward axis which manifests most classically as a terminal R wave in aVR; ventricular tachycardia is also characteristic.[2] QRS is usually 50% wider, PR is 30% prolonged, and QTc is 15% prolonged, producing an EKG characteristic of flecainide toxicity in particular.[3] Clinically, toxicity can range from nausea and headache, to seizures and malignant dysrhythmias, to complete cardiovascular collapse.

Treatment includes advanced cardiac life support, gastrointestinal decontamination, and aggressive administration of sodium bicarbonate. As with tricyclic overdose, sodium bicarbonate probably works to offset the sodium channel blocking effects of the drug, in addition to alkalinizing the serum and thus increasing the protein bound portion of flecainide as well as helping to dissociate the drug from myocytes.[4] Refractory cases are not well studied in the literature, but a perusal of prior case reports reveals that frequent sodium bicarbonate boluses or infusion, intravenous lipid emulsion (ILE) administration, and extracorporeal membrane oxygenation (ECMO) are the mainstay, and show promising outcomes with the increasing availability of these modalities.[5-8]

Flecainide is a Vaughan Williams Class 1C antiarrhythmic. It works as a sodium channel blocker to prolong depolarization of myocytes and thus inhibit ventricular ectopy.

Intravenous Lipid Emulsion Therapy

ILE is thought to work via a twofold mechanism: acting as a "lipid sink," thus sequestering the drug within the emulsion, and as source of fatty acids for poisoned myocytes.[9] Dosing varies, but the American College of Medical Toxicology recommends a loading dose of 1.5 mL/kg pushed over 2-3 minutes, which can be repeated, followed by a 0.25 mL/kg/min infusion over an hour.[10] Maximum dosing is approximately 10-12 mL/kg.[11]

The adverse effects of ILE include Lipid Overload Syndrome which is characterized by headache, fever, liver toxicity, coagulopathy, and anemia.[12] It also interferes with lab analysis and can cause spurious results on both blood counts and electrolytes, so efforts ought to be made to collect labs prior to administration.[13] In addition, ILE is associated with fat deposition and clotting in ECMO circuits, leading to circulatory machine failure; however, ILE is not an absolute contraindication to ECMO.[14] When choosing treatment modality for a poisoned patient, clinicians have to take into consideration the stability of patient and availability of ILE and ECMO in the hospital. Finally, there is some evidence that ILE alters the hemodynamic response to vasoactive agents, and patients in shock receiving ILE may need higher-than-expected doses of vasopressors to achieve goal blood pressures.[15]

Case Conclusion

The patient was given a 1.5 mL/kg bolus of ILE twice, resulting in near instantaneous resolution of wide complex into a sinus rhythm. An infusion of the drug was started along with maximized vasopressor support, and the patient was transferred to the cardiac intensive care unit in stable condition but with a guarded prognosis. Unfortunately, the patient had already suffered devastating anoxic brain injury prior to the resuscitation, and care was withdrawn later on hospital day 1, with the patient quickly dying thereafter.

We had drawn a flecainide level in the ED just prior to giving the ILE, which resulted later as a whole blood concentration of 2.01 mcg/mL, more than double the upper limit of normal. Family who had later brought the patient's pill bottles from home noted only 10-15 tablets of flecainide missing, which is suggestive of the drug's narrow therapeutic window.

TAKE-HOME POINTS

Flecainide overdose is a rare but serious toxic ingestion. Antiarrhythmic overdose should be considered in refractory cardiac collapse in otherwise healthy individuals or in those with access to these medications, in addition to anyone with a characteristic ECG. Treatment includes the aggressive administration of sodium bicarbonate and a low threshold to initiate ILE or ECMO.✶

References available online

Fixing a Cuff Leak

Learning to Improvise with Airway Problems

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A 59-year-old male with a history of Type II diabetes and hypertension presents to the emergency department with greater than 50% body surface area burns, shortness of breath, and stridor after being trapped in a confined space on fire. The patient has third-degree burns of his entire face, scalp, and anterior neck. He is obtunded, tachycardic, tachypneic with respirations of 28-32, and marked stridor on inspiration, as well as an oxygen saturation of 90% on non-rebreather face mask at 15 LPM. On examination of the mouth and nose there are singed nasal hairs, erythematous oral mucosa with secretions, and soot visible in the posterior oropharynx.

The decision is made to emergently intubate the patient as a result of his inability to protect his airway, respiratory distress, and anticipated swelling of his airway secondary to burns. Equipment is prepared, medications are drawn up, the patient is successfully preoxygenated with oxygen saturations approaching 99%, and other interventions for effective burn resuscitation are completed. After appropriate RSI medications are administered a senior resident attempts intubation but encounters difficulty because of profound soft tissue swelling in the posterior oropharynx. An experienced attending is able to successfully place in the endotracheal tube (ETT), with position confirmation by equal bilateral breath sounds, quantitative waveform capnography, and chest X-ray.

Several minutes later, the team is called back to the bedside by a respiratory therapist who has identified a substantial cuff leak of the ETT resulting in inadequate ventilation of the patient. A quick evaluation of the tube reveals a damaged pilot balloon line causing cuff deflation. The team is concerned about removal of the tube, even using bougie, Aintree, or other device to exchange the tube, given the difficulty with initial intubation and severe, progressive soft tissue swelling.

What can be done to repair the pilot line?

Literature Review

A malfunctioning ETT pilot balloon or pilot line (either incompetent valve, damage to balloon, or damage to the pilot line) might lead to inability to ventilate because of a large leak. Changing the ETT can prove challenging in certain clinical scenarios (eg, full stomach patient, difficult airway, or prone position). Being able to troubleshoot the problem and avoid changing the ETT decreases the risk to the patient and saves time.

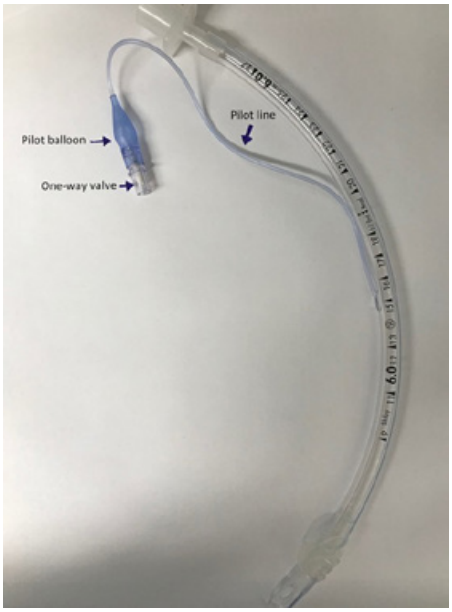

FIGURE 1. Anatomy of an endotracheal tube

Cuff defects top the list of structural causes of air leak from endotracheal tubes.[1] Damage to the cuff during tube insertion, especially during multiple intubation attempts, on teeth or hardware in the mouth is often the culprit.[2] Also, damage can occur as a result of inadvertent contact with other medical equipment such as needles, scalpels, or forceps.[3] Even application of local anesthetic to the cuff material has been implicated in cuff defects sufficient to cause a leak.[4] If the cuff itself is damaged, the only viable solution to reestablish a secure airway is to change the tube.[1] However, damage to the pilot line or pilot balloon may be repairable without having to exchange the tube.

Cases of pilot balloon and pilot line failure, although rarer, also have been reported previously in the anesthesia, critical care, and EM literature. The one-way inflation valve of the pilot balloon can be incompetent as a result of poor manufacturing, mechanical trauma, or even routine use of connecting an air syringe to inflate the cuff.[5] The pilot balloon, much like the cuff, can be torn, punctured, or otherwise damaged on teeth or sharp equipment, causing leakage of air or failure to inflate altogether.[6,7] Likewise there have been reports of the pilot tubing being damaged by tube securing devices, accidently cut, or faulty manufacturing.[8,9]

Other techniques for pilot line and pilot balloon repair have been described, but often require specialized equipment. Ideally, one would be able to acquire a commercially available repair kit.[10] However, these are not readily stocked in every ED. Rao et al. described a novel, improvised way of fixing an ETT pilot line with a Portex epidural connector.[11] While anesthesia colleagues may have

easy access to this device, it is not a common piece of equipment in the ED. Other techniques such as using an IV catheter and stopcock setup[11,12,13] or even a hypodermic needle in various configurations[14,15] have been proposed. These solutions may be more feasible given the equipment available to prehospital and ED providers.

Techniques in the ED or Prehospital Environment

Here are 4 easy techniques with equipment readily available in any ED, operating room, intensive care unit, or ambulance to fix the leak and avoid changing the ETT.

Situation No. 1. The one-way valve malfunctions, but the pilot balloon and line are intact.

Attach a T-piece connector or a clave to the pilot balloon, and then inflate with a syringe. Clamp the T piece with a hemostat or IV tubing clamp, and disconnect the syringe.

Situation No. 2. The pilot balloon or pilot line is ruptured.

Cut the line below the break. Insert either a blunt needle or a 22G catheter into the lumen of the line. Connect it to a syringe and inflate the cuff. Clamp the line, and cover needle or catheter with a transparent film dressing or clave.

