EM Resident
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Apathy Is Not Cool

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When did organizational medicine become a dirty word? When did getting involved in physician administration and advocacy become uncool?

I was recently at a conference and after attending EMRA events all day, including Representative Council where resolutions were discussed, a conference participant who happened to also be an EM physician stated: "I don't know how you can sit in meetings for hours! That sounds annoying and boring." This was not only such an inaccurate statement of my day — as it actually involved no meetings and instead was consistent of resolutions and important health care policy discussions — but more damagingly, his ignorant statement was made around impressionable residents.

This impression that being involved in health policy or organizational medicine is tedious and thus "below us" is such a prevalent opinion about emergency physicians. I can tell you, after being involved in health policy and organizational medicine the past few years, it's anything but boring. Physicians are motivated. We are passionate. We are competitive. This field is made for us!

Somehow, people with business degrees and MBAs have convinced us this is tedious work. We were told enough times that this is boring and useless work that we have convinced ourselves of it. We have been successfully removed from the conversation. We are no longer the primary decision-makers (nor sometimes even part of the conversation) in many health care or government-related decisions. We complain and blame "administration" when our apathy put them in charge in the first place.

So I say, I hear the criticisms and naysayers and I reject it. There is such a need for physician leaders. We have seen that attendings, fellows, residents, and medical students are more motivated and want to get involved in administration and governmental affairs than our predecessors have ever been. EMRA supports these efforts through various avenues. Join our committees — it's as simple as providing your email addresses in a website link. Show up — come to EMRA committee events at national conferences including ACEP, LAC, CORD, etc. Apply for Leadership Academy through EMRA/ACEP. Apply for the Health Care Policy elective as a medical student or resident. And of course, get involved by writing resolutions.

One of the most unique, powerful, and exciting privileges of membership is that your EMRA membership allows you to be part of the policymaking process. These policies are set at our bi-annual Representative Council. These resolutions help to set agenda for EMRA by providing guidance on projects to pursue, products to develop, and for the Board of Directors to hear about the issues that matter to you.

Do not let attendings, co-residents, or fellows sway you from pursuing leadership or administration positions because of their personal apathy or lack of interest. I think it is imperative to understand most people are uncomfortable when others understand concepts and ideas better than they do, and often use discouraging language as a defense mechanism.

Do not be that physician who ONLY complains and makes no effort to set forth any positive change. Do not be part of the problem. Work with EMRA, your hospital leadership organizations, national organizations to be part of the solution. EMRA will help you get you there. We are here to serve you. Together we can have a stronger voice in hospital leadership and health policy leadership. ✶

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Protecting the Integrity of Residency Training

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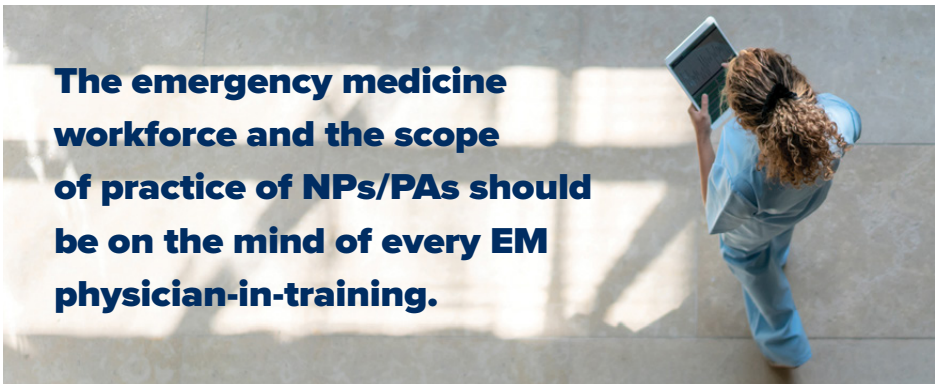

Student Doctor Network. Reddit. Facebook. Twitter. Seemingly every social media outlet used by emergency medicine physicians-in-training blew up on Feb. 22 in response to a proposed non-physician emergency medicine "residency" training program at UNC.[1] The question is: Why?

There's a lot to unpack from this. While the timing was not ideal, only days before rank lists were due, the underlying issue for medical students is how non-physician post-graduate training programs will impact their residency training and post-residency employment opportunities. Medical students are becoming increasingly concerned about the implications for their future careers with attacks coming from so many different angles. With rapid expansion of residency programs, NPs and PAs pushing for independent practice nationwide, and legislation negatively affecting physician compensation, medical students are becoming increasingly tuned in as they recognize the impact of these important issues. Medical students feel that their efforts and future training are being devalued and taken for granted. If we don't protect the integrity of our training, what does that mean for our specialty and our patients?

The emergency medicine workforce and the scope of practice of NPs/PAs should be on the mind of every EM physician-in-training. EMRA firmly believes "**the only pathway to independent practice in emergency medicine in the 21st century is the completion of an ACGME accredited emergency medicine residency training program and board certification by ABEM or AOBEM**."[2] EMRA has held this stance for nearly 30 years.

The question of post-graduate training for NPs/PAs is more complex. More than 40 post-graduate training programs for PAs already exist, including ones at UCSF Fresno, Yale, Cornell, and Carolinas.[3] To protect resident training, the ACGME Common Program Requirements state:

- "The program's educational and clinical resources must be adequate to support the number of residents appointed to the program." (1.D.4.)
- "**The presence of other learners and other care providers**, including, but not limited to, residents from other programs, subspecialty fellows, and advanced practice providers, **must enrich the appointed residents' education**." (1.E.)
- "The program **must report circumstances when the presence of other learners has interfered with the residents' education** to the DIO and Graduate Medical Education Committee." (1.E.1.)[4] *To report, search "ACGME Report an Issue."*

But despite these protections, the allocation of GME funding to residencies is at risk. In the U.S. Government Accountability Office's report, "Health Care Workforce: Views on Expanding Medicare GME funds to NPs and PAs," some members of Congress suggest that prioritizing funding for NPs/PAs may "mitigate the effects of a physician shortage."[5,6] At a time when rates of medical student graduates are increasing faster than rates of new GME spots, expanding funding to NP/PA training does not make sense. This is why All EM Resident Organization and Students (AEROS) wrote a joint statement opposing such.[7]

Is the specialized training for NPs/PAs working in emergency departments better for patient care? Or do the increased hours of specialized training blur the lines between board certified emergency physicians and non-physician providers? The waters are muddy.

Here is what is clear: The Emergency Medicine Residents' Association is the **voice** of emergency medicine physicians-in-training and the future of our specialty. Together, our voices have the power to make change. Your EMRA Board of Directors will be taking resolutions to the AMA and ACEP Council to protect the use of the word "resident" by limiting its use to physicians-in-training only.

Make your voice heard. Share your opinion through EMRA's Representative Council (RepCo), a democratic process where more than 16,000 members' views are collectively turned into policy and affect real change. ✶

References available online

Joint Statement Opposing Expanding Graduate Medical Education Funding to Nurse Practitioners and Physician Assistants

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— STATEMENT —

On Dec. 18, 2019, the Government Accountability Office (GAO) released a report considering the expansion of federal General Medical Education (GME) funding to include nurse-practitioners (NPs) and physician assistants (PAs).[1] All Emergency Medicine Resident Organizations and Students (AEROS) opposes the expansion of GME funding to include non-physician practitioners (NPPs). The report was created to consider the utilization of NPPs as an avenue to mitigate the anticipated primary care physician shortage as outlined by the Health Resources and Services Administration (HRSA) in 2016.[2]

While NPP programs experience rapid expansion, the U.S. physician pipeline faces persistent shortages. Any expansion of GME must prioritize the physician shortage. There has been an increase in medical school enrollment (allopathic and osteopathic) by an average of 4% from 2005-2018 which far outpaces the 1% annual increase in residency positions per year since the Balanced Budget Act (BBA) was passed in 1997.[3,4] The BBA was created to avoid what was thought to be an impending physician surplus at the time. Based on the HRSA report from 2016, the surplus will never come to fruition. The restricted supply of GME positions results in medical students who are unmatched to GME training, delaying their entry into the physician workforce, and further exacerbating the physician shortage. While the physician shortage is growing, NPP programs have no limitation on training positions. According to the HRSA report from 2016, NPs and PAs are growing at a rate that will result in a 74% and 61% surplus by 2025, respectively.[2] Even with the projected surplus, NPPs receive $41 million in annual funding from the Centers for Medicare & Medicaid Services (CMS).[1] The stark contrast in proliferation of new NPPs to new physicians based on the current funding model, further supports the argument against the expansion of funding for NPP education.

GME funds a well-developed and consistent model physician training. GME funding of NPP programs would fund an unstandardized curriculum with highly variable cost of training. Medical students accrue roughly 6,000 clinical hours in addition to the thousands of hours dedicated to independent study and lecture.[5] Furthermore, prior to receiving funding from GME, physicians have completed 3 United States Medical Licensing Exams with a fourth to be completed in clinical training. Once residency begins, physicians take part in a time-tested model of training with predictable budgets. The varied training pathways of NPPs and lack of formal clinical training requirements results in "limited and incomplete" estimates of NPP training, as stated in the GAO report.[1]

The GAO previously recommended that the Department of Health and Human Services (HHS) develop a comprehensive plan to address the physician shortage and noted the vast disparity in GME funding between rural and urban communities.[6,7] In response, HHS has included efforts to redistribute physicians from densely populated areas to rural communities using multiple incentives, including distribution of CMS funds.[8] Of note, GAO addressed the use of non-physician practitioners to improve access to healthcare in rural areas. However, according to the Agency for Healthcare Research and Quality (AHRQ), NPPs remain far more concentrated in urban areas.[9] With such measures already presented for federal funding to address the physician shortage and geographic distribution, expanding GME funds to include NPPs only poses additional planning and costs without guaranteeing improving access and quality of primary care.

AEROS steadfastly opposes the expansion of GME funding to NPPs. GME funding of NPP training would lead to poorly vetted expenditures that would divert funds away from the unsolved physician shortage. To address the unsolved physician shortage, as well as access to high quality and compassionate care, we need to focus our time and resources on training the next generation of physicians. ✶

References available online

The Undifferentiated Pediatric Ingestion

What to Expect and How to React

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Background

A 2-year-old is brought into your ED after her grandmother noticed some of her pills were missing. The grandmother is worried her granddaughter may have taken the pills. The physical exam and vital signs are normal. The grandmother's medications include glipizide and lisinopril.

How long do you observe the patient? What worrisome exam or diagnostic findings should you look for?

In 2017, almost half of pediatric ingestions were for unintentional exposures in children less than 5 years old.[1] Approximately 70,000 ED visits are for unintentional ingestions each year.[2] Most ingestions are benign because of the small quantity/dose and benign nature of the material ingested. However, it is important to know which ingestions can be fatal in infants and toddlers, even at small doses, and what exam and diagnostic findings to look for.

Furthermore, on initial exam, the child may be completely asymptomatic with normal vital signs and labs. It is important to get a good history and observe or admit them based on the potential ingestion and not the exam findings or labs. This is especially true for slow or sustained/extended release medications.

Caveat:

- Although beta blockers are not considered in the "one pill kill" category, it is important to understand the pathophysiology of the beta blockers and its presentation. Thus, it is included in this section.

Calcium Channel Blockers (CCBs) and Beta Blockers (BBs)

Both CCBs and BBs can lead to cardiogenic shock by antagonizing myocardial and vascular calcium channels, resulting in decreased contractility (inotropy and chronotropy) and hypotension resulting from decreased vascular tone. Although CCBs cause direct calcium channel blockade, and BBs cause indirect calcium channel blockade, it is very difficult to differentiate a CCB versus BB ingestion based on presentation. Although, CCB toxicity tends to have hyperglycemia as part of the patient presentation while BBs do not.

CCBs can be fatal at a dose of 15 mg/kg consistent with exploratory ingestions, with the largest dose coming in 360 mg instant release.[3] CCBs are type IV antiarrhythmics and act on the AV node, resulting in heart block (PR prolongation on EKG), bradydysrhythmias, hypotension, and potentially heart failure. In 2004, a case report illustrated the dangers of CCBs after a 14-month-old girl died after ingesting just one nifedipine 10-mg tablet.[4]

BBs are almost never fatal in exploratory ingestions but are important to consider in an older pediatric patient with suicidal intentions. BBs are type II antiarrhythmics and act on the SA and AV nodes, resulting in sinus bradycardia, PR prolongation and bradydysrhythmias similar to CCB toxicity. However, some BBs can result in intraventricular conduction delay (QRS prolongation) and QTc prolongation via sodium and potassium channel blockade similar to type I/III antiarrhythmics.[5] Furthermore, BBs are lipophilic and can have a large distribution and extended effects.[5] Propranolol is most lipophilic, allowing it to cross the blood-brain barrier and result in seizures. Propranolol, along with sotalol, is most likely to cause conduction delays as well as wide complex tachycardias.[5]

Due to CCBs and BBs effects of bradycardia and bradydysrhythmias there is a theorized increased risk of early cardiogenic shock and heart failure in children, who are highly dependent on Heart Rate (HR) for Cardiac Output (CO=Stroke Volume × Heart Rate).