Case Resolution

A resident on the team quickly grabs a 22 gauge IV catheter, clave, 10 cc syringe, and Tegaderm™ from the IV access cart in the resuscitation room. She quickly identifies the site where air is leaking from the pilot line. With her trauma shears, she cuts the line just beyond leak, proximal to the cuff. Then, she removes the plastic 22 gauge catheter from the IV needle and inserts it snugly into the open end of the pilot line. A clave is attached to the end of the catheter and a 10 cc syringe is connected. The cuff is then successfully reinflated, with cessation of the air leak and return of effective ventilation. Finally, the Tegaderm™ is wrapped around the pilot line, catheter, and clave to ensure an airtight connection.

TAKE-HOME POINTS

- Cuff leak from damage to an ETT cuff, pilot line, pilot balloon, or valve can cause inadequate ventilation and presents a significant patient safety issue with an unsecure airway.
- While damage to the ETT cuff itself necessitates replacement of the tube, equipment failure proximal to cuff in the pilot line, pilot balloon, or valve can be easily fixed.
- A faulty valve can be repaired with a clave for IV extension tubing.
- A leaking pilot line or pilot balloon can be repaired by first cutting distal to the site of damage, then using a conduit such as a 22 ga catheter or blunt needled to reinflate the balloon, and finally prevent egress of air by clamping or otherwise occluding the pilot line.✶

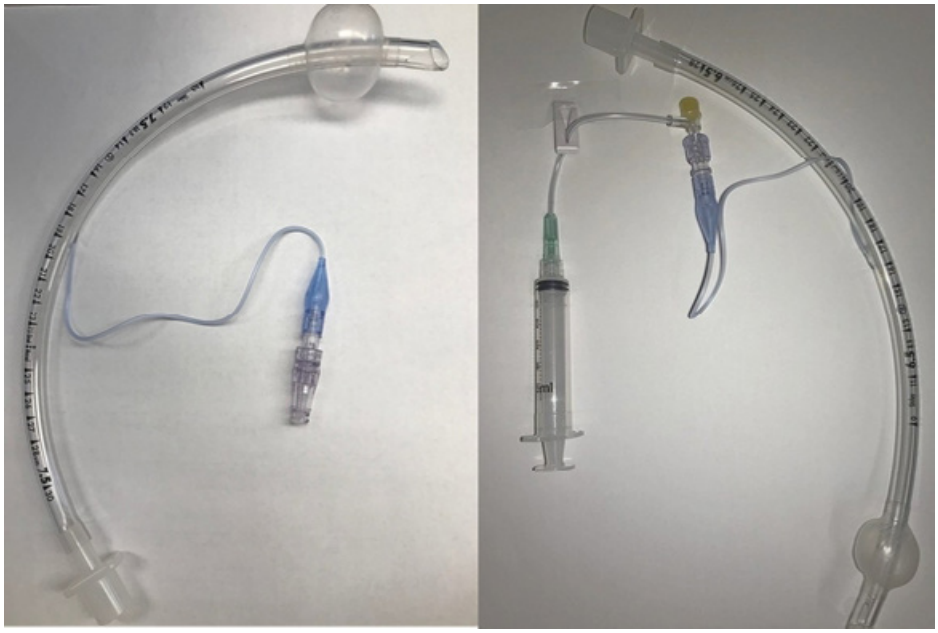

FIGURE 2 (left) showing clave technique and **FIGURE 3** (right) showing IV extension tubing technique for repairing a faulty pilot balloon valve.

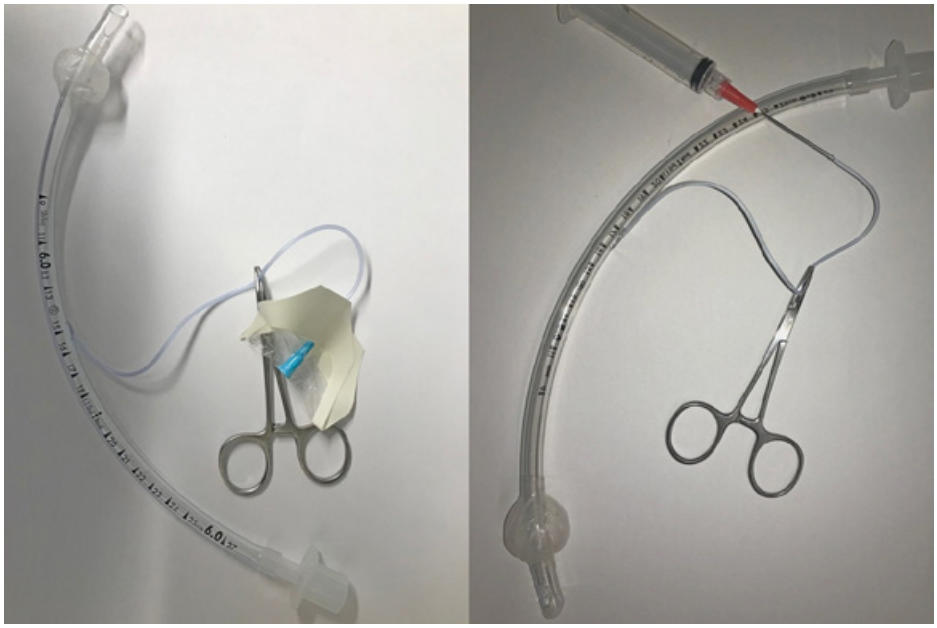

FIGURE 4 (left) showing catheter technique and **FIGURE 5** (right) demonstrating needle technique for damaged pilot balloon or pilot balloon line

References available online

Early Aggressive Whole Bowel Irrigation

Prevent Severe Lithium Toxicity After Acute Overdose

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A 22-year-old male with a history of bipolar disorder presents to the ED, saying he ingested 60 lithium carbonate extended release tablets (450 mg each) in a suicide attempt 30 minutes ago. He reports no symptoms, and his physical exam is unremarkable. Initial laboratory analysis includes serum lithium (0.6 mmol/L), serum sodium (140 mEq/L), and serum creatinine (0.97 mg/dL).

Whole bowel irrigation is initiated in the ED, and the patient is admitted to the intensive care unit for further observation and management.

Discussion

Lithium is a very commonly prescribed agent for the treatment of bipolar disorder and acute mania. Despite its narrow therapeutic window, it is still considered a first line treatment for bipolar disorder and acute mania. In 2016, 6,901 cases of lithium intoxication were reported to the American Association of Poison Control Centers.[1] For this reason, it is important for emergency physicians to understand how to approach the patient with acute lithium toxicity.

Lithium's mechanism of action is poorly understood. Its effects on decreasing intracellular inositol monophosphate are theorized to contribute to its mood stabilizing properties. It also inhibits glycogen synthase kinase-3, which is known for its effects on neuroplasticity, neuroprotection, and energy metabolism.[2]

In a patient with suspected acute lithium poisoning, a serum lithium level should be obtained upon initial presentation. The therapeutic range for lithium is 0.8 to 1.2 mmol/L. It should be noted that many patients take extended release formulations of the drug and thus the initial serum lithium level may not reflect the amount ingested.

Clinical Signs

Clinical presentation of acute lithium poisoning often manifests with gastrointestinal symptoms including nausea, vomiting, and diarrhea. Few reports also have displayed adverse cardiovascular events including arrhythmia, prolonged QTc, and bradycardia.[3] Neurologic findings are late to develop in acute poisoning and manifest as ataxia, agitation, tremors, seizures, encephalopathy, and lethargy. Of note, clinical findings often do not correlate with serum lithium levels. Neurologic findings indicate that the drug has had time to be absorbed and penetrate the CNS, so it is critical to treat these patients aggressively before they manifest.

Treatment

Hemodialysis is the definitive treatment for severe lithium toxicity because of its low molecular weight, low protein binding, and small volume of distribution. Hemodialysis is indicated in patients with serum lithium levels >4.0 plus evidence of renal impairment and/or the presence of decreased level of consciousness, seizures, or dysrhythmias.[4]

However, hemodialysis can be complicated to initiate due to the need to place a central dialysis catheter and have close nephrology consultation.

Long-term neurologic complications have been described in the literature despite lithium removal by hemodialysis. A syndrome of irreversible lithium effectuated neurotoxicity (SILENT) is characterized by cerebellar dysfunction, brainstem dysfunction, extrapyramidal symptoms and cognitive impairment. The neuropsychiatric sequelae of SILENT can persist for years after an acute poisoning.[5]

In patients who present with early lithium poisoning, aggressive whole bowel irrigation offers a viable option to avoid short- and long-term neurologic manifestations of lithium toxicity. It is a non-invasive and effective method to prevent lithium absorption in patients who present within two to four hours of ingestion following ingestion of an extended-release formulation.

Case Conclusion

A total of 10 serum lithium measurements were obtained over 19 hours. Serum lithium concentration increased from 0.6 mmol/L upon presentation to a peak of 2.1 mmol/L 12 hours later.

The timing of the patient's peak concentration at 12 hours aligns with pharmacokinetic data for extended-release lithium ingestion. The patient's peak lithium concentration was significantly lower than would be predicted from his self-reported ingestion of 27 grams of lithium carbonate. This suggests that early gastrointestinal decontamination may be sufficient in preventing systemic lithium toxicity. The possibility of the patient over-reporting total lithium ingestion should be considered.

The patient received 500-1000 mL/hour of polyethylene glycol until rectal effluent was clear 12 hours after presentation. Serum lithium levels were obtained every 2 hours for 19 hours, at which point lithium levels consistently down-trended.

The patient remained asymptomatic and without signs of lithium toxicity throughout his hospitalization; he was discharged on Hospital Day 4.

Key Takeaway

Hemodialysis is the definitive modality of lithium clearance in severe toxicity. In early overdose, however, whole bowel irrigation may prevent lithium absorption and subsequent toxicity if mental status is preserved. ✶

The Elusive Foreign Body

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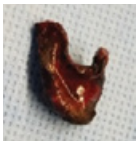
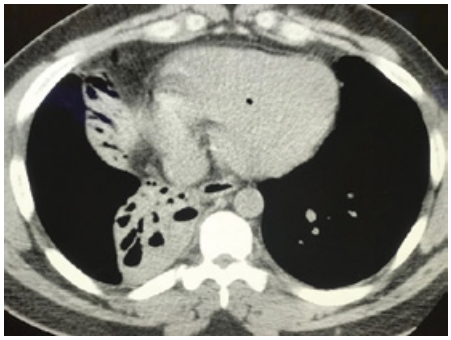


A 29-year-old male with history of pneumonia presents to the ED with complaint of cough. The patient reported he dropped a box he was delivering, releasing a cloud of white powder. The patient states he inhaled the material just prior to arrival in the ED. The box was believed to contain a stove cleaner called "Bar Keepers Friend Cleaner and Polish." The patient reported pleuritic chest pain, cough, dizziness, diaphoresis, nausea, and epigastric abdominal pain following exposure. He has no other pertinent medical history but mentions 2 prior hospitalizations at an outside hospital for pneumonia within the past month.

Physical exam is notable for right lower lobe rhonchi but otherwise unremarkable. Basic labs are only notable for a mild leukocytosis of 12000/mm3 without bandemia. Chest x-ray shows a right lower lobe infiltrate. CT imaging shows a severely narrowed and occluded bronchus intermedius with collapse of the right lower lobe. Bronchiectasis and/or cavitary changes in the atelectatic right lower lung are seen. In addition, a hyperdensity suspicious for foreign body is noted in the right lower lobe.

These findings are discussed with pulmonology, who complete a bronchoscopy that reveals a small object, thought to be a piece of bone, removed from the right lower lobe.

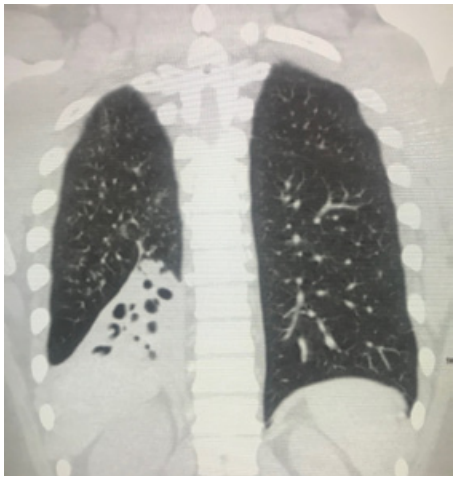


The patient initially fails extubation immediately following the bronchoscopy and is subsequently transferred to the ICU, where he is treated for aspiration pneumonia with antibiotics and nebulizer treatments. After 3 days, he is weaned and extubated. Review of EMR indicates the patient has not had subsequent ED visits or hospitalizations for pulmonary complaints.

Discussion

Foreign body aspiration is a frequent complaint in the ED. In adults, it is most frequently seen in the elderly and in those with underlying risk factors such as neuromuscular disease, altered mental status, intoxication, and trauma.[1] Symptoms are usually acute in onset and involve historical and physical evidence of airway obstruction such as cough, difficulty swallowing, or stridor. However, foreign body aspiration may also have a more indolent course with serious and life-threatening complications.[1,2,3,4]

The patient in this case was initially brought to the ED for suspected inhalation injury. Poison Control reviewed the contents of Bar Keepers Friend and doubted the symptoms were related to significant inhalation injury. The patient denied any recent history of choking or ingestion foreign bodies. An initial CXR was concerning for an infiltrate in the right lower lobe. Although retained foreign body was low on the differential initially, the exam and CT findings were highly suspicious for pulmonary bronchiectasis, atelectasis, and pneumonia as a result of a possible foreign body.[2]



The diagnosis of foreign body can be difficult to establish in patients without a clear history, or when presentation occurs weeks to years after the initial aspiration event. While it remains unclear how long this patient's retained foreign body had been present, he mentioned 2 previous admissions at an outside institution for pneumonia in the past month. Cough, fever, and recurrent pneumonia are the most common symptoms in delayed diagnosis of foreign body.[2,3]

When diagnosis is significantly delayed, patients may develop significant bronchiectasis, bronchial stenosis, asthma, mucosal edema, or recurrent hemoptysis.[3] A surgical lobectomy may be warranted if extraction of foreign body is unsuccessful or when sequela of foreign body aspiration has resulted in extensive damage to pulmonary tissue.[3]

Consider a delayed presentation of foreign body aspiration in those presenting with chronic cough and recurrent pneumonia. Radiologic findings include pneumonia, bronchiectasis, and atelectasis, but may not show evidence of obvious foreign body, but bronchoscopy can be both diagnostic and therapeutic.

PEARLS

- While foreign body aspiration is often seen promptly after initial event, delayed diagnosis of foreign body should be considered in patients with recurrent cough and/or recurrent pneumonia that has been unresponsive to antimicrobial therapy.
- Undiagnosed and retained foreign bodies may result in serious complications such as pneumonia, bronchiectasis, and atelectasis.
- Removal of retained foreign body requires bronchoscopy for both diagnosis and treatment. If extraction is extensive or pulmonary disease is severe, surgical evaluation may be necessary. ✶

References available online.

From Dizzy to Torsades de Pointes

Afrah A. Ali, MBBS
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University of Maryland School of Medicine

A 59-year-old female presents to the ED complaining of dizziness and unsteady gait that started a day ago. She was found lethargic and confused by her family. Further information was unavailable because of the patient's altered mental status. Past medical history includes diabetes and hypertension; medications are insulin, amlodipine, and gabapentin. Vital signs are HR 48 bpm, blood pressure 224/100 mmHg, respiratory rate 18, temperature 36.8 C, and oxygen saturation 98% on room air. On physical examination, patient is lethargic and disoriented. There is loss of nasolabial fold on the right side with decreased motor strength 4/5 in the right upper and lower extremity. Patient also has slurred speech.

Initial EKG showed sinus bradycardia with heart rate 48 bpm, Qtc 498 ms, no acute STE, STD, TWI noted. (Figure 1).

One dose of 20 mg of labetalol via IV was given, for suspected hypertensive crisis with intracranial pathology (ischemic vs. hemorrhagic stroke). Workup included CBC, CMP, EKG, chest XR, CT and CTA head and neck, coagulation panel, ammonia, thyroid profile, urine drug screen, urinalysis, and alcohol level. While in the ED, patient experienced an episode of torsades de pointes, with loss of consciousness, that lasted for 20-30 seconds (Figure 2).

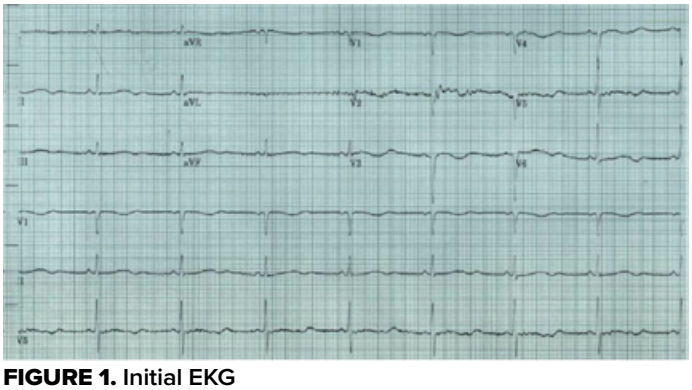

FIGURE 1. Initial EKG

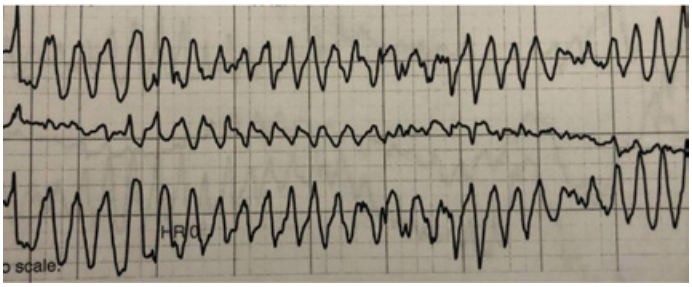

FIGURE 2. Torsades de pointes

Repeat EKG showed heart rate 65 bpm, with prolonged QTC of 628 ms with ventricular bigeminy (Figure 3).