Exam findings in symptomatic patients are due to myocardial suppression and vascular relaxation resulting in fatigue, poor feeding in infants, lethargy, poor perfusion (poor capillary refill, faint pulses), etc. Patients will be bradycardic and hypotensive with little response to atropine/fluids and even cardiac pacing. CCBs may lead to hyperglycemia due to impaired insulin release, while BBs may lead to hypoglycemia due to increased insulin release.[4]

Treatment is targeted at the symptoms, vital signs, and diagnostic findings. Administer sodium bicarbonate for QRS widening and magnesium sulfate for QTc prolongation. Treat cardiogenic shock with fluids, atropine, and vasopressors in both CCB and BB ingestions. Consider early IV glucagon in BB overdose (50 mcg/kg IV initial bolus) with doubling the dose and redosing Q10-20mins.[4] If efficacious, then start a drip of the effective dose.[4] For example, if a 1-mg dose is effective start a drip at 1 mg/hr.

PEARL. Always give glucagon with an antiemetic to avoid airway emergency due to aspiration.

With CCB toxicity you can try giving IV calcium. However, if nothing is working then you must resort to hyperinsulinemia-euglycemia therapy, which combines an insulin infusion with dextrose, as needed, to maintain normal blood glucose. Give an IV insulin bolus at 1 IU/kg along with 10 or 25 g dextrose bolus.[4,5] Then start an insulin drip at 0.5 IU/kg/hr with IV glucose replaced as needed with Accu-Cheks Q30 min within a range of 100-250. It is vital to get a potassium level prior to initiation and to monitor potassium levels closely. If the patient is still unresponsive, then intralipid therapy and/or Extracorporeal Membrane Oxygenation (ECMO) may be considered.[5]

IV calcium is typically used for CCB overdose, not BB overdose. Glucagon is used for BB overdose, but not CCB overdose. In addition, for CCB overdose, standard pressures can also be utilized. HIE therapy works for both CCB and BB.

PEARL. CCBs or BBs should be observed for at least 24 hours due to possible long-acting formulations.

Alpha-Adrenergic and Imidazoline Agonists

Alpha-2 and imidazoline receptor agonists commonly involved in toxic ingestions include clonidine, nasal decongestants (oxymetazoline, tetrahydrozoline, and xylometazoline), and glaucoma agents (brimonidine, apraclonidine).[4] All of these agents can have a delayed presentation of up to 4 hours, but typically present within 30-90 minutes and can have persistent effects for 1-3 days. Agonistic effects at the centrally (CNS) located alpha 2 receptor result in decreased release of norepinephrine and, therefore, globally reduced sympathetic tone.[4] This results in bradycardia and hypotension, but it can also result in altered mental status, miosis, and decreased respiratory drive similar to an opioid toxidrome.[4]

PEARL. Toxic effects may be delayed up to 4 hours and may persist for 1 to 3 days

Hypotension is managed with fluids/vasopressors, bradycardia is managed with atropine/pacing, and bradypnea/decreased respiratory drive is managed with naloxone/positive pressure ventilation/intubation.[4] The dose of naloxone is 0.1 mg/kg, up to a total of 10 mg and may be beneficial for clonidine toxicity, but less so for imidazolines.[4]

Sulfonylureas

Sulfonylureas are insulin secretagogues used in treating type 2 diabetes. They stimulate pancreatic beta cells to secrete insulin.[4,6] Time of onset is within 30 minutes, while peak concentrations are reached at 4-6 hours with long-term effects up to 24 hours.[4,6]

Sulfonylureas have narrow therapeutic indexes with small doses of just 0.1 mg/kg resulting in severe hypoglycemia in children.[3,4,6] Hypoglycemia has been reported in children who have ingested just 1 tablet of chlorpropamide, glipizide, or glyburide.[4,6] In pediatric patients, hypoglycemia is typically observed within 8 hours of ingestion.[3,4,6] However, there are reports of delayed onset (11-45 hrs) and recurrence up to 30-70 hours after ingestion.[3,4,6]

Hypoglycemia is commonly referred to as the syphilis of metabolic disorders and can mimic a wide array of presentations. Exam findings can include irritability, poor feeding, lethargy, seizures, altered mental status, coma, etc.[3] Vitals may be normal or demonstrate bradycardia, hypotension, or irregular respirations.[3] Therefore, it is important to always check a glucose on every patient with any of the above presentations and have ingestion on your differential for unexplained hypoglycemia. Typically, dextrose boluses are sufficient to manage hypoglycemia. However, octreotide can be useful for patients with refractory hypoglycemia.[6]

Confirmed or suspected sulfonylurea ingestions may require 24 hours of observation with glucose monitoring. Therefore, it is easy to justify admission for observation.

PEARL. Patients with sulfonylurea ingestion typically require 24 hour observation

Tricyclic antidepressants (TCAs)

TCA ingestions can be deadly and easy to miss. They have a narrow therapeutic index and long half-life (often > 24 hours). They act by inhibiting the reuptake of norepinephrine, dopamine, and serotonin in the CNS and also block alpha and muscarinic (acetylcholine) receptors, which can result in reflexive tachycardia.[3,7] Furthermore, they act as sodium channel antagonists in cardiac myocytes, which can result in dysrhythmias.[3,7]

Exam findings can be normal or include lethargy, poor feeding, coma, and seizures. Vitals may be normal or include tachycardia, bradycardia, and hypotension.[3] Tachycardia without any other findings responds to benzodiazepines.[7] Hypotension from

TCAs will typically respond to fluids but may need vasopressors to work against the alpha antagonistic effect of TCAs.[7]

The most dangerous effect of TCA toxicity is due to cardiac sodium channel antagonism and resultant QRS prolongation (>0.10 secs) and prominent R-wave in aVR, which can lead to dysrhythmias and cardiac arrest.[4,7] Sodium bicarbonate is the treatment of choice for QRS prolongation and should be given promptly to any patient who is suspected of having a TCA overdose with EKG changes. It is given as a bolus of 1 mEq/kg followed by an infusion.[4,7] Because of the long half-life of TCAs, patients with suspected TCA ingestion require 24 hour observation with continuous cardiac monitoring.[4,7] Patients who are alkalotic should be given hypertonic saline, which competes with TCAs' antagonism at cardiac sodium channels.

If a patient with TCA ingestion continues to be symptomatic, both ECMO and lipid emulsion therapy can be considered and maybe live saving.

PEARL. Suspected TCA toxicity with EKG changes of QRS prolongation (>0.10 secs) should be emergently treated with Sodium Bicarbonate.

INGESTIONS SIMPLIFIED

Ingestion	Mechanism of Action	Symptom Onset	Potential Findings	Treatment*
Calcium Channel Blockers	Direct myocardial and vascular calcium channel antagonism	Typical: 2-4 hrs Delayed: 24 hrs	Cardiac depression; Poor perfusion; Hyperglycemia	**Glucagon:** 50 ug/kg IV initial bolus, double and triple subsequent bolus if no effect; Start infusion at response dose per hour **Glucose-insulin:** 0.5 U/kg regular insulin IV bolus, followed by 0.1–1.0 U/kg/h, titrate to hemodynamic effect; D10 W infusion, titrate to euglycemia **Monitor potassium**
Beta Blockers	Indirect myocardial and vascular calcium channel antagonism Sodium and potassium channel blockade		Cardiac depression + QRS prolongation, QTc prolongation; Poor perfusion; Hypoglycemia	
Clonidine & Imidazolines	Decreased release of norepinephrine via alpha-2 and imidazoline receptor agonism	Typical: 30-90 mins Delayed: 4 hrs	Reduced Sympathetic Tone: Bradycardia;; Hypotension; AMS; Miosis; Decreased respiratory drive	**Naloxone** *(Effective only for clonidine)* **Age <5y:** 0.01–0.1 mg/kg IV/IO/IL/ET q3–5 min; Max 2 mg/dose **Age >5y:** 0.4–2.0 mg IV/IO/IL/ET q3–5 min Max dose: 10 mg
Sulfonylureas	Pancreatic beta cell stimulation	Typical: 30 mins to 8 hrs Delayed: 11-45 hrs	Symptomatic hypoglycemia	**Dextrose Boluses + Glucose Checks q30min** **Octreotide**
Tricyclic Antidepressants	Reuptake inhibition of CNS norepinephrine, dopamine, and serotonin Alpha and muscarinic acetylcholine receptor blockade Sodium channel antagonism	Typical: 2-4 hrs Delayed: 24 hrs	QRS prolongation; prominent R-wave in aVR Hypotension; CNS depression	**Sodium Bicarbonate:** Bolus of 1 meq/kg followed by an infusion
Cinchona Alkaloids	Inhibit fast inward sodium channels, mainly myocardial but penetrates nearly all tissues	Typical: 1-2 hrs Delayed: 3 hrs	Prolonged QT, paroxysmal ventricular tachycardia; hypotension; seizures, retinal damage; tinnitus	**Hypertonic Sodium Bicarbonate** **Monitor potassium**
Camphor	Unknown mechanism neurotoxicity Direct mucosal irritation	Typical: 1-2 hrs Delayed: 4 hrs	Seizures; CNS hyperactivity then depression; Elevated LFTs; Mucosal Irritation	**None** *(Supportive only)*
Carbamates & Organophosphates	Acetylcholinesterase inhibition	Typical: 5-60 minutes Delayed: 24 hrs	Most commonly CNS depression Muscarinic & Nicotinic Overstimulation	**Atropine** 0.02 mg/kg every 5 minutes until respiratory secretions dry and bronchoconstriction ceases; Min dose: 0.01 mg **Pralidoxime** Loading dose of 25-50 mg/kg, followed by IV infusion

*Treatments are in addition to supportive measures or standard treatments such as benzodiazepines for seizures, magnesium sulfate for torsades de pointes, respiratory support, fluids/vasopressors for hypotension, etc.

Cinchona Alkaloids

Cinchona Alkaloids include antimalarials like quinidine and chloroquine, which are commonly prescribed at doses up to 600 mg per tablet and can be fatal at doses of 20 mg/kg, a mere third of a tablet for a 10 kg child.[8,9] Have a high suspicion of ingestion of antimalarials if there is a history of recent travel or planned travel, as patients or family may be on malarial prophylaxis.

Cinchona Alkaloids are Class 1A antiarrhythmic agents and inhibit cardiac myocyte sodium channels, which results in prolonged QRS and QT intervals and ventricular tachydysrhythmias.

Acute toxicity can present with confusion, syncope, palpitations and hypotension.[9,10] Cinchona Alkaloids can also cause retinal damage and present with blurry vision.[9,10] Other potential presentations can include headache, hearing impairment, flushing, hypersensitivity reactions such as fever, rash, blood dyscrasias, hepatitis, tinnitus, and gastrointestinal upset.[10] Maximum concentrations will occur within 1-2 hours of oral intake.[9,10]

PEARL. Think Cinchona Alkaloids when a patient presents with blurred vision and/or hearing impairment in the setting of an arrhythmia

Management of acute intoxication includes magnesium sulfate, potassium, and overdrive pacing for QT prolongation and Torsades de Pointes.[10] Activated charcoal readily absorbs quinine and may be considered early after ingestion, with standard pediatric dosing of 0.5-1 gm/kg PO.[11] Sodium bicarbonate should be given for prolonged QRS interval or heart block to antagonize the inhibitory effects on sodium channels, with a target pH of 7.45-7.50.11 Be sure to monitor and correct potassium when administering bicarbonate. ECMO should be considered in any patient with continued cardiac depression not responding to treatment.

Camphor

Camphor ingestions often come from household products such as Vaporub®, Orajel™, and Tigerbalm® used as a cough suppressant and for mouth and muscle pain, respectively. Due to rapid absorption, effects can occur within 5-20 minutes, although typical onset is closer to 1-2 hours.[11,12] A single teaspoon (~5 mL) can be fatal with the doses available at 550 mg/tsp; therefore, high suspicion and immediate action is crucial.[9] The exact mechanism of neurotoxicity is unknown, but the concurrent liver toxicity seems to transpire through a mechanism similar to Reye Syndrome. Direct mucosal irritation can cause oropharyngeal burning, abdominal pain, vomiting, and diarrhea.[12,12] Seizures are often the first sign of toxicity. However, CNS symptoms can transpire in 2 phases, with an initial phase of agitation, anxiety, hallucinations, hyperreflexia, and myoclonus and a second phase of lethargy, coma, and respiratory depression.[4,9,12]

PEARL. Keep camphor ingestion on your differential when considering elevated LFTs, altered mental status, neurologic changes with subsequent respiratory depression. Look out for its unique potent smell (mothball-like odor).

Treatment is supportive. Manage respiratory and CNS depression with positive pressure ventilation/intubation. Seizures are often short-lived and may not require any active intervention, but any prolonged seizures should be treated with benzodiazepines.[4,12] Asymptomatic patients, 4 hours post-ingestion, can be safely discharged home.

PEARL. Asymptomatic patients, 4 hours post-ingestion, can be safely discharged home.