Metabolic panel revealed electrolyte abnormalities: hypokalemia with a potassium of 2.6 mmol/L, hypomagnesemia with magnesium of 1.4 mg/dL, and hyperglycemia with glucose of 486 mg/dL. Urine drug screen was positive for methadone, and urinalysis was positive for leukocyte esterase with white blood cells > 50/hpf. Patient was given 40 mq potassium and 2 g magnesium bolus IV. She had another short episode of Tdp with loss of consciousness.

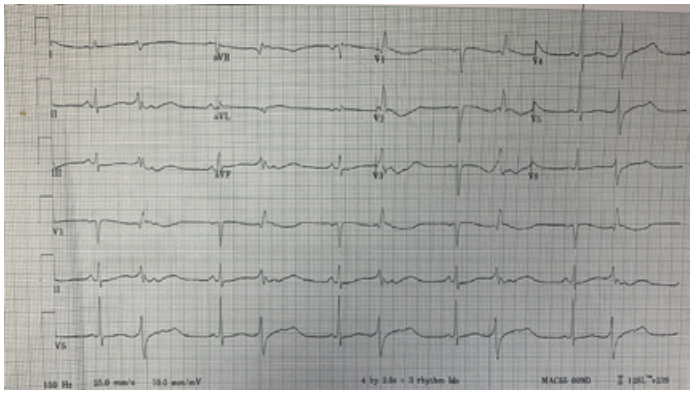

FIGURE 3. Repeat EKG

Another 4 g of magnesium was given. Isoproterenol was unavailable, so transcutaneous overdrive pacing was attempted. Mechanical and electrical capture was obtained with heart rate 110 bpm at 80 mA. CTA head and neck showed complete occlusion of the left middle cerebral artery at the M1 branch with an Acute to subacute infarct in the left frontal, caudate, and basal ganglia (Figure 4).

Findings were consistent with MCA distribution infarct. The patient was transferred to a tertiary hospital for further treatment and admission to cardiac intensive unit.

Discussion

Torsades de pointes is a type of polymorphic ventricular tachyarrhythmia in which the QRS complexes appear to twist around the isoelectric line. It is universally associated with congenital or acquired long QTc syndrome (LQTS).[1] Congenital LQTS has been found to be associated with 1 of 6 identified mutations in genes coding for cardiac ion channels (LQT 1–6).[2]

The most common cause of torsades de pointes is acquired prolongation in the QT interval because of medication. Acquired

QT prolongation has been the single most common cause of withdrawal or restriction of drugs' use after they have been marketed in the past decade.[3]

Multiple risk factors are implicated in prolonged QT interval progressing to torsades de pointes:[4-7]

- Hypokalemia
- Female sex
- Hypomagnesemia
- Digitalis therapy

The prolongation of the QT interval to longer than 500 ms during drug therapy should prompt a critical re-evaluation of that therapy and consideration of therapeutic alternatives. Many drugs are known to cause QTc prolongation, including chlorpromazine, metoclopramide, atypical antipsychotics, thioridazine, TCAs, macrolide antibiotics, methadone, lithium, and antiarrhythmic drugs (class IA, IC, and III).

Magnesium sulphate suppresses torsades de pointes by decreasing the influx of calcium ions, which in turn results in decreased amplitude of early afterdepolarizations.[8] The initial dose is 2 g (20 mL of 10% solution), given IV over 1-2 minutes. This can be repeated up to a total of 6 g, with 5–15 minutes between doses. Following a bolus of 2-4 g of magnesium, very little magnesium is absorbed into cardiac tissue. Most is excreted in the urine, and unless the patient has severe renal failure, magnesium levels will drop back to baseline over next several hours.[9] Hence, it's preferred to follow the bolus with a magnesium infusion at a rate of 1-2 g/hr.

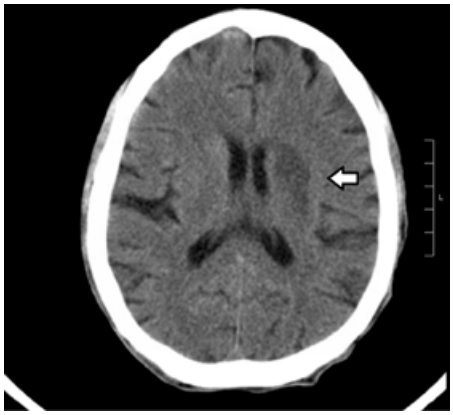

FIGURE 4. Imaging of the head and neck

In some circumstances, pharmacologic pacing (using isoproterenol) or transvenous pacing may be necessary to suppress arrhythmias by increasing the baseline heart rate to 100-150 bpm.[10-11] Temporary transcutaneous pacing has also been successfully used to convert torsades de pointes.[12] IV lidocaine, a Class IB antiarrhythmic that shortens QT-interval, can also be used. An acute episode prolonged enough to cause hemodynamic compromise is treated with unsynchronized cardioversion, beginning with 100 joules.

As in our case, it is important to remember that isoproterenol may not always be available, and mechanical pacing may be necessary. Targeting higher magnesium serum levels with magnesium infusion and IV lidocaine can be life-saving.✶

References available online.

Physician-Led Health Care

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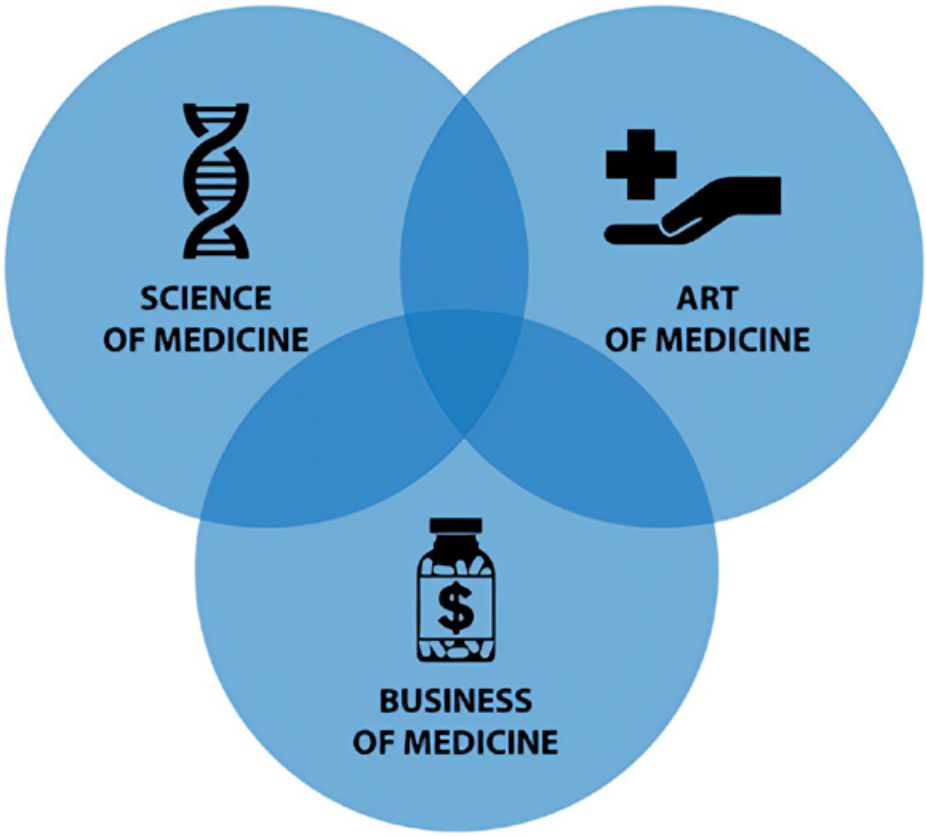

In the thousands of U.S. hospitals, only 3-5% of hospital leaders are physicians.[1,2] This is a change in paradigm from the historical origins of establishing hospital systems. Originally, hospitals were founded by physicians with the help of wealthy and influential sponsors; fast forward more than a hundred years, and the trend of health care physician leaders has moved in the opposite direction. More hospitals are opening and the physician workforce is increasing, but **the proportion of leadership roles occupied by medically trained professionals is decreasing**.[3]

The importance of the business of medicine becomes more apparent as we move farther into our medical careers. Medical practice is a triad of science, art, and business, and in our pursuit of improving the world through medicine, we gain understanding of all three. We spend 4 years in medical school learning the science of medicine and 3+ years in residency to master the art of clinical practice, but we receive little education on *operational* practice.

In residency, exposure to ED operations management, business administration, patient safety, and quality improvement is frequently limited to eye-roll-inducing Press Ganey scores, metrics of patients per hour, or sepsis bundle checklists. One could easily argue against the utility of some of these metrics. Notably, an interesting nuance of Press Ganey scores is that they are only recorded for patients who are discharged from the ED. This represents a flaw, as those patients have lower acuity and thus endure longer wait times and receive the least attention (both associated with poor Press Ganey scores) while attention and time are devoted to critical patients fighting for survival.[4] Hence, other methods of measuring the satisfaction of patients treated by emergency physicians, such as Emergency Department Patient Experiences with Care, are currently being pursued.[5]

Simple metrics such as patients per hour are also not without problems. For example, a normal patient with absolutely no pathology might actually require more time and resources of an ED — extended monitoring, thorough diagnostic testing for atypical presentations, and repeat examinations, in order to determine if the patient is safe for discharge. Contrast this with a sicker patient, such as a triage-identified STEMI, who would be in and out of the ED on their way to a cardiac catheterization lab within minutes. Similarly, the safety of the sepsis bundle, which mandates that patients receive a 30 cc/kg fluid bolus and early broad-spectrum antibiotics, is being questioned, as not all that triggers SIRS criteria is bacterial infection.[6] Numbers such as these are placed on spreadsheets and are then connected to compensation rates, such as CMS reimbursement, to affect clinical practice.[7]

Without having on-the-ground clinical experience, non-medical professionals in the boardroom can have difficulty understanding the intricacies of working at the bedside. Early exposure to such operational dilemmas during residency trains us to balance guidelines aimed at the population level with the reality of serving patients on the front line.

Same Skills, Different Applications

Emergency physicians practice business skills and principles on a daily basis without recognizing them as such, perhaps because it's framed in medical terminology rather than business terminology. For instance, the phrase "*negotiating a contract*" seems foreign and daunting considering we never negotiated a salary for residency or bargained the tuition for schooling, but we employ the concepts of negotiation every day in the department. We conduct shared decision-making to develop disposition plans with consultants, families, and patients; to phrase it another way, we "*negotiate the terms*" of safe discharge in order to develop a plan of care in which all parties are in agreement. Another foundational example that we practice routinely: when we call radiology to bump a critical, time-sensitive patient up first for imaging, such as a stroke protocol potential thrombolytics candidate patient, in business terms, we are allocating resources and leveraging the assets of the department.

Many administrative issues can be alleviated through similar problem-solving analytical reasoning strategies that we have been developing through the years. During winter, many EDs suffer from boarding, which is a known phenomenon that increases morbidity.[8] Is there someone in the waiting room who isn't being seen immediately because of the lack of bed availability who might undergo unrecoverable pathological damages in the next hour while waiting? Framing this issue as an attending at our institution likes to put it, boarding is essentially a congestive heart failure exacerbation of the hospital; the solution is to diurese the hospital floor census and prevent flash pulmonary edema of the ED.

Developing operational and business competency is an integral element of residency training. With thousands of hours of training to develop clinical acumen, we can manage life-altering complex medical issues such as severe traumatic brain injury flawlessly, but when it comes to managing complex financial issues that affect patients in the ED, we are sometimes underprepared. From a quantitative perspective, a small ED can produce annual revenues of $1 million; similarly, helping to run a large ED means being responsible for the operations of a business that could be producing $10 million of revenue per annum.[9] Being involved in these managerial decisions and optimizing health care is our obligation to our patients.

Where to Learn Administrative Skills

There are many outlets and opportunities to get involved and gain further proficiency during residency training. Depending on program curriculum, residents can choose to complete an administrative elective rotation and run their own project to solve a problem identified in their department. Residents can also learn about a broad range of topics by attending courses and conferences at a local, regional, or national level. One such conference is the Emergency Department Directors Academy (EDDA), sponsored by ACEP, a development course to introduce medical directors and physicians to the foundations of managing an ED. Dr. Robert Strauss, the chief editor *Strauss and Mayer's Emergency Department Management* and program chair of the EDDA, stated, "It would be wonderful if more residents took advantage of the opportunity to attain leadership skills along with the medical directors who attend. We have the best of the best among EM leadership educators, and they are passionate in their desire to share these skills." Residents can apply for a travel scholarship through EMRA to travel and attend this conference for free.

Residents can also get involved on national resident committees, such as EMRA's Administration and Operations Committee, which furthers resident interest in the subject and encourages residents to add operational management to their fund of expertise.

It is imperative that we as residents focus on being excellent clinicians but to the exclusion of other facets of medicine. **Learning about strategies of running an effective ED, addressing the needs of the department, negotiating patient interests, and dealing with complex departmental issues all lead residents to develop leadership skills.** Through the business of medicine, we can fulfill the physician's oath to do right by our patients and become the best physicians we can be. ✶

References available online

All About EMTALA
The Law that Runs the ED

Eric Maughan, MD
Maine Medical Center

Enacted by Congress in 1986, the Emergency Medical Treatment and Labor Act (EMTALA) was designed to provide emergency care to all patients, regardless of insurance status or ability to pay. But some of the unintended consequences of EMTALA have not been as positive.

EMTALA defines 3 responsibilities of participating hospitals (defined as hospitals that accept Medicare reimbursement):[1]

1. Provide all patients with a medical screening examination (MSE)
2. Stabilize any patients with an emergency medical condition
3. Transfer or accept appropriate patients as needed

Responsibilities of Hospitals and Providers

1. Medical Screening Examination

According to EMTALA, all patients, regardless of insurance status, nation of origin, race, religion, etc., are entitled to an MSE if they are on a "hospital campus" (within 250 yards of a hospital building). The purpose of the MSE is "to determine whether or not an underlying emergency medical condition exists."[2]

2. Stabilization

If the MSE reveals an emergent condition, EMTALA mandates that the hospital stabilize the patient, meaning you are reasonably sure they can be transferred or discharged without clinical deterioration. Of course, stabilizing a patient often requires other consultants, which means the EMTALA requirements — and penalties — extend to them as well.

3. Transfers

EMTALA also requires that hospitals perform an "appropriate transfer" to a higher level of care if required by the patient's condition. To satisfy this aspect of the law, the transferring hospital must treat and stabilize the patient to the fullest extent of its resources, provide care en route, contact the receiving hospital (who has agreed to accept the patient), and transfer the patient with appropriate copies of medical records. (Transferring a patient without copies of the medical record, including imaging, is an EMTALA violation.)[3] Correspondingly, the law mandates that the receiving hospital accept the patient, as long as it has the appropriate resources to care for the patient.

Penalties

EMTALA is tied to Medicare reimbursement, and severe violations can lead to termination of the hospital or provider's Medicare Provider Agreement. Fines can reach $100,000 per violation, and hospitals may be held liable for civil lawsuits, either from patients or from transferring or receiving hospitals.

Consequences

The law has ensured that all patients can receive the emergency care they need, which has transformed the ED into society's de facto safety net. Our specialty's ability to treat anyone, with anything, at any time[4] fits EMRA's position that "all individuals should have access to quality, affordable primary and emergency health care services."[5] Unfortunately, these benefits have not come without cost.

U.S. hospitals provided $38.4 billion in uncompensated care in 2017.[6] That number decreased slightly after passage of the Affordable Care Act (ACA), but has worsened since then. The ACA was designed to eliminate uncompensated care, but the reluctance of some states to expand Medicaid has hampered those efforts, and states that have expanded Medicaid have seen a bigger drop in uncompensated care than those that haven't.[7]

Resistance to Medicaid expansion is not the only threat to hospital finances due to uncompensated care.[8] Many insurers are now implementing policies to reduce reimbursement for ED visits retroactively deemed to be non-emergent. In an age where 40% of health insurance plans are considered high deductible,[9] this policy threatens to increase the amount of care that hospitals (and particularly EDs) provide without reimbursement.

Hospitals have been closing at alarming rates across the country, especially in rural America. Many have cited uncompensated care as a cause of hospital closures.[10]

Bottom Line for EM Residents

Emergency physicians treat any patient with any emergent condition regardless of other factors, as EMTALA mandates. Stabilizing and transferring patients appropriately are aspects of the law, and should also be aspects of good patient care. But while EMTALA has helped our patients and society, it has also put a significant financial burden on hospitals. We need to keep pushing against policies that increase uncompensated care, so that we can continue to care for any one, with anything, and any time. ✶

References available online

What is Informatics?

P.S. It's Not Just for Nerds

Fran Riley, MD
EMRA Assistant Vice Chair of Informatics

Informatics has been a burgeoning field for decades, and it's time for even more people to join!

I know what you're thinking. "*Informatics… Isn't it boring? Isn't it for nerds and programmers?*"

The answer is no to both — hear me out.

Many people avoid informatics thinking it requires programming or technical experience, but like any worthwhile effort, the most important aspect is a passion for the cause. In nutshell, informatics is using data to gain insight, and hopefully improve things. This can be applied to anything, including advocacy, resident education, wellness, health policy, simulation, administration. I want to illustrate this with my own personal cause: patient safety (really, equality).

While in graduate school, I was working on research at a tertiary pediatric ICU, specifically optimizing the care of pediatric traumatic brain injury (TBI). I was particularly struck by the significant proportion of TBIs caused by non-accidental trauma. Data varies, but approximately 21-33% of patients admitted for TBI were injured by non-accidental causes. Children from age 0-3 are more likely to die from TBI.[1] The quality of care we deliver has lifelong consequences that affect cognitive ability, and therefore happiness, socioeconomic status, and potentially these patients' ability to stay alive at all.

My project essentially took patient data from charts and bedside monitors, and presented it in a consolidated interface. One of the cruxes of this was to graph and trend intracranial pressure data, so that clinicians could be notified of increasing rates of change or when pressure rose to a dangerous threshold. A second aspect was to provide clinical decision support (CDS) based on practice guidelines published by the critical care society. This required reading a 40-page practice guideline, translating it into a flowchart, and linking it to the patient data to help apply the guidelines to the patient's current clinical picture.