Carbamates & Organophosphates

Organophosphates and carbamates are still found commonly in insecticides. Although less commonly ingested, they have the potential of being fatal in children in a single swallow.[12] Depending on the route of exposure, the effects can take place within minutes.[11] These poisons act as acetylcholinesterase inhibitors, leading to excessive stimulation of muscarinic and nicotinic receptors. Unlike in adults, children manifest commonly with CNS symptoms, such as seizures, coma, and respiratory failure.[12]

Treatment should target each patient's specific symptoms. For wheezing, airway secretions, and other muscarinic symptoms, administer atropine 0.02 mg/kg every 5 minutes until respiratory secretions dry and bronchoconstriction ceases.[12] For seizures, follow your normal seizure algorithm; however, consider atropine as it can fix the underlying problem. Also give concurrent pralidoxime chloride (2PAM) with atropine in all organophosphate poisonings. 2PAM prevents "aging" (irreversible inhibition) of acetylcholinesterase, primarily, at the neuromuscular junction. It is not helpful for carbamate poisoning.

If asymptomatic on arrival, children should be monitored for at least 4 hours before considering discharge.[12] Typically symptoms will be seen within the first hour, and most agents will cause symptoms within 8 hours at most.[11] However, lipophilic agents such as fenthion, or those like malathion that require metabolic activation, may have a delayed presentation up to 24 hours.[11] Therefore, the observation time should be based on the pharmacokinetics of each agent.

PEARL. Tachycardia is not a contraindication for atropine use.

TAKE-HOME POINTS

- ✓ Do not underestimate ingestions; a child who comes in looking well can quickly become unresponsive and in shock.
- ✓ Investigate the type of ingestion in order to appropriately disposition the patient, as some long-acting agents can present many hours after ingestion.
- ✓ When presented with a critically ill pediatric or adult patient, do not underestimate the possibility of ingestion, as targeted treatments may be the difference between life and death. ✶

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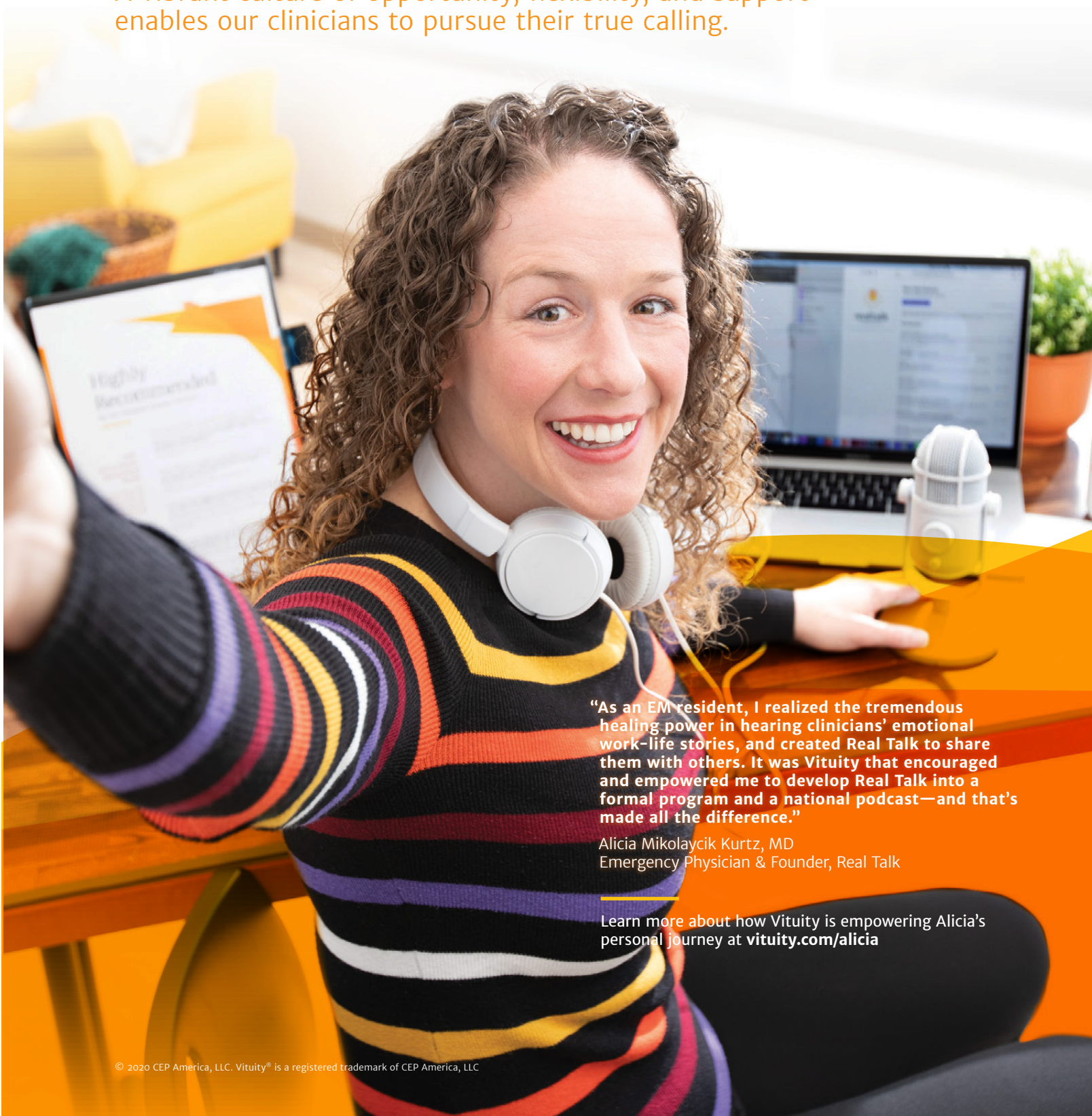

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Alicia Mikolaycik Kurtz, MD
Emergency Physician & Founder, Real Talk

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A CASE REPORT

Pneumomediastinum Secondary to Medication-Induced Esophageal Microperforation

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A 23-year-old male presented to the hospital complaining of odynophagia which he first noticed prior to his presentation to the ED, and which occurred after ingesting a single over-the-counter cold medication. Prior to taking the gel capsule which contained acetaminophen, dextromethorphan, guaifenesin, and phenylephrine HCl, the patient had a sore throat and no difficulty swallowing. The patient reported difficulty swallowing the gel capsule but an inability to expel it. Afterward, the patient attempted to swallow liquids but was unable to do so. He had no difficulty breathing or speaking. He denied retching, vomiting, drooling, abdominal pain, chest pain, cough, shortness of breath, hemoptysis, recent travel, recent illness, other new medications, new foods, excessive alcohol use, drug use, or smoking. His last meal was at 7 pm the day prior.

His initial vital signs were a temporal temperature of 97.8 F, blood pressure of 128/70 mmHg, heart rate of 97 beats per minute, respiratory rate of 20 breaths per minute, and oxygen saturation of 100% on room air.

The physical examination revealed a well-appearing male who was in no distress and with a patent airway without stridor. His oral mucosa was moist with no pooling of secretions. On examination of the oral cavity, no foreign bodies or obvious oral lacerations were noted, and his dentition was intact with no abnormalities. The patient demonstrated normal phonation and was tolerating secretions without difficulty. No neck masses, asymmetry, bruising, or rashes were identified, and no thyroid enlargement, asymmetry, or nodules were noted. There was no jugular venous distention and no lymphadenopathy. His heart sounds were normal, and lungs were clear to auscultation with no wheezing, rhonchi, or rales. There was no chest wall tenderness or sternal crepitus. The abdomen was nontender and bowel sounds were present. The patient was alert, awake, and oriented and had no neurological deficits. He was given 30 mL of aluminum and magnesium hydroxide-simethicone, 15 mL of 2% lidocaine oral solution swish and spit, followed by 5 mL phenobarbital-hyoscyamine-atropine-scopolamine, all of which did not alleviate his symptoms.

A soft tissue x-ray of the neck revealed no foreign body, but there was questionable linear retropharyngeal extraluminal gas noted. Due to this finding, Computed Tomography (CT) of the neck was performed without contrast and identified a non-obstructing soft tissue density and pneumomediastinum with parapharyngeal gas in the neck. Chest x-ray confirmed a faintly visualized pneumomediastinum, and Gastroenterology was consulted. After evaluating the patient, the gastroenterology team recommended an esophagram to assess for esophageal perforation.

An esophagram with barium contrast confirmed the pneumomediastinum but was unable to identify a site of perforation with no fluoroscopic evidence of contrast extravasation. However, given preceding imaging confirmed a pneumomediastinum as well as air within the wall of the esophagus, suspicion remained high for an esophageal injury. A CT of the chest without contrast was performed and further confirmed the pneumomediastinum extending mostly around the esophagus to the level of the gastroesophageal junction with no new acute changes. Oral contrast from the previous esophagram was identified within the central portion of the distal esophagus which was reported as intraluminal. No contrast extravasation into the mediastinum was identified. Also noted was air within the wall of the proximal stomach as well as a few droplets of apparent extraluminal air posterior to the stomach in the left subdiaphragmatic region. This air was likely secondary to extension from the pneumomediastinum/paraesophageal air. No free contrast was identified in the upper abdomen.

The patient's EKG revealed a normal sinus rhythm with a heart rate of 75 bpm with no ST changes or QT prolongation. Labs were drawn, including a CBC, BMP, PT/PTT, and the only appreciated pertinent lab finding was a leukocytosis with a WBC of 15.41. Otherwise, all other lab results were within the normal range. The patient was started on intravenous Normal Saline fluids and 3.375 g of IV piperacillin-tazobactam was administered for broad-spectrum antibiotic coverage. The patient was then admitted to the Medical Intensive Care Unit (MICU) for further monitoring, and he was not allowed anything by mouth (NPO).

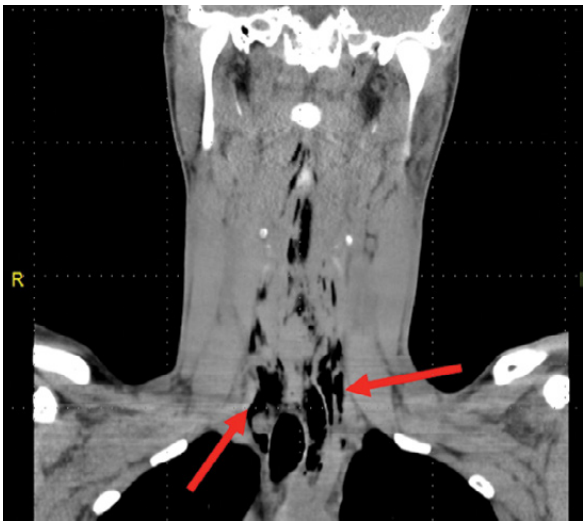


FIGURE 1. CT Neck without contrast revealing pneumomediastinum

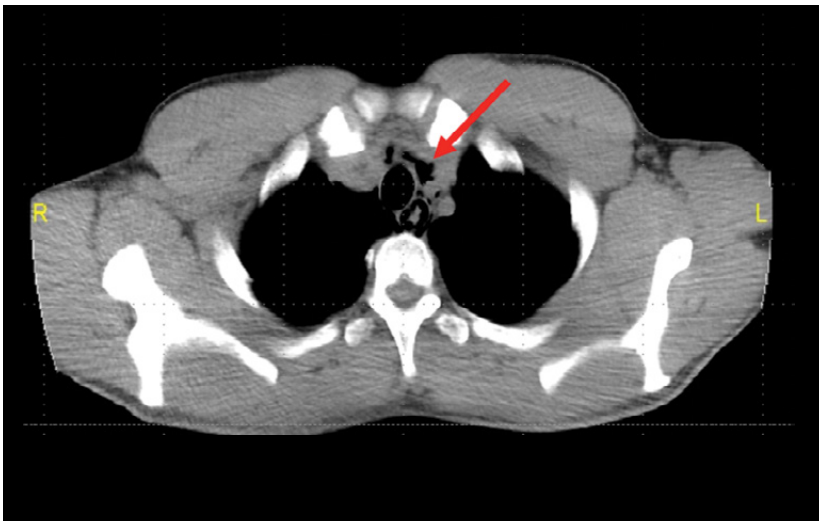


FIGURE 2. CT Chest without contrast demonstrating free air around the esophagus

Discussion

Esophageal perforation is a full-thickness tear in the esophageal wall.[1] The esophagus lacks a serosal layer, thus making it more vulnerable to injury.[2] The vast majority of perforations are caused by medical instrumentation.[1,2,3] Other common causes include trauma, post emesis injury, esophageal disease, and caustic liquids.[2] An ingested substance, whether it be food, foreign body, or medication, rarely causes a perforation. The rarity of which is what makes this case of suspected medication-induced esophageal perforation interesting.