To me, this was valuable for two reasons.

1. These patients could achieve the timeliest attention and intervention possible, particularly important in this patient population.
2. By using established expert guidelines across the country, and standardizing care, patients were provided the best opportunity to receive excellent care, regardless of their geographic location or facility.

Of course these are lofty goals, and we are not there yet. However, the point is that informatics is a tool that can help you achieve your passion in medicine, whatever that may be.

Informatics is still in early stages, thus, the benefit is that there is a lot to do within our profession, and much low-hanging fruit. Having completed graduate school with a degree in computer science, people excitedly ask me if I will be integrating Artificial Intelligence or Natural Language Processing (nerd speak for teaching computers to understand human language). I hope medicine gets there someday, but definitely not now. Right now, so many things can be done simply by extracting the data, displaying it in a graph, even by getting notifications to work. In the ED, this is particularly important because we make a lot of high-stake decisions for a large volume of patients, often with split attention.

Overall, informatics is about obtaining data, using it to measure something of interest to you, and acting on that data. If you are working with EMRs, they can be difficult to get data from. However, there are reporting features in major EMRs that can help with this. If not, there is always a team of people who support the EMR at your hospital or by the EMR vendor.

My last parting thought is the list below. These are the informatics-related projects I heard about or discussed with people recently after attending EMRA at CORD. In the next few months, the EMRA Informatics committee will plan to put forth a repository of projects, both new ideas and existing ideas with contacts of people already working on them. We look forward to having more of you join us to change Emergency Medicine with Informatics! ✶

- ✓ **Wellness:** Tracking resident sleep and exercise habits throughout residency. One collaboration between EMRA informatics and EMRA wellness will be to extra insight from the questions asked of residents during the 2019 In-Service Exam.
- ✓ **Workplace Safety:** Investigating discrepancy between security logs vs EMRs in terms of violent patients, and potentially being able to notify future providers at the next visit.
- ✓ **Public Health:** Measuring epidemiological data to determine outbreaks. Finding correlations between diagnosis and neighborhoods to better understand determinants of health.
- ✓ **Consumer:** Detection of arrhythmias (Apple and a few other companies are working on this). Imagine having this data extracted at triage and included as part of your chart.
- ✓ **Clinical:** Giving people a device where they can enter their own history while waiting to see a doctor, including graphical ways to enter pain scale, patterns, etc.!

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My Experience with the EMRA Tactical EMS Scholarship

Gregory N. Jasani, MD
University of Maryland

You're out of the hospital enjoying a day off when suddenly the unthinkable happens in your community: a mass shooting. You find yourself at the scene with dozens of victims, many of whom are critically ill and require immediate assistance. How do you respond?

I got to practice and train for this scenario, thanks to the Tactical EMS Scholarship from EMRA and Mercyhealth. As I was entering my final semester of medical school, I had the honor of being selected for the EMRA Tactical EMS Scholarship. EMRA offers accepted members $500 for travel expenses to attend a tactical EMS course at Mercyhealth in Janesville, Wisconsin. Mercyhealth also waives the registration cost for the course and provides hotel accommodations. There are two courses: the Tactical EMS (TEMS) course (40 hours over 3 days) and the Tactical Emergency Casualty Care (TECC) course (which is 8 hours), which I attended.

Since I had no prior EMS experience, I arrived on the day of the class not entirely sure what to expect. There were 8 of us, including a resident physician, EMTs, firefighters, and police officers from the area. A police officer and a paramedic who both operate in austere and dangerous environments regularly instructed our class. The TECC course focused on providing immediate care to shooting victims in the event that you are the first responder (either intentionally or through circumstance).

I learned how to assess and manage gunshot injuries in the first half of the day. While we as emergency physicians are taught to always go through our ABCs when treating a trauma victim, this course taught me the BATH pneumonic for how to prioritize gunshot injuries:

- Bleeding
- Airway
- Tension pneumothorax
- Hypothermia

BATH is how many first responders approach a gunshot victim on scene. The instructors went through this pneumonic and taught us how to assess and manage injuries within these domains. We learned how to apply tourniquets, pack wounds, place nasopharyngeal airways, perform needle decompressions, place victims in thermal blankets, and even how to use ropes to move victims in the event that you need to rapidly extricate a wounded person from a dangerous area. We had mannequin sessions interspersed throughout our lectures to practice what we just learned.

The second half of the day was the practical portion in which we ran a simulation where we would apply the skills we learned in the morning. We were all given first aid bags filled with the supplies we would need and split into teams of 4.

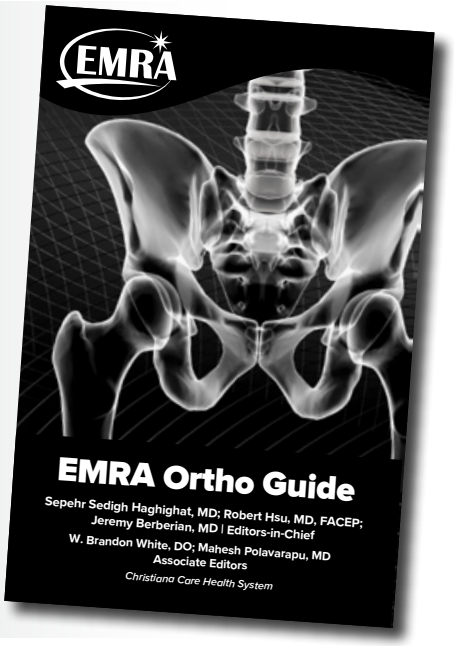
Like most residents, I had been through a lot of simulations in medical school — but all of those assumed I was in the hospital setting, where the loudest stimulus I had to worry about was a beeping monitor. This was incredibly different.

Our simulation started as we entered a very poorly lit hallway and found a mannequin on the ground. Trying to stabilize a patient who was bleeding out in near darkness was unlike anything I'd been asked to do before. After the initial mannequin was stabilized, we moved between 3 different settings: a movie theater, a classroom, and a dance club. Each setting had multiple victims with differing injuries requiring us to use all of the skills learned earlier in the day. Each setting also had different kinds of disorienting sounds: a loud movie, screaming, and loud techno music complemented by strobe lights. The intent of all of this was to completely overwhelm the senses. Fortunately, we were able to run the simulation twice, and I handled the sensory overload better on my second run.

I cannot recommend the TECC scholarship highly enough. It teaches an invaluable skill set that, while I hope to never need, is incredibly important to know. The simulation also forced me to think and act in an environment I had never encountered before. Finally, it gave me even more respect for my EMS, fire, and police colleagues. It occurred to me after the simulation that, while *I* never expect to operate in such a dangerous environment, *they* are all ready, willing, and able to do so at a moment's notice.

I sincerely hope you all consider applying for this wonderful opportunity that EMRA is offering us. Find details at emra.org/awards.✶

New EMRA Guide Helps Manage Orthopedic Injuries



How many times do you encounter musculoskeletal injuries on-shift? If you need a quick refresher for managing those conditions, turn to the brand-new *EMRA Ortho Guide*.

This new pocket reference helps you speak ortho more confidently. The book provides imaging recommendations, treatment options, dispo recommendations, and general pearls for assessing and managing the orthopedic injuries most commonly seen in the ED. Plus, you can review splinting techniques, reduction tips, and key neurologic exam findings.

Many thanks to the **Christiana Care Emergency Medicine Residency Program** for leading the development of this valuable resource! EMRA especially recognizes editors-in-chief **Sepehr Sedigh Haghighat**, MD, **Rob Hsu**, MD, FACEP, and **Jeremy Berberian**, MD; senior editors **Brian Levine**, MD, FACEP, and **Dan Grawl**, PA-C; and associate editors **Mahesh Polavarapu**, MD, and **W. Brandon White**, DO.

The book will be included in EMRA resident member kits and will be available in the ACEP Bookstore and on Amazon. ✶

Keep an Eye on These Future Stars

The EMRA Medical Student Council has announced the inaugural winners of its **National EMIG of the Year Award**, recognizing exemplary emergency medicine interest groups (EMIGs) across the country. Congratulations to these programs!

- **Winner:** Philadelphia College of Osteopathic Medicine
- **1st Runner-up:** Wayne State
- **2nd Runner-up (tied):** Pacific Northwest University College of Osteopathic Medicine and Chicago Medical School of Rosalind Franklin University

These 4 EMIGs won their respective Regional EMIG of the Year title before being considered for national honors.

The EMRA Medical Student Council developed the award because EMIGs represent an essential meeting ground for students, residents, and faculty with a shared excitement for emergency medicine. An effective EMIG provides programming to help students learn more about the specialty, network effectively, navigate the match process, and form relationships with other organizations.

EMRA Medical Student Council Chair Corey McNeilly, MA, said the level of engagement among EMIGs is impressive.

"EMIGs are the primary avenue for students to gain exposure to emergency medicine," he said. "EMRA wants to encourage the amazing programming and work they do by providing recognition on a national scale."