Patients presenting with esophageal perforation can have a variety of signs and symptoms. Patients can present with symptoms including but not limited to chest pain, dyspnea, vomiting, hematemesis, dysphagia, neck pain, odynophagia, hoarseness of voice, and shoulder pain.[2] Physical exam findings are generally benign, though it can be possible to note crepitus, local tenderness, or subcutaneous emphysema. The diagnosis is primarily made with the use of imaging. A Chest X-Ray is performed to evaluate for free air and for pleural effusions. An esophagram is performed with contrast to identify an esophageal perforation by evaluating for contrast extravasation. Complications of esophageal perforation include mediastinitis, an intrathoracic abscess, shock, respiratory failure, and pneumomediastinum. Treatment depends on symptom severity, patient presentation, and on the patient's clinical course. Given the possibility of deterioration and for significant complications, as listed above, patients commonly require ICU admission, NPO status, and observation.

Pneumomediastinum, also known as mediastinal emphysema, is the presence of air in the mediastinum.[4,5] It can result from barotrauma, direct trauma, or bowel trauma.[6] Patients present most commonly with chest pain, which is usually retrosternal. Other common symptoms include shortness of breath (75%), cough (80%), vomiting, and difficulty swallowing.[6,7,8] A common finding on physical exam is Hamman's crunch, which is a "crunching" sound noted on auscultation over the precordium.[6,7,8] The diagnosis of pneumomediastinum can be made through plain films. An anteroposterior (AP) chest x-ray can demonstrate lucent streaks or air tracking along mediastinal structures.[8] A chest CT can help to assess the extent of the injury and can aid in the confirmation of the diagnosis. Further testing is usually not necessary.[8] Laboratory studies and an EKG are reviewed to evaluate for other possible etiologies. The clinical course commonly follows the spontaneous or assisted reabsorption of the free air and thus treatment most commonly consists of observation and supportive care.

In this case, the patient's presenting complaint of difficulty swallowing,

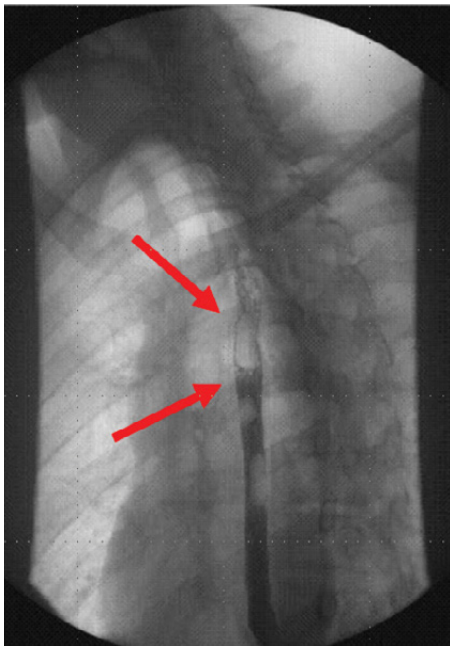


FIGURE 3. Esophagram revealing pneumomediastinum

medically termed odynophagia, lead us to evaluate for an esophageal pathology. An x-ray of the neck was ordered to assess for a possible foreign body, which instead noted retropharyngeal air. The radiograph finding and absence of a foreign body prompted further imaging with a CT of the neck for a better evaluation of the upper airway and of the upper gastrointestinal tract. Although the CT scan identified a soft tissue density as the possible inciting cause of the patient's symptoms and imaging results, the incidental pneumomediastinum

References available online

warranted further evaluation with an esophagram. Neither CT imaging nor the esophagram identified a perforation with no demonstrated extravasation of contrast. However, the esophagram noted free air in the esophageal wall thereby further supporting that the patient's pneumomediastinum was induced by an esophageal injury.

The active ingredients in this particular brand of gelcap-acetaminophen, dextromethorphan, guaifenesin, and phenylephrine HCl-are not known to be caustic. Most cases of medication-induced esophageal injury heal without intervention and within a few days.[1] In this patient's case, the esophagram noted air in the esophageal wall, but the repeated CT chest, performed afterward, only noted air within the stomach wall and posterior to the stomach. The combination of these findings suggests that the perforation had initially been large enough to allow free air into the mediastinum, but small enough to have healed within hours and thereby precluding the identification of perforation on further imaging.

Pneumomediastinum, in the context of microperforation, presents similarly to spontaneous pneumomediastinum and will likely resolve on its own. An accurate diagnosis of pneumomediastinum was made through the combination of multiple imaging studies. As explained above, imaging confirmed the presence of free air, but likely due to a healed perforation, could not identify a perforation. The source appears to have been through an esophageal microperforation that healed within hours as demonstrated by a lack of fluoroscopic contrast extravasation on esophagram imaging. Esophagram and CT chest found no extravasation of contrast to neither locate nor confirm perforation.

Case Conclusion

Once the patient was able to tolerate clear liquids, 2 days after MICU admission, he was transferred to the medicine floor. The following day, while on the general medical floor, the patient tolerated a soft diet and was medically cleared by the gastroenterology team for discharge home. In total, the patient spent three nights in the hospital.

Summary

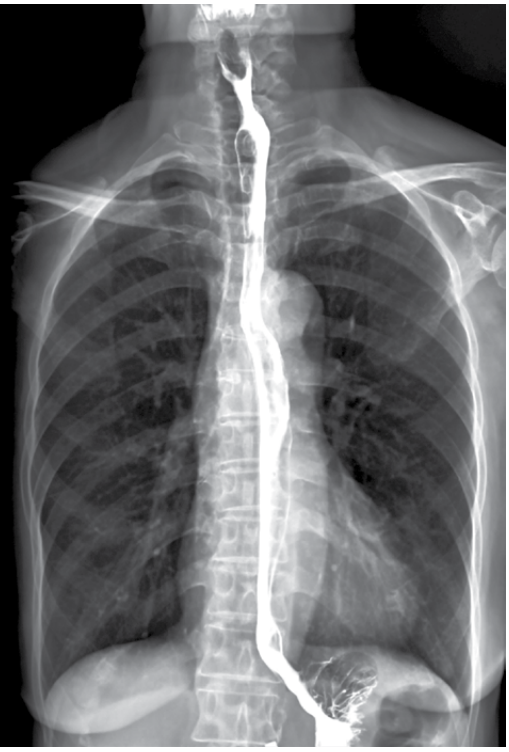
The patient in this report had a good outcome. The patient tolerated oral fluids within 48 hours and was discharged within 72 hours without direct medical or surgical intervention. As in most other cases of medication-induced esophageal perforation, this patient's injury healed on its own within a few hours to days.[4,10] Long term follow up will likely not be necessary as previous studies have noted that the occurrence of reinjury from a non-caustic offending agent, particularly a substance the patient had previously ingested, is low.[4,9,10]

To our knowledge, there does not appear to be any reported cases of esophageal perforation due to the ingestion of a non-caustic, over-the-counter medication causing pneumomediastinum. Previous reports have commonly attributed the diagnosis of medication-induced esophageal injury to caustic fluids, certain antibiotics, nonsteroidal anti-inflammatory drugs (NSAIDs), aspirin, bisphosphonates, potassium chloride, and iron.[9,11]

While finding the root cause of pneumomediastinum can be important regarding further management and interventions, the treatment goal regarding the diagnosis of pneumo-mediastinum should be to ensure patient stability through observation and to intervene with resuscitative measures if indicated throughout the patient's clinical course. ✶

TAKE-HOME POINTS

- **Esophageal microperforations can heal within a matter of hours, masking the root cause of pneumomediastinum.**
- **There appear to be no previously reported cases of a non-caustic, OTC medication leading to pneumomediastinum.**



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Rethinking Emergency Contraception

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A 17-year-old woman presents to the ED after being sexually assaulted while at a party approximately 36 hours prior. She believes she was drugged and does not recall most details of the incident. She is tearful, but otherwise uninjured. The patient is not currently using any form of contraception. In the ED the patient consents to a forensic examination. She is also provided with STI screening, empiric antibiotic prophylaxis, Hepatitis B booster vaccine, and counseling regarding the risk of HIV exposure. She also confirmed prior completion of the HPV vaccination series.[1] She is concerned about pregnancy and wishes to discuss her options for emergency contraception. What would you tell her?

Introduction

The timely provision of emergency contraception (EC) for sexual assault survivors is a critical intervention for preventing unwanted pregnancy.[2] While providing EC is the standard of care if there is any concern for pregnancy, counseling regarding the most effective methods as well as provision of those methods is highly variable among emergency physicians.[3,4] A recent study of patients treated in pediatric EDs around the country found that 6% to 89% of patients were receiving STI and pregnancy testing and 0% to 57% were receiving all recommended prophylaxis, including EC.[4] Another small survey from 2016 found that very few emergency physicians regularly offer the newly available and highly effective EC options to women.[3] Specifically, this article highlights the 2 most effective methods of EC currently underutilized in the ED: Ulipristal Acetate and the Copper Intrauterine Device (Cu-IUD).

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TABLE 1. Comparing Available Emergency Contraceptive Methods At-a-Glance[2,5,6]

Type	Brand Names	Timing	Dose	Efficacy	Over the Counter	Price
Cu-IUD	Paragard®	≤120 hours	N/A	>99%	No	$800-$1100
Ulipristal	ella®	≤120 hours	30 mg ulipristal acetate	98%	No	$45
Progestin Only Pills	Plan B One-Step®, Take Action, My Way, Aftera®, etc.	≤72 hours	1.5 mg levonorgestrel	60%-94%	Yes	$16
Combined Oral Contraceptive (COC) Pills	Many; see a complete list[5]	≤120 hours	100-120 µg ethinyl estradiol + 0.5-0.6 mg levonorgestrel per dose; 2 doses 12 hours apart	56%-89%	No	Variable; $15-$40

References available online

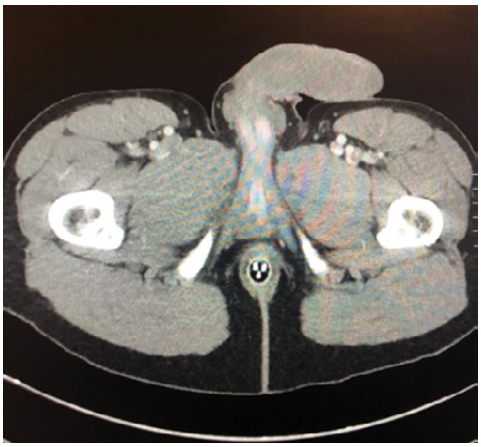

FIGURE 1.

Hooked on Colonics

Colon Cleansing Gone Wrong

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A 52-year-old male with a past medical history of deep vein thrombosis on apixaban presented to the ED complaining of lower left quadrant abdominal pain, rectal pain, and a scant amount of bright red blood per rectum. The pain began approximately 1 day prior, shortly after a procedure he called colon hydrotherapy. The patient reported that multiple times over the course of the past year he has sought treatment from a holistic health center due to a health recommendation from his Reiki master. As a regular part of his therapy routine an acupuncturist would insert an aluminum tube into his rectum and spray pressurized water with the intention to improve overall gastrointestinal health. The pain was described as sharp, mostly in the left lower quadrant and rectum. The evening prior he felt the urge to defecate, but the pain was exacerbated upon pushing and he noticed bright red blood on tissue paper when cleaning. He decided to seek care in the morning as his pain was progressively worsening. A CT scan was obtained showing the following images.

Learning Points

A retained rectal foreign body is a complaint that emergency physicians will encounter multiple times throughout their careers. In an urban population-based study, it was found that there is a patient encounter for a retained rectal foreign body at least once per month per hospital. Commonly encountered objects include bottles and glasses (42.2%), but the type of foreign body can vary widely. Patients are more frequently male and the average age at presentation is 44 years old. The reason people insert objects into their rectum is most often autoeroticism, concealment, attention seeking, or reported as accidental insertion. Patients with this presentation are often embarrassed so it is important to respect their privacy. During these patient encounters, providers may want to have a chaperone present at all times due to the psychological nature of this presentation. It is important to remember the potential that the object was placed involuntarily; always ask about the circumstances and offer help or counseling. On initial history taking, as was seen in this case, the patient may not endorse that there is a foreign body in their rectum up to 20% of the time. The most common complaint is abdominal pain, rectal pain, constipation, obstipation, or bright red blood per rectum.

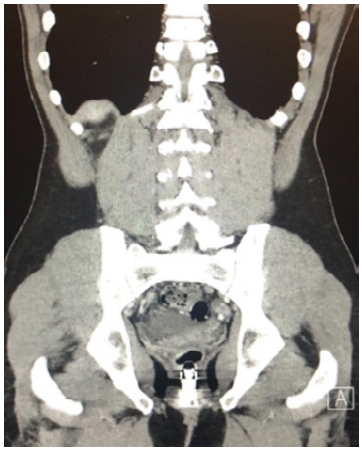

FIGURE 2.

Once the foreign body is discovered through history or imaging, it is vital to repeatedly assess for peritonitis and hemodynamic instability as these situations require emergent surgical intervention. Removal of sharp objects should not be attempted within the ED due to the risk of perforation. If the patient is stable with limited risk factors, transanal extraction can be safely attempted within the emergency department. The patient should be placed in the lithotomy position and receive adequate amounts of analgesia. A perianal nerve block can be used if the object is not easily visualized or palpable. Extraction can proceed while using sufficient lubrication and asking the patient to assist using the Valsalva maneuver. Tools utilized for extraction vary widely depending upon type, shape, and depth of the object. Our first attempt was unsuccessful using a nasal speculum for retraction with ringed forceps to attempt grasping the object. Colorectal surgery was consulted and used special anal retractors with Debakey forceps. Always observe for potential signs of instability secondary to surgical complications following extraction. Obtain a post-retrieval x-ray to rule out potential colorectal injury sustained during the procedure.

Regarding the patient's colon irrigation, which is also referred to as colon hydrotherapy, or a "colonic," there is currently no scientific evidence to support its use to promote

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References available online

Rethinking Emergency Contraception

Types of Emergency Contraception

Four major types of EC are currently recommended by the American College of Obstetrics and Gynecology.[2] Method choice may be based on institution-dependent practices, time since intercourse, method efficacy, availability over the counter or the woman's individual long-term contraceptive and reproductive goals (Table 1).

A Note on Efficacy

No method of EC is perfect, and studies regarding the efficacy of each method are highly variable. In general, efficacy of all EC methods decreases as time since intercourse increases. All oral regimens have reduced efficacy in overweight and obese women; body mass index (BMI) has no effect on efficacy of the Cu-IUD.[2,5]

Nuts and Bolts

Cu-IUD

The Cu-IUD is currently being used off-label as EC. Insertion must be done by a trained provider. All OB/GYNs and an increasing number of primary care providers receive training in IUD insertion.[7] The Cu-IUD is the most effective form of EC and will reliably prevent pregnancy for up to 10 years after initiation. Unlike other methods, its efficacy as EC is not lower in patients with high BMI.[2] While insertion in the ED as EC is currently uncommon, emergency physicians may consider a consult to OB/GYN for patients who prefer this method.

Ulipristal Acetate

Ulipristal Acetate is the newest form of oral EC. Given its simplicity, efficacy and side effect profile, emergency physicians should strongly consider recommending this method before offering other oral regimens to patients.

Progesterone Only Pills

Progestin only pills are the over-the-counter option for EC, most commonly referred to as "Plan B." They are available at Walgreens, CVS, Amazon, and any other major drugstore, and can be purchased by any individual. These pills are typically taken in a single dose.

Combined Oral Contraception Pills

Also called the Yuzpe regimen, this off-label method consists of taking a high dose of certain combined oral contraceptive pills that contain both Estradiol and Levonorgestrel. Depending on particular formulation, this method involves ingesting 4-6 pills (100-120 ug Ethinyl Estradiol and 0.5-0.6 mg Levonorgestrel) per dose. Two doses should be taken 12 hours apart. This is widely considered the least effective method of EC.

Adverse Effects

Cu-IUD insertion is typically accompanied by cramping; uterine perforation is a rare but serious complication (1/1000). Common long-term adverse effects include dysmenorrhea and heavy menstrual bleeding.[2]

Nausea, headache and irregular bleeding are common with all forms of oral EC. Nausea is most common with Combined Oral Contraceptive regimens. Less commonly women will experience dizziness, fatigue, breast tenderness and abdominal pain.[2] ✶

TAKE-HOME POINTS

- Emergency physicians are responsible for caring for women after some of the most traumatic experiences of their lives. Provision of EC is an essential component of post-sexual assault care, but highly effective methods are currently underutilized in the ED setting.[3]
- Ulipristal Acetate and the Copper IUD should be considered first line options for pregnancy prevention.
- Knowledge regarding all available options will allow emergency physicians to better counsel and care for their patients.

Colon Cleansing Gone Wrong

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gastrointestinal health. In contrast, there are serious potential risks. Patients often seek such modes of care in an attempt to cleanse their gastrointestinal system of harmful bacteria, or toxins. The procedure results in the disruption of the normal gastrointestinal flora, which is potentially harmful. There have been numerous case reports displaying the adverse events secondary to colon irrigation, ranging from abdominal pain, vomiting, nausea, diarrhea, bowel perforation, electrolyte imbalance, renal dysfunction, and, as seen in this case, retained rectal foreign bodies. A systematic review published by the *American Journal of Gastroenterology* in 2009 concluded that there is no evidence to support colon hydrotherapy's use to promote any form of health and recommended against this modality due to the risks listed above.

Case Resolution

This is not your typical rectal foreign body case, as the structure retained within the rectal cavity is not definitively known, the patient is anticoagulated, and he recently underwent a procedure that is not well known to most medical professionals. The patient was unable to initially tolerate a rectal examination due to the severity of pain. One attempt was made for retrieval after the foreign body was identified on CT scan. The patient was given 100 mcg of fentanyl beforehand to allow inspection, but the foreign body was not able to be visualized. Upon digital insertion, the patient again was unable to tolerate further examination due to pain. Colorectal surgery was consulted and the patient was taken to the operating room where he required high levels of sedation. He was placed in the lithotomy position, underwent sedation, and 2 metal foreign bodies were retracted using Debakey forceps. ✶

Should We Be Vaccinating Our Patients Against Influenza?

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Influenza is among the top 10 causes of death in the United States,[1] with more than 700,000 flu-related hospitalizations annually and 50,000+ flu-related deaths.[2] The annual economic burden of influenza has been estimated to be $87 billion.[3] Despite these statistics, influenza vaccination coverage has been shown to be poor, with only 37.1% coverage for the 2017-2018 influenza season. This is a 6% drop from the 2016-2017 season,[4] raising alarm for improved efforts to address poor vaccination compliance.

Emergency department personnel are uniquely positioned to vaccinate a substantial number of patients who would not otherwise be vaccinated, including many high-risk populations. This represents a substantial missed opportunity as 2.8 million patients seen in the ED annually who are at high risk for complications from influenza leave without being vaccinated,[5] despite such initiatives for emergency department-based vaccination programs being supported by national organizations. A 2015 ACEP Policy Statement supports the immunization of high-risk patients in the emergency department against influenza,[6] and more broadly, ACEP has recommended influenza vaccination of hospitalized patients for more than 20 years.[7,8]

Only 36% of adults aged 65 and older receive the influenza vaccine annually, a population which is the fastest growing proportion of emergency department patients[9] and a population particularly at risk for influenza related complications.[10] Influenza vaccination of the elderly can have a profound impact, with studies showing upwards of a 75% reduction in death and up to a 39% reduction in hospitalization.[11] For the institutionalized elderly there is a 50% reduction in hospitalization.[12] The FLUVACS Trial, a prospective randomized study, demonstrated a 23% relative risk reduction for severe ischemia, nonfatal myocardial infarction and cardiovascular death among those immunized against influenza compared with unimmunized matched controls.[13]

Influenza outbreaks have been associated with a substantial increase in ED utilization for those 65 and older for influenza related infections and its complications. In fact, it has been shown that for every 10 new cases of influenza, there is a 1.5% increase in the proportion of elderly patients in the ED who presented with influenza-related infections and upper respiratory infections.[14] Prevention of influenza can offset the surges seen in ED utilization during the 2009 H1N1 pandemic.[15-20] During times in which the CDC declared "widespread influenza activity" there is a significant increase in department resource use,[21] particularly among patients with underlying respiratory illness.[22] Additionally, influenza outbreaks are associated with increased ambulance diversion.[23] For every 100 cases of influenza per week, ambulance diversion increased by 2.5 hours per week. Taken together, these data suggest that influenza infections contribute to all the complications associated with ED overcrowding.

Differences in perspectives regarding vaccination are not uncommon among nurses and physicians.[24-26] While survey data collected from ED nurses suggests a negative view of influenza vaccine programs in the department,[27] it has been shown that such vaccination implementation programs can be effective, easily administered, and do not have a detrimental effect on quality indicators.[28] Research shows higher vaccine rates among resident and attending physicians and lower rates among nursing staff,[24,29] which may help explain these divergent findings. Similarly, clinicians who receive vaccinations are more likely to recommend them for patients.[30-32] From a patient perspective, survey data suggests that most unvaccinated ED patients would be amenable to vaccination if it were offered.[33,34]

While concerns have been raised about the feasibility of ED-based vaccination programs - such as perceived disruption of department flow and lack of time[29] - these programs are well-received by emergency physicians, nursing staff, and patients alike.[28,35]

Nearly half of U.S. medical care is now provided through the ED.[36] and emergency physicians represent a safety net.[37] This is not a new concept. The ED has a front-line role in combating vaccine-preventable illness. In fact, due to recent increases in the number of pertussis cases the CDC recommended TDAP in lieu of Td for those requiring tetanus vaccination as part of wound management,[40] and this approach has been found to be beneficial and cost effective.[41]

ED vaccination strategies have been successful and reimbursable and are advocated by several major clinical practice advisory groups. This represents a cost-effective opportunity to address the wellbeing of an underserved population, without disrupting workflow. ✶

References available online

25-year-old STEMI
What a Paradox!

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University at Buffalo

A 25-year-old, thin West African male without any reported medical history presented for a chief complaint of chest pain. He was in his usual state of health when he developed an acute-onset crushing central chest pain that woke him up from sleep. He had associated shortness of breath with no prior history of similar symptoms. Pre-hospital care included 325 mg of aspirin and 2 sublingual nitroglycerin without changes in his symptoms. Pre-hospital EKG was concerning for possible inferior ST-elevation myocardial infarction (STEMI).

On initial presentation, the patient was in moderate distress, ill-appearing, and diaphoretic. Vital signs showed a heart rate of 58, blood pressure of 131/77, respiratory rate of 28-32, and saturating at 100% on room air. An EKG in the ED was obtained (Figure 1), which showed improvement of the initial ST-elevations in the inferior leads with inverted T waves in the inferior and anterolateral leads. While on telemetry monitoring, the patient went in and out of slow atrial fibrillation and high-degree heart block resulting in bradycardia in the 30s and associated lethargy. Interventional cardiology was consulted, but they did not recommend cath lab activation. Instead, they recommended further workup in the ED.

Bedside echocardiography was done showing subtle inferior wall hypokinesis with no significant right heart strain and no apparent aortic pathology. Interventional cardiology was called again, but they did not believe the patient warranted catheterization lab given his age and lack of cardiac risk factors. Repeat EKG was performed (Figure 2) which showed dynamic changes in ST-segments, T waves, and new arrhythmias. The patient continued to be symptomatic with chest pain and shortness of breath. Additional discussion with the interventional cardiologist was had regarding the patient's continued presentation and findings. They continued to advise medical management and workup.

Additional history revealed the patient was a daily smoker with occasional marijuana use. The patient recently took a road trip to Texas 5 days prior. Although he was originally from West Africa, he had not had any recent foreign travel. There was no significant family history reported. All immunizations were up-to-date.

Given the initial limited history and presentation, a case for atraumatic chest pain in a young adult elicited a broad range of differentials. A comprehensive workup was obtained and the patient was started on a heparin drip. A CT head scan was unremarkable and CT chest scan was negative for pulmonary embolism (PE). The patient was admitted to the cardiac intensive care unit (CCU).

On CCU's initial evaluation, he continued to be bradycardic with an intermittent high-degree heart block that was unresponsive to atropine therapy. The patient maintained good perfusion and mental status throughout this course. The second troponin at 3 hours increased from 0.07 to 1.38 and a CK-MB of 7. The decision was made to transport the patient to the cath lab for an angiogram. Results showed abrupt cut-off of the inferoapical segment of the left anterior descending artery (LAD), which was likely secondary to a clot embolism (Figures 3, 4). Preserved left ventricular systolic function with akinesis of the apical and inferoapical wall segments was noted on ventriculogram (Figure 5). The right coronary artery (RCA) was unremarkable. Given the extreme distal positioning of the embolism, no further interventional therapy was done at that time. Further workup with transthoracic echocardiogram complete with bubbles demonstrated Chiari network and a small patent foramen ovale (PFO). Extremity Dopplers showed no deep vein thrombosis. The patient's blood test was negative for coagulopathies, viral or bacterial etiologies, and toxicology testing was negative for cocaine use.

Introduction

Patent foramen ovale (PFO) is traditionally recognized as a component of fetal circulation and closes shortly after birth. However, roughly 25-30% of individuals live with a PFO, with autopsies reporting prevalence at about 27%.[5] A PFO potentially increases in size with each decade of life, subsequently increasing the risk of paradoxical embolism. Paradoxical coronary emboli are rare phenomena and account for approximately 5-10% of all paradoxical emboli.[11] Various case reports have noted occurrences of paradoxical embolism in those who are at risk for thrombus formation including patients who are pregnant, have coagulopathies, history of cancer, or congenital heart disease, and even those who are mechanically ventilated.[1,2,4,6,10] In the setting of a young healthy patient with no significant coronary artery disease, they are rarely reported and, thus far, there is no concrete epidemiology on the incidence or prevalence of acute myocardial infarction (AMI) caused by a paradoxical embolus.