All EMIGs are encouraged to apply for the 2020 title. Applications will be run through the EMRA Spring Awards process, so watch for details at emra.org/awards.

For tips on organizing and running an EMIG, visit emra.org/students/emergency-medicine-interest-groups. ✶

Nominate a Colleague for EMRA's 45 Under 45

As EMRA celebrates its 45th anniversary, we want to celebrate YOU — the young influencers who are shaping the future of emergency medicine by making a difference in your community, your hospital, and our field.

Do you know a rock star who is making an impact — in big or small ways? Someone who is quietly — or boldly — shaping the future of our specialty? Nominate them to be one of EMRA's 45 Under 45!

This special recognition shines a light on the amazing work our members are doing. This fall, we will celebrate 45 superstars in emergency medicine under age 45.

The deadline to nominate yourself or someone else is July 15.

Visit https://www.emra.org/be-involved/awards/45under45/ for criteria, additional information and the nomination form. ✶

Embrace the Race Outdoors

EMRA's MedWAR (Medical Wilderness Adventure Race), sponsored by BTG, gives you a chance to combine your love of the great outdoors with your medical skills and proficiency.

The 2019 MedWAR race will be held Wednesday, Oct. 30, in Castlewood Canyon State Park near Denver.

Space is limited, and team selection is through a lottery. **Get your team's application in by July 15! Apply at emra.org/medwar.** ✶

Free Service Can Help with Moving

EMRA alumni member Arun Ganti, MD, MPHS, FAWM, has devised a new service to help defray the cost of moving.

"The idea stems from my own experience moving for residency," Dr. Ganti said. "I experienced the benefits of having a shared move firsthand, and I'm hoping to re-create this for others who are in the same situation."

Called **Backlode**, the service is free and straightforward:

- Sign up at backlode.com by providing your email address, the cities you›re moving from/to, and the desired time frame for your move.
- Browse the Backlode database to look for users with whom you can share a move. (All email addresses will be kept private.)
- Contact a moving company of your choice and negotiate a rate. ✶

NRMP Seeks New Leader

The National Resident Matching Program® is seeking a new leader, in the wake of the news that President and CEO Mona M. Signer will retire at the end of 2019.

Under her leadership, applicant participation in the Main Residency Match and the Specialties Matching Service for fellowship training grew by more than 20% and 60%, respectively, while the number of positions placed in those Matching Programs rose by more than 35 and 100%.

"Ms. Signer has been an extraordinary leader for the organization," said Dr. Susan Guralnick, Chair of the Board and Co-Chair of the Search Committee. "Throughout her tenure, she has ensured that the NRMP has always met the highest standards of fairness, integrity, and reliability." ✶

UPCOMING EVENTS

June 30: 20 in 6 Resident Lecture Competition applications due
July 15: Fall Awards applications due
July 15: 45 Under 45 nominations due
July 15: EMRA Resident SIMWars applications due
July 15: EMRA MedWAR team applications due
Aug. 1: Early-bird discount deadline for exhibitors at the EMRA Residency Fair and Job/Fellowship Fair
Aug. 5: *EM Resident* article submission deadline
Aug. 18: EMRA B oard candidates application deadline
September (all month): EM Day of Service
Sept. 13: Fall Rep Council Resolutions due

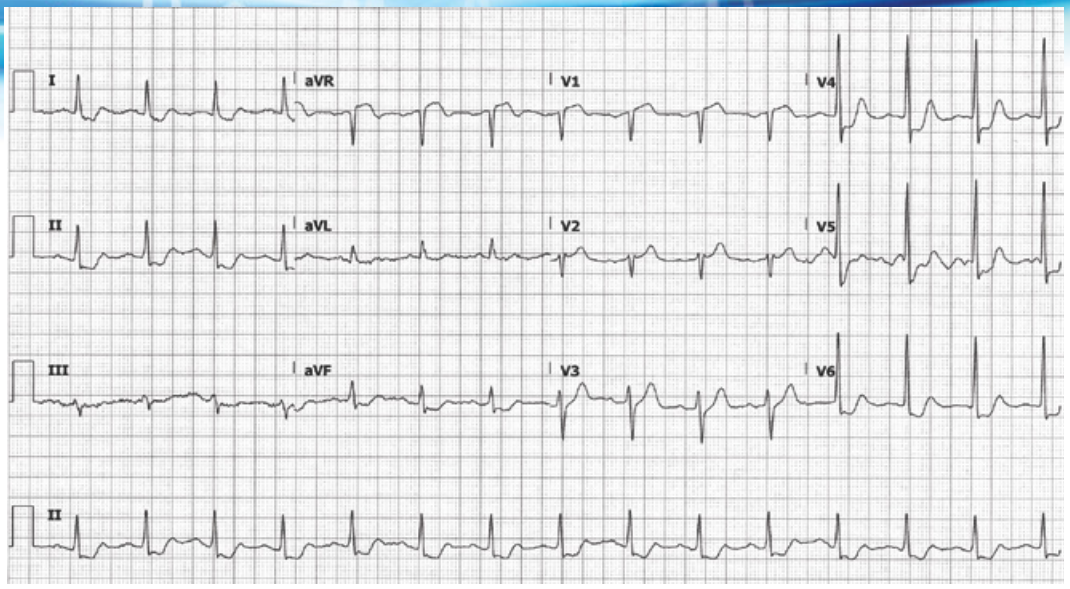
EMRA HANGOUTS
First Thursday of every month

EMRA CAST
EMRA-Cast: Listen at your convenience

ECG Challenge

Paige Ostahowski, MD
Christiana Care Health System
Mahesh Polavarapu, MD
Christiana Care Health System
@mpolavarapu

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



CASE.

A 62-year-old female presents with 1 hour of crushing substernal chest pain.

What is your interpretation of this EKG?

See the ANSWER on page 46

ECG Challenge

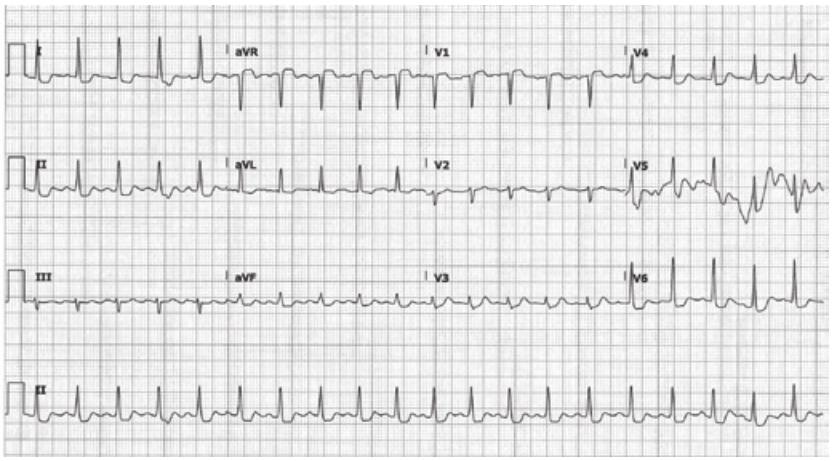
Answer

This ECG shows a normal sinus rhythm with a ventricular rate ~90 bpm, STE in aVR and V1, and STD in leads I, II, aVF, V3-V6 (and possibly III, but it's hard to tell because of the baseline artifact).

The pattern of STE in aVR +/- V1 with diffuse STD is seen in both ACS and non-ACS presentations.

In ACS presentations, it can represent left main coronary artery insufficiency (commonly with STE aVR > V1), proximal LAD insufficiency (commonly with STE V1 > aVR), or triple vessel disease.[1] While STE in aVR with diffuse STD does not meet "traditional" STEMI criteria, the 2013 ACCF/AHA Guidelines for the Management of STEMI recommend invasive angiography for patients with this ECG pattern and a presentation concerning for ACS.[2]

Since we treat patients and not ECGs, it is important to recognize the non-ACS etiologies of this ECG pattern. Consider the following ECG from a 65-year-old female presenting with chest pressure and shortness of breath.



The patient was noted to be hypoxic to 85%, which corrected with supplemental oxygen. Initial CXR showed pulmonary edema and subsequent CTA was negative for PE. Labs were notable for a hemoglobin of 6.0, down from 12.2 a week prior. In this case, the patient's severe acute onset anemia led to subendocardial ischemia and decompensated heart failure, which led to pulmonary edema and hypoxia. The patient received a blood transfusion and judicious diuresis, correcting the underlying problem, after which her ECG normalized.