Pathophysiology

Paradoxical embolism originates from the venous vasculature and transverses through an intracardiac (eg, PFO) or pulmonary shunt when right atrial pressure exceeds left atrial pressure (such as during Valsalva maneuvers) resulting in shunting of blood and passage into the systemic circulation.[13]

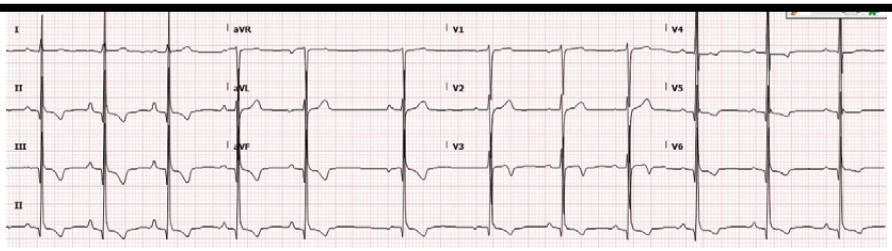

FIGURE 1. Initial ED EKG

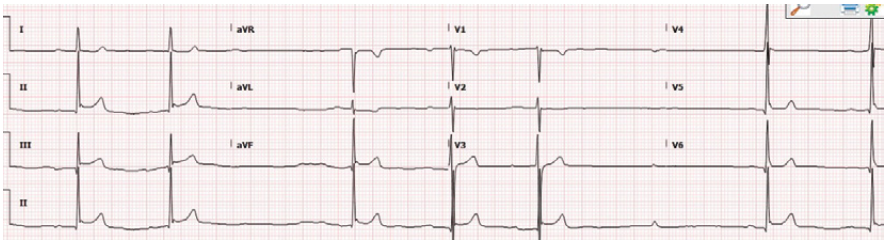

FIGURE 2. Repeat EKG

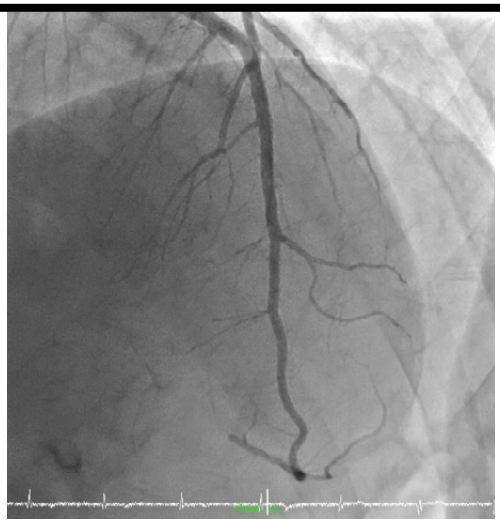

FIGURE 3. Left Anterior Descending Artery

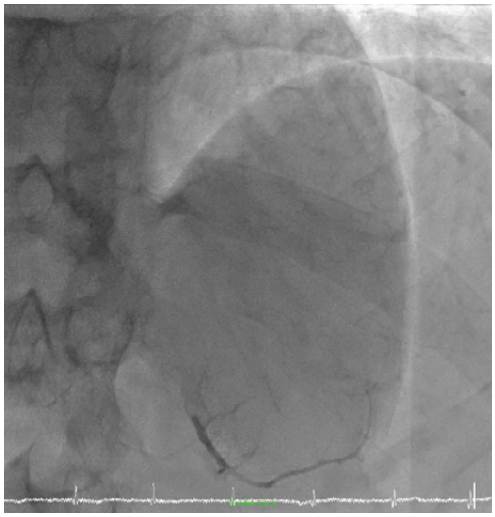

FIGURE 4. Distal Left Anterior Descending Artery

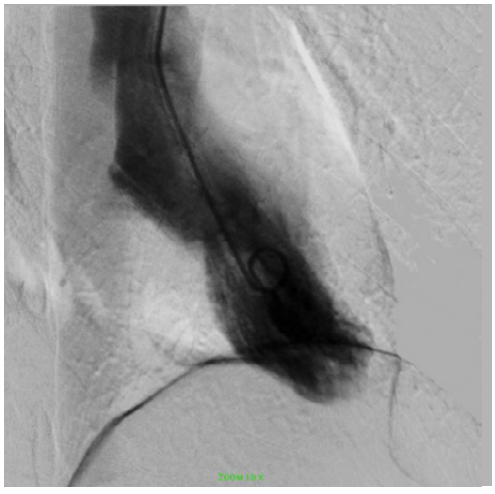

FIGURE 5. Ventriculogram in Systole

Paradoxical embolism in patients with PFO has been better studied and documented in cases of cerebrovascular accidents and peripheral vascular occlusions with fewer data looking at AMI. Prior literature evaluating the incidence of PFO in patients with AMI and angiographically normal coronary arteries demonstrated no significant increase in prevalence of PFO, suggesting that paradoxical embolism through a PFO is a rare cause of AMI.[3] Similarly, to date, it has not been shown that having a right-to-left shunt is an independent risk factor for AMI.[7] This is likely due to the low frequency of these events along with the underreported amount of cases, which make it difficult to establish any controlled studies of significant power.

Additionally, there is always the possibility that the presence of a PFO was found incidentally without a true cause-effect relationship with the AMI. This is a frequently debated topic as a visualization of a thrombus within the PFO is not always feasible. However, we do know this is a plausible physiologic mechanism. Prior literature has evaluated PFO as a potential pathway into additional vasculatures, such as the coronary arteries, which included the histopathological evaluation of the thrombus.[8,9,13]

Case Discussion

This case presented with multiple pathophysiologic points that did not follow the typical statistical and physiological variant for ACS. The patient had a seemingly unprovoked embolus that formed and traveled through a small PFO into the left heart during a momentary right-to-left shunting process. The embolus subsequently was not pushed out into the systemic circulation during systole and instead went into the left coronary artery during diastole when the ventricle relaxed and allowed for filling of the coronary arteries. Furthermore, the initial primary concern was for RCA involvement. This patient had dynamic EKG changes along the inferior leads, bedside echocardiogram was notable for inferior wall motion abnormality, and intermittent episodes of high degree heart block on telemetry monitoring. Approximately 90% of the population's AV node is supplied by the RCA. This patient, however, had a LAD that supplied down to the inferoapical wall, which reproduced similar findings of RCA involvement.

Currently, there is a dearth of literature to guide initial management of a paradoxical coronary artery embolism causing AMI. Standard treatment and prevention also remain unclear with guidelines that do not take non-cerebral embolic events into account. Nevertheless, options to be considered are percutaneous angioplasty/stent placement, thrombolysis, or thrombus aspiration embolectomy.

Case Conclusion

The patient continued to remain stable and was further monitored with anticipation of PFO closure. Paradoxical coronary artery embolism through a PFO causing AMI is rare and often underdiagnosed. A high degree of clinical suspicion should be had in patients with atraumatic chest pain who have low or no risk factors for coronary artery disease. ✶

References available online

Pressure! Pushing Down on Me, Pushing Down on You

The Underlying Danger of Pressure Washer Injuries

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Pressure washer injuries are rarely seen in the emergency department but have major consequences if underlying injury or extent of the injury is missed or overlooked. Although underlying trauma is normally examined and ruled out by CT scans, initial x-rays can help effectively triage and diagnose deeper soft tissue damage. In this case, initial x-rays in the emergency department were able to demonstrate significant soft tissue trauma, thus enabling the patient's treating physicians to quickly initiate appropriate antibiotics and analgesics and begin trauma consultation and admission.

Introduction

We present a patient with extensive underlying tissue damage diagnosed on initial x-rays after sustaining a pressure washer injury. We report here how a high-pressure injection injury with only superficial physical exam findings can have significant damage that extends beyond the wound or injury itself secondary to shearing forces and energy dispersion after the initial insult. Diagnosis of underlying trauma in the emergency department is usually completed by CT scans, however, we demonstrate that initial use of x-rays can be an efficient way to triage and begin appropriate treatment for high-pressure injection injuries and to effectively diagnose underlying trauma. This is important in the emergency department since major complications of these types of injuries include acute extremity compartment syndrome, infection and can lead to limb amputation.

Case

A 32-year-old male presented to the emergency department with a chief complaint of right forearm pain. The patient, who is left-handed, was using a pressure washer at work, about 20 minutes prior to emergency department arrival, when it slipped and hit the volar aspect of his right forearm. On arrival,

the patient complained of severe pain in the volar aspect of his arm extending through his wrist and elbow, which was made worse with the slightest extension movement. He held his wrist in a flexed position secondary to pain. The patient noted that he was otherwise healthy and denied any other medical problems. On further questioning he did not know if his tetanus was up to date.

On inspection, the patient endorsed pain from his right elbow down to his hand. He had a 1-2 cm circular superficial injection wound on the distal third of this volar forearm without surrounding erythema or edema. He had full range of motion of his fingers, wrist, and elbow. His sensation and motor strength were intact. Due to suspicion for extensive subcutaneous injury at bedside, x-rays of the patient's right arm were ordered immediately after initial evaluation. Examination of the images showed extensive subcutaneous air dissecting through multiple tissue planes extending from the initial injection wound location both distally to his wrist and proximally down his forearm. This can be a common finding after high-pressure injection injuries. The patient was then started on intravenous antibiotics, unasyn and gentamicin, analgesia, and trauma surgery was consulted. The trauma team examined the patient at bedside and he was admitted for further monitoring with frequent neurologic checks and pain control. Ultimately, the patient received a fasciotomy and wash-out secondary to worsening compartment pressures and concern for underlying infection. Several days later he was discharged with a fully intact neurologic function and outpatient follow-up.

Discussion

High-pressure injection injuries most often occur on the patient's non-dominant hand or arm.[1] The degree of underlying damage has a high risk of being underestimated on presentation secondary to small or superficial surface wounds. Up to 30% of pressure injuries eventually require amputation and expert surgical opinions in case reports even recommend that all high-pressure injection injuries receive a fasciotomy within six hours from injury onset in order to decrease amputation risk.[2] Pressure washer injuries, like other high-pressure injection injuries (including paint, gas, air, and grease), can inject gas or liquid into the surrounding and underlying soft tissues increasing risk for infection.[3] The foreign material dissects quickly through tissue planes, increasing pressure within the muscle compartments and restricting blood flow due to extraluminal pressure on blood vessels. Additionally, the disseminated foreign material can seed bacteria throughout the affected areas, creating a risk for deep space infections and worsening inflammation. Several case reports throughout the United States and Europe illustrate worse outcomes for those with high-solute and non-organic solvent injections as these exposures require larger debridements and serial washouts to remove foreign material.[4] For these reasons, patients should be admitted to the hospital for continuous monitoring and serial physical and neurologic exams even if initial imaging does not reveal any significant underlying damage.[5-6] Consensus statements from both trauma and plastic surgeons recommend broad spectrum antibiotics as well as updating tetanus status.[7] After admission, physicians may have difficulty controlling severe pain from these injuries; however, digital and other nerve blocks should be avoided as they can mask worsening pain or loss of sensation which are often the first signs of compartment syndrome.

Conclusion

Although high-pressure injection injuries may only leave small superficial wounds, they can create widespread multilayer soft tissue injuries, deep-space infections, and lead to compartment syndrome. Even if x-ray and CT scans are negative, it is highly recommended that these injuries receive urgent consultation, admission, antibiotics, and analgesia. ✶

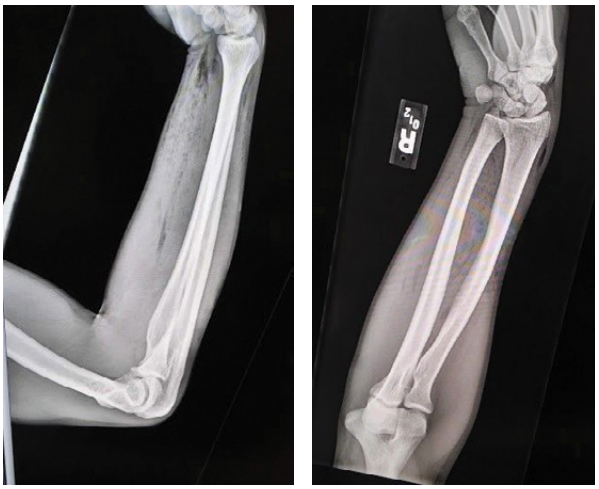

FIGURE 1. Right forearm lateral x-ray showing extensive subcutaneous air beyond superficial wound location outlined by the white circle.

FIGURE 2. Right forearm anterior-posterior x-ray showing subcutaneous air into the wrist and hand.

TAKE-HOME POINTS

1. If a pressure washer or other high-pressure injection injury is suspected, consider initial screening x-rays instead of CT scans to detect deeper, underlying damage.
2. However, always maintain a high degree of suspicion for high-pressure injection injuries regardless of imaging findings. Early fasciotomy may lead to improved outcomes.
3. All high-pressure injection injuries require an updated tetanus status, broad-spectrum antibiotics and effective analgesia. Nerve blocks should be avoided a they may cloud serial neurovascular and compartment checks.
4. Remember, even if initial imaging does not show signs of underlying damage, it is important to still consider deeper injuries in these patients as they will require admission for close monitoring and IV antibiotics.

References available online

Non-Sustained Ventricular Tachycardia

Following Modified Valsalva Maneuver for Atrioventricular Nodal Reentry Tachycardia

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Supraventricular tachycardias (SVTs) are a diverse group of dysrhythmias that prompt patients to seek emergency care because of palpitations. The "Modified Valsalva Maneuver" has recently been reported to be a safe method of terminating a subset of these cases. Here we present the case of a 47-year-old female who arrived at the ED in a hemodynamically stable atrioventricular nodal reentry tachycardia (AVNRT). She was cardioverted utilizing the modified Valsalva maneuver, which was complicated by a period of non-sustained ventricular tachycardia (NSVT).

Background

The term supraventricular tachycardia is a catch-all phrase that refers to any rapid dysrhythmia that originates from a source superior to the ventricle. This includes atrial sources such as flutter and atrial fibrillation. More recently the term has come to colloquially refer more to atrioventricular reciprocating tachycardia (AVRT) such as that resulting from Wolff-Parkinson-White (WPW) and AVNRT.[1] These rhythms alone rarely cause significant hemodynamic compromise as they most frequently occur in younger patients without structural heart disease, but they can certainly cause symptomatic palpitations, which is usually the presenting complaint.[2] In the rare cases of hypotension, altered mentation, or other findings consistent with hemodynamic instability, electrical cardioversion is the appropriate initial therapy. However, for most patients, there are a variety of less invasive modalities. Vagal stimulation has long been an accepted first-line therapy for breaking supraventricular tachycardias in hemodynamically stable patients and the recent publication of the REVERT trial[3] has led to increased awareness of the modified Valsalva maneuver as an effective intervention for terminating these dysrhythmias.

Case

A 47-year-old female with a medical history only significant for recurrent SVT previously responsive to vagal maneuvers presented to the ED with a chief complaint of a rash (diagnosed as shingles). While driving to the hospital she developed (presumably unrelated) palpitations. EKG on arrival revealed a narrow complex tachycardia with a rate of 123 with retrograde P-waves consistent with AVNRT (Figure 1). The patient noted mild discomfort and palpitations but had normal blood pressure and no other concerning findings. The patient was placed on the cardiac monitor and cardioversion was attempted utilizing the modified Valsalva maneuver as described by Appelboam, et al.[3] The procedure initially appeared successful, with a brief pause consistent with AV nodal blockade, but patient then developed non-sustained monomorphic ventricular tachycardia for a total of 10 beats, followed by a brief junctional escape rhythm, then resumption of AVNRT (Figure 2). Given this more malignant dysrhythmia, IV access was established, laboratory studies were sent, and the patient was attached to a defibrillator and planned for admission. A vagal maneuver was then

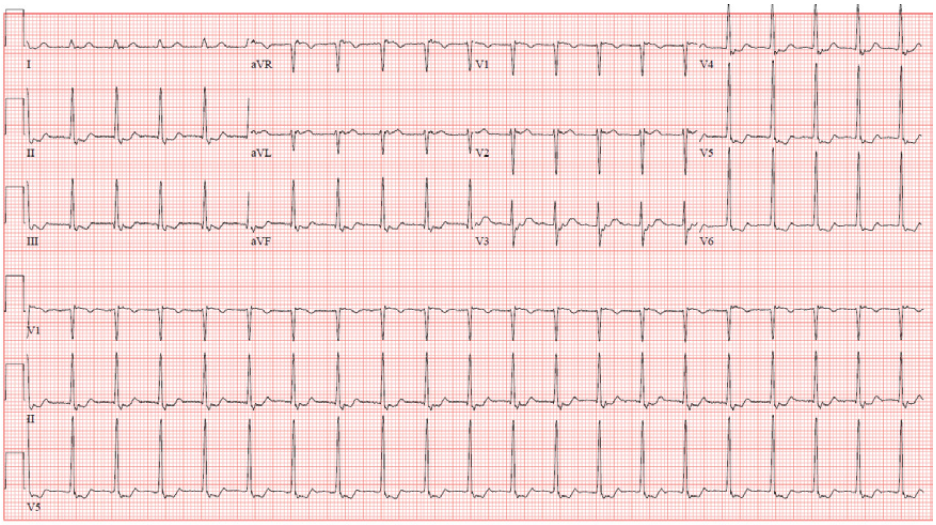

FIGURE 1. Initial EKG, narrow complex tachycardia with rate of 123 bmp, notable for retrograde P-waves buried in the ST segment.

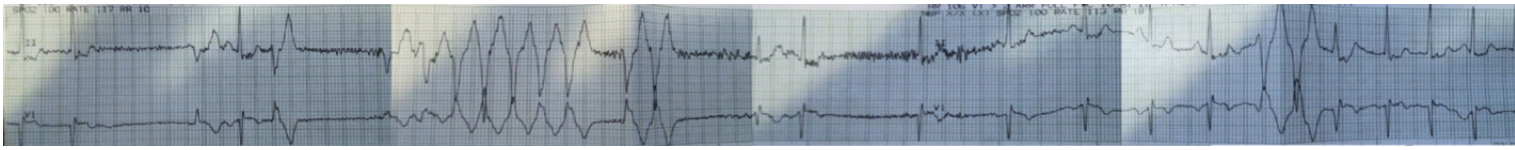

FIGURE 2. Rhythm strip immediately post cardioversion, as captured by telemetry monitor.

The term supraventricular tachycardia is a catch-all phrase that refers to any rapid dysrhythmia that originates from a source superior to the ventricle.

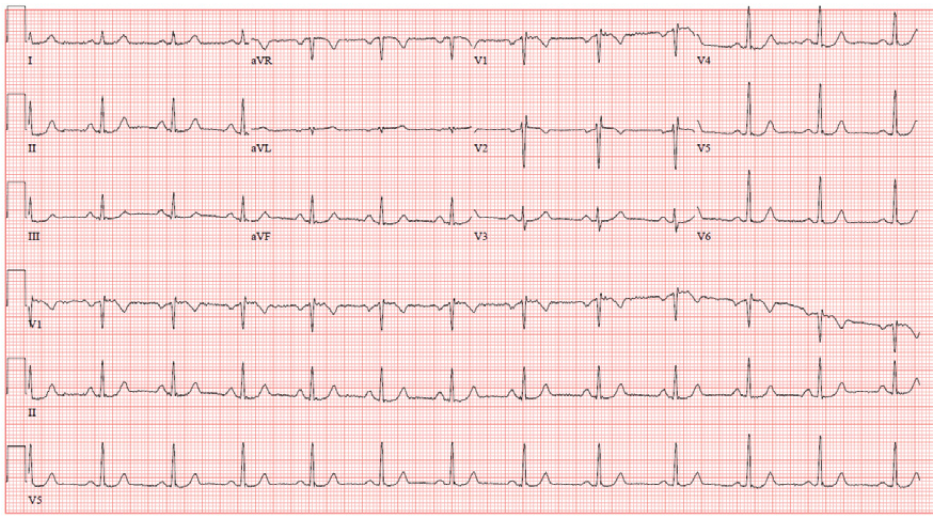

FIGURE 3. EKG after second (successful) cardioversion; patient remained in this rhythm for the rest of her hospital course.

attempted, this time a simple Valsalva without leg-raise modifications, and the patient was successfully cardioverted to sinus rhythm without additional incident (Figure 3). Blood work was unremarkable with normal electrolytes, troponin, and thyroid function, and she was admitted overnight for cardiology evaluation. Subsequent testing during admission was notable for peak Troponin I of 0.10 ng/mL several hours after the event, but there were no further significant dysrhythmias noted on telemetry. The patient's previous records were obtained, including an echocardiogram that did not show evidence of hypertrophic cardiomyopathy. The patient was offered an ablation procedure but elected to follow up with her cardiologist and has had no further visits within our hospital system.

Discussion

Symptomatic supraventricular tachycardias (SVTs) are a common phenomenon, with a documented incidence of 35 cases per 100,000 people per year.[4] Vagal maneuvers, including carotid massage, diving response, and Valsalva, previously have been used, with success rates from as low as 6% to a high of 54%.[1] The mechanism of vagal response is likely due to post-synaptic innervation of the atrioventricular node via parasympathetic ganglion in the epicardium.[5] While not always successful, the procedure is generally very well tolerated and there rarely adverse events. There are a limited number of case reports noting malignant dysrhythmias after vagal stimulation, and only 2 well-documented occurrences found through a search of the literature after treatment for SVT, neither of which resulted in any permanent harm.[6,7]

The modified Valsalva maneuver as first introduced by Appelboam, et al., in 2013 adds a period of passive leg raise after a "standardized strain" to further increase venous return. In theory this leads to stronger vagal stimulation and should increase the success rate of the procedure (indeed, in their paper cardioversion rate with the new technique was 43% vs 17% in the control group). However, this perhaps raises concerns that the vagal nerve may be overstimulated. While the rate of adverse effects in the original paper was not significantly different between the 2 groups (and most events were minor, such as mild nausea or transient muscle soreness from straining), the treatment group had 5 "electrograph captured events" (which were not further elucidated) vs 0 in the control group (no P value reported). Despite these concerns, the rate of significant adverse events with any form of vagal maneuver remains extremely low, and they should remain the first-line therapy in younger patients who are hemodynamically stable, per AHA class 1 recommendations.[8] Exercise caution, however: Despite the appearance of stability, patients presenting with symptomatic SVT have, by definition, abnormal electrical conductivity through the heart and should always be treated with an appropriate level of caution.

Conclusion

While the modified Valsalva maneuver offers a simple, effective, minimally invasive, and overall very safe option for the treatment of supraventricular tachycardias, it is important to remember that the procedure alters the electrical conduction pathway through the atrioventricular node via vagal mediation. Strongly consider appropriate precautions, such as establishing IV access and placing defibrillator pads prior to attempting the procedure, in case a more malignant dysrhythmia develops, as it did in this case. ✶

References available online

The Old, Cold Trauma Patient
At Risk of Anchoring Bias

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The treatment of geriatric trauma patients in the ED can be challenging because of concomitant medical pathology. Care of geriatric patients is further complicated by end of life wishes, which are often not immediately known. This case describes a geriatric patient who presented as a critical trauma but was found to have a severe underlying medical issue. She was stabilized, and invasive resuscitation measures were avoided until her wishes were known. In this case we discuss anchoring bias in trauma patients and end of life care for geriatric patients in the ED.

Background

The number of geriatric patients who visit the ED is increasing. Many of these patients present as trauma activations, while also having serious medical conditions. **It is important to avoid anchoring on a patient's "trauma" status in order to avoid missing important medical diagnoses.** In this case, we discuss a geriatric patient who presented as a trauma; however, that trauma was mild and secondary to a more critical medical condition. We also discuss anchoring bias in trauma patients as well as end of life care for geriatric patients in the ED.

Case

A 90-year-old female presented as a critical trauma to the ED. The patient arrived via EMS, who reported that she fell at home and was found minimally responsive on the floor by her son. She was last seen normal 15 hours before presentation. EMS noted her to have signs of a head injury, hypotension, a Glasgow Coma Scale (GCS) of 9, and glucose of 49 mg/dL. EMS administered glucagon and 10% dextrose en route, with no change in mental status.

On arrival to the ED, vital signs were heart rate 65 bpm, blood pressure 84/49 mmHg, and oxygen saturation 90% on room air. The patient was responsive only to pain. The primary survey revealed she was extremely cold to the touch. Her exam was otherwise notable only for a parietal scalp laceration. The extended focused assessment with sonography in trauma (eFAST) was positive for bilateral pleural effusions.

Soon after, the patient became bradycardic to 32 bpm and was noted to be in complete heart block. The ED attending discussed with the trauma surgery attending that this was most likely a medical patient who fell, instead of a primary trauma patient. The trauma surgery attending deferred to the ED attending, who transitioned to medical resuscitation. The patient was given oxygen via non-rebreather mask, 50% dextrose, atropine, low dose intravenous epinephrine, bicarbonate, empiric antibiotics, peripheral norepinephrine infusion, and a Bair Hugger® was placed for hypothermia. The patient's pulse and blood pressure subsequently improved.