Other non-ACS causes of STE in aVR include LVH with strain pattern, LBBB, aortic dissection, tachydysrhythmias, GI bleed/acute blood loss anemia, and pulmonary embolism. Accordingly, it is important to consider the non-ACS causes of this ECG pattern based on the patient's overall clinical picture.

aVR LEARNING POINTS

- aVR "looks at" the right upper portion of the heart including the basal part of the septum.
- STE in aVR with diffuse STD can result from ACS or non-ACS etiologies, so always consider the clinical context when going through the differential.
- ACS causes include proximal LAD or LMCA insufficiency or triple vessel disease.
  - Does not meet "traditional" STEMI criteria, but the 2013 ACCF/AHA Guidelines for the Management of STEMI recommend invasive angiography for patients with this ECG pattern and a presentation concerning for ACS[2]
- Non-ACS causes include LVH with strain pattern, LBBB, aortic dissection, tachydysrhythmias, GI bleed/acute blood loss anemia, and pulmonary embolism.
  - ECG changes should resolve with resolution of non-ACS cause.
- Anterior or inferior AMIs with STE > 1 mm in aVR are associated with an increased 30-day mortality.[3]

Case Conclusion

The case was discussed with cardiology, and after the patient's symptoms resolved with medical management, she was admitted to the Cardiac ICU. Trended troponins were negative and cardiac catheterization the next morning showed a 90% ulcerated occlusion of the mid-LAD and non-obstructive left circumflex and RCA disease. The LAD occlusion was treated with a single drug eluting stent and the patient was discharged to cardiac rehab after an uneventful recovery. ✶

For more details, please see pages 1 and 67 of the *EMRA EKG Guide*.

References available online

Board Review Questions

***PEER (Physician's Evaluation and Educational Review in Emergency Medicine)* 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" at emresident.org

Order PEER at acep.org/peer

1. A 54-year-old farm worker presents with low back pain of 3 weeks' duration that radiates down his right leg. An examination reveals decreased sensation on the lateral side of the right lower leg, dorsum of the right foot, and the lateral edge of the right foot. The right patellar reflex is normal, but the right ankle reflex is absent. A motor examination is mostly normal, except that he has trouble standing on his toes. Reflexes in the left leg are normal; he denies urinary or fecal incontinence. If an MRI of the spine were obtained, what would it show?
   A. Broad-based disc bulge compressing the cauda equina
   B. Herniated disc between L5 and S1
   C. Lytic lesion causing cord compression between L2 and L3
   D. Spinal stenosis with narrowing at L3

2. A 57-year-old man presents with fever and chills. He had a left ventricular assist device implanted 5 weeks earlier as a bridge therapy for cardiac transplantation; he has a history of severe ischemic cardiomyopathy. No obvious source of infection is noted on examination. Which component of the assist device is the most likely source of the infection?
   A. Cannula
   B. Driveline
   C. Incision site
   D. Pump pocket

3. A 24-year-old woman who is 22 weeks pregnant presents with a 1-day history of shortness of breath on exertion and an occasional nonproductive cough. She also has had mild bilateral lower leg swelling for 3 weeks. She denies fever, previous illness, orthopnea, and chest pain as well as contact with sick persons, recent travel, and surgical procedures. Vital signs are BP 131/77, P 105, R 18, and T 37°C (98.6°F); SpO_2 is 98% on room air. Laboratory tests reveal a slight leukocytosis and a D-dimer of 600 ng/mL; all other findings are normal. Which of the following diagnostic studies should be ordered initially?
   A. Bilateral DVT ultrasound
   B. CT angiogram of the chest
   C. Echocardiogram
   D. MRI of the chest

4. Which of the following agents has clinical manifestations of acute toxicity most similar to those of tetanus?
   A. Arsenic
   B. Cyanide
   C. Ricin
   D. Strychnine

5. A 26-year-old man presents by ambulance after being stabbed in the neck. Paramedics established large-bore intravenous access in the patient's right upper extremity in the field, and fluids are infusing. Vital signs include BP 118/72, P 115, and R 24; SpO_2 is 99% on 6 L oxygen via nasal cannula. On examination, the patient is awake and alert. The wound is on his left anterior neck between the cricoid cartilage and the angle of the mandible. There is active bleeding from the site and an underlying expanding hematoma. The patient says his throat is tight and that he cannot swallow, and his girlfriend says his voice sounds strange. In addition to applying direct pressure to the wound, what is the best next step in management?
   A. Order angiography
   B. Order CT of the neck with intravenous contrast
   C. Perform cricothyrotomy
   D. Perform endotracheal intubation ✶

ANSWERS
1. B; 2. B; 3. A; 4. D; 5. D

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EMRA gets you in front of the decision makers!

EMRA Residency Program Fair

Saturday, October 26
1:30 p.m. - 3:30 p.m.

Are you a medical student looking to do a little reconnaissance to help better prepare for the interview trail? With close to 130 residency programs in attendance, here is an opportune time to get a head start on the process. Save yourself time and money by doing some early prep.

EMRA Job & Fellowship Fair

Sunday, October 27
5:00 p.m. – 7:00 p.m.

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Exhibitor Registration Now Open!

emra.org/exhibitors

Need more information?

James Bryant
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Fairbanks: New full-time position for a BC/BE Emergency Medicine physician to join a stable, democratic group of 10 physicians. This is a hospital practice based at Fairbanks Memorial Hospital. Annual visits exceed 36,000. Fairbanks Memorial Hospital is a JCAHO accredited 159-bed hospital that is the primary referral center for the 100,000 citizens of Alaska's interior. Fairbanks is a truly unique university community with unmatched accessibility to both wilderness recreation and urban culture. We aim to strike a balance between life and medicine, offering excellent compensation and benefits with a 2-year partnership track. 10 hour shifts with excellent mid-level coverage. For additional information please contact: Michael Burton, MD, President (907) 460-0902 mrb5w@hotmail.com or Art Strauss, MD, Medical Director (907) 388-2470 art@ghepak.com.

CALIFORNIA

Ventura: Newly constructed hospital and ER department functioning since December 2018. Practice with a stable ER group on the central coast of California and only 70 miles from LAX. Positions available in two facilities for BC/BE emergency physician. Main facility is a STEMI Center, Stroke Center with on-call coverage of all specialties. This is a teaching facility with residents in Family Practice, Surgery, Orthopedics and Internal Medicine. Admitting hospital teams for Medicine and Pediatrics. 24-hour OB coverage in house and a well-established NICU. Annual volume is 48K patients with nearly 70 hours of coverage daily and 12 hours of PA/NP coverage. All shifts and providers have scribe services 24/7. Affiliated hospital is a smaller rural facility 20 minutes from Ventura in Ojai. Malpractice and tail coverage is provided. New hires will work days, nights, weekends and weekdays. Come work with a well-established high caliber group with expected volume growth potential at our new facility. Enjoy the life style of a beach community yet outside the hustle of the LA area. Please send a resume to Alex Kowblansky, MD, FACEP, at kowblansky@cox.net.

TEXAS

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For more information or to submit a CV/cover letter please contact:

Lewis S. Nelson, MD
Chair, Department of Emergency Medicine
185 South Orange Avenue, MSB 609
Newark, NJ 07103 **E-609**
Email: Lewis.Nelson@njms.rutgers.edu

Rutgers University is an AA/EEO employer. All applicants will receive consideration for employment without regard to race, color, religion, sex, sexual orientation, gender identity, national origin, citizenship, disability or protected veteran status.

WESTERN PENNSYLVANIA

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- Completion of an accredited Emergency Medicine Residency Program
- BE/BC by ABEM or ABOEM
- Observation experience is a plus

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FOR ADDITIONAL INFORMATION PLEASE CONTACT:

PennState Health

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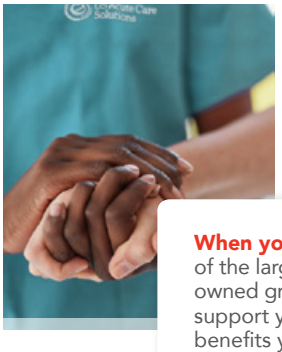

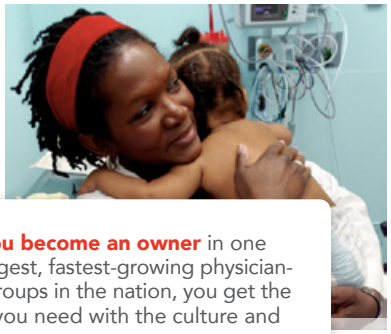

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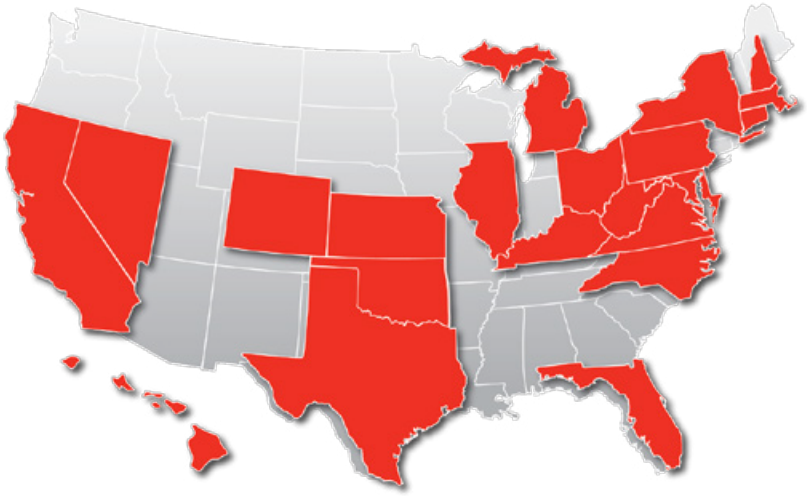

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