Laboratory work-up was notable for anemia, lactic acidosis, and elevated creatine phosphokinase (CPK) (Table 1). After stabilization, the patient was transported for imaging, where computed tomography (CT) of the head showed a small frontal hematoma and chest x-ray showed moderate bilateral pleural effusions (Figure 1). Additional

TABLE 1. Notable Laboratory Values

Lab item	Value
Sodium	131 (136–145 mmol/L)
Bicarbonate	16 (21–31 mmol/L)
Anion Gap	18 (2–12 mmol/L)
Creatinine	2.9 (0.6–12 mg/dL)
Blood Urea Nitrogen	85 (7–25 mg/dL)
Glucose	294 (85–125 mg/dL)
AST	124 (13–39 U/L)
CPK	1,783 (30–223 U/L)
Lactic Acid	8.5 (0.5–2.3 mmol/L)
White Blood Cells	6.2 (4.0–10.5 thousand/µL)
Hemoglobin	7.2 (11.5–15.0 g/dL)
Hematocrit	21.4 (34–44%)
Platelets	65 (150–400 thousand/µL)
Prothrombin Time	38.7 (12.2–15.0 sec)
INR	3.94 (0.87–1.14)
Partial Thromboplastin Time	59.2 (24.9–34.5 sec)
CKMB (repeated)	94.2, 133.8 (0.0–5.0 ng/mL)
Troponin I (repeated)	0.3, 0.4 (0.0–0.3 ng/mL)
Thyroid Stimulating Hormone	1.510 (0.450–4.120 µIU/mL)

mmol = millimoles, L = liter, mg = milligrams, dL = deciliter, U = units, µL = microliter, sec = seconds, ng = nanograms, µIU = micro-international units, AST = aspartate aminotransferase, CPK = creatine phosphokinase, INR = international normalized ratio, CKMB = creatine kinase-muscle/brain

CT imaging studies were obtained with no significant further findings. The patient's son arrived and provided information that the patient had a history of congestive heart failure and a pacemaker with an outdated battery and explained that his mother chose not to have the battery replaced. The son confirmed that she did not desire any lifesaving intervention. The patient was then transitioned to comfort care, with the cessation of vasopressors and initiation of a fentanyl drip for pain, and was placed on hospice.

Discussion

This case illustrates the importance of avoiding anchoring bias in designated trauma patients in the ED. The clinical environment of the ED makes the use of protocols and algorithms helpful; however, these tools can potentially contribute to cognitive biases.[1–3] A common cognitive biases in medicine is the anchoring effect, in which the clinician fixates on a diagnosis based on initial presenting features, failing to adjust when new information becomes available.[3–5] A situation in which emergency physicians are prone to anchoring bias is in trauma code activations. Trauma care has been noted to be particularly susceptible to medical error given the combination of unstable patients, incomplete information, and multiple medical services and providers working together.[6] Additionally, trauma activations can be susceptible to anchoring bias because of the protocolized framework of trauma assessment and intervention. However, some of these pre-determined trauma patients may also have critical medical conditions that can be overlooked if the treatment team focuses exclusively on the patient as a trauma case. It is important that emergency physicians maintain a broad differential and direct the care team for the patient's benefit as new information arises. In this case, it was quickly realized that the patient was not a critical trauma patient, but rather had serious medical conditions that likely led to a fall from standing. If the trauma protocol had been blindly followed, critical medical interventions may have been missed or severely delayed in order to follow protocol. This was averted by avoiding the inclination to anchor on the patient's status as a "trauma" patient.

This case also highlights the value of considering end of life care in geriatric patients in the ED. Geriatric patients (age > 65) make up an increasingly large percentage of patients presenting to the ED, leading to an expanding role of emergency physicians in end of life discussions. This necessitates a focus on the ethics of resuscitation in geriatric patients.[7] More than half of geriatric patients visit the ED in the last month of their life; of those, most will be admitted, and many will die in the hospital.[8] Resuscitation of critically ill geriatric patients in the ED can be challenging both medically and ethically, as the principle of "do no harm" can be difficult for physicians to navigate, especially if the patient's wishes are not immediately known.[9] Numerous studies have found that geriatric patients resuscitated in the hospital have worse outcomes than younger patients, with a recent large study of geriatric patients intubated in the ED reporting that 33% died in the index hospitalization and only 24% were discharged home.[10–12] **With the risks of aggressive resuscitation in the elderly, it is important for emergency physicians to discover patients' end of life wishes.** This task is complicated because the majority of geriatric patients do not have an advance directive upon presentation to the ED, they present to a hospital different than the hospital they normally utilize, and/or the patient is often not able to personally discuss their wishes.[13] In the present case, the patient's end of life wishes were unknown, yet aggressive and unnecessary invasive measures were avoided. When her son arrived, he was able to confirm that the patient had an underlying severe medical disease and clarified the patient's end of life goals, allowing the physicians to honor the patient's wishes. **Because this case was approached conservatively, it was feasible to both stabilize the patient and respect her unknown wishes.**

Once the patient's wishes were verified, the patient was transitioned to comfort care and hospice placement. Initiation of palliative measures in the ED has been shown to decrease length of stay, increase patient quality of life, and increase patient satisfaction.[14] Though it is often challenging to identify candidates for these measures, when patient wishes are known, initiation of palliative measures increases the quality of care provided to these patients in the ED. This patient was able to be discharged home with her son, avoiding intubation and a stay in the intensive care unit.

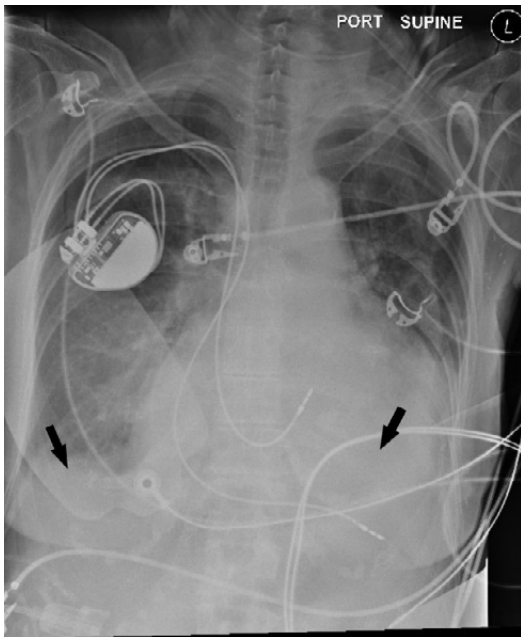

FIGURE 1. Chest x-ray showing bilateral pleural effusions (arrows).

TAKE-HOME POINTS

1. Be mindful of the tendency toward anchoring bias, especially in patients who arrive as trauma activations.
2. When patient wishes are unknown, strongly consider the risks and benefits of aggressive resuscitation in geriatric patients, as aggressive resuscitation has been shown to lead to worse outcomes in the elderly population.
3. When appropriate, seek to initiate end of life care measures in the ED, which has been shown to increase patient quality of life and improve patient experience.

Conclusion

One day after admission, the patient's condition had stabilized; however, the patient remained unresponsive. The patient was discharged home on full-time hospice care with her son. ✶

References available online

Progressive Weakness in a Child

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Juvenile dermatomyositis (JDM) is a rare cause of progressive proximal muscle weakness in the pediatric population that typically presents with symmetric muscular weakness and classic dermatologic findings. JDM must be considered early in acquired weakness in the pediatric patient, as early treatment can reverse symptoms and prevent illness progression.[1] We present the case of a 6-year-old male experiencing significant weakness and hyporeflexia, who was found to have advanced JDM.

Case

A 6-year-old male presented to the pediatric emergency department with significant weakness of all extremities. He developed leg pain and weakness 6 weeks prior, which progressed to dragging his feet when walking. Three weeks after onset, while vacationing in Central America, the pain resolved. However, lower extremity weakness progressively worsened and the patient had a new onset of upper extremity weakness. He was unable to ascend stairs, sit from a supine position, elevate his arms above his head, or dress himself. At that time, he was hospitalized and presumptively treated for Guillain Barre Syndrome (GBS), initially displaying improvement following intravenous immunoglobulin therapy and physical therapy. He later developed a faint red rash on the upper half of his face, along with skin-colored papules on his elbows. Two weeks prior to the ED presentation, he began experiencing dysphagia, and a week prior, he developed abdominal pain and constipation. During symptom progression, there was no fever, weight loss, vision change, urinary change, vomiting, or diarrhea.

On arrival to the ED, he had a temperature of 36.9° Celsius, blood pressure 92/46 mm Hg, heart rate 97 bpm, respiratory rate 20/minute, and oxygen saturation 99% on room air. The examination was significant for a quietly speaking patient reclining on the gurney, with no signs of respiratory distress. The patient was profoundly weak, with head lag, strength 4/5, and hyporeflexia 1+ in all extremities. The musculoskeletal exam was negative for swelling, erythema, or tenderness. The patient was able to ambulate slowly but could not lift himself back to the gurney. There was a faintly erythematous rash around both eyes and over the malar prominences, flesh-colored papules over the elbows, and hyperpigmented papules over the dorsal metacarpophalangeal and proximal interphalangeal joints.

Laboratory results were significant for elevated Erythrocyte Sedimentation Rate (ESR) 37 mm/h (range 0–13 mm/h), creatine kinase (CK) 454 units/L (range 30–223 units/L), aspartate transaminase (AST) 111 units/L (range 13–39 units/L), aldolase 28.8 units/L (range 3.3–10.3 units/L), and Lactate Dehydrogenase (LDH) 934 units/L (range 140–271 units/L). Normocytic anemia was demonstrated by hemoglobin 11.3 g/dL, hematocrit 34.3%, and mean corpuscular volume (MCV) 83.9 fL. Urinalysis was significant for protein 12 mg/dL (range < 12 mg/dL) and ketones (1+). White blood cells, platelets, electrolytes, blood urea nitrogen, creatinine, C-reactive protein, and alanine transaminase (ALT) were within normal limits.

Given the clinical exam and associated elevated muscle enzymes, rheumatology consultation and pelvic MRI were completed. MRI was significant for pelvic musculature and subcutaneous tissue inflammatory changes consistent with JDM, showing a diffuse T2 hyperintense signal extending from the paraspinal muscles down to the knee extensor muscles bilaterally (Fig. 1, 2).

Treatment with a regimen of high-dose steroids, methotrexate, and physical therapy commenced, and the patient progressively improved. The patient received methylprednisolone 30 mg/kg for the first 3 days, followed by daily prednisolone 50 mg and folic acid with weekly methotrexate 12.5 mg subcutaneously.

Discussion

JDM is an autoimmune, idiopathic inflammatory myopathy that affects an estimated 3.2 children per million annually in the United States.[2] It is a systemic inflammatory disease that demonstrates proximal muscle weakness

and can have characteristic skin findings of heliotrope rash, Gottron papules, nailfold capillary abnormalities, and calcinosis.[1,3,4] Dermatologic findings may not begin concurrently with muscular symptoms, sometimes occurring months prior to or even after weakness onset.[1]

Up to 80% of patients with JDM also present with signs and symptoms of systemic disease.[4] These include fever, weight loss, fatigue, arthritis, interstitial lung disease, and gastrointestinal symptoms such as nausea, pain, and constipation, which can indicate ischemic complications from bowel vasculitis.[1,4] Focal or generalized edema can occur, with both postulated as occurring secondary to vasculitis.[5,6] While the more common periorbital and peripheral edema presentations have not been associated with a poorer prognosis, the rare generalized edema presentations are strongly associated with clinical resistance to steroids.[5] Systemic capillary leak syndrome, although rare, can occur, causing hypovolemic shock, compartment syndrome, and death.[7] Involvement of the cardiac system is uncommon; however, pericarditis and subclinical cardiac dysfunction have been described in some cases of JDM.[1,4] Effects to the neurologic system are also uncommon in the pediatric population.[4]

Diagnosis is determined clinically by proximal muscle weakness, dermatologic findings, muscular enzyme elevation, and MRI evidence of inflammation of the proximal muscle groups.[1,4,8] Creatine kinase levels are not typically over 1000 units/L, so other etiologies must be investigated with significant elevation, and muscle enzyme levels can also be normal in patients with JDM[4]. Diagnosis of JDM previously relied heavily on muscle biopsy and electromyography, though these methods are now less commonly used because of their invasive nature. The yield was also a concern with these methods, as biopsy could potentially miss areas of inflammation when muscle disease was not diffuse, and electromyography necessitated cooperation of young patients.[4,8]

First-line management consists of early high dose corticosteroids, disease-modifying anti-rheumatic drugs such as methotrexate, and physical therapy.[1,4,8]

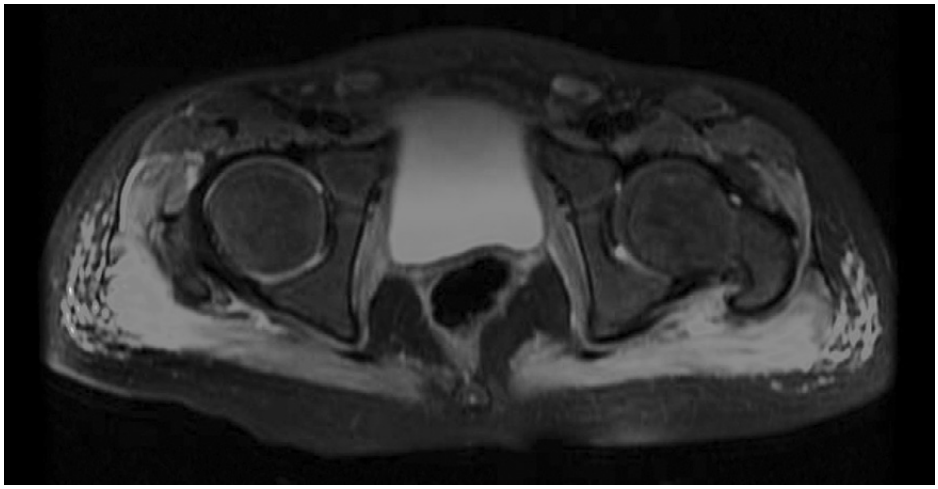

FIGURE 1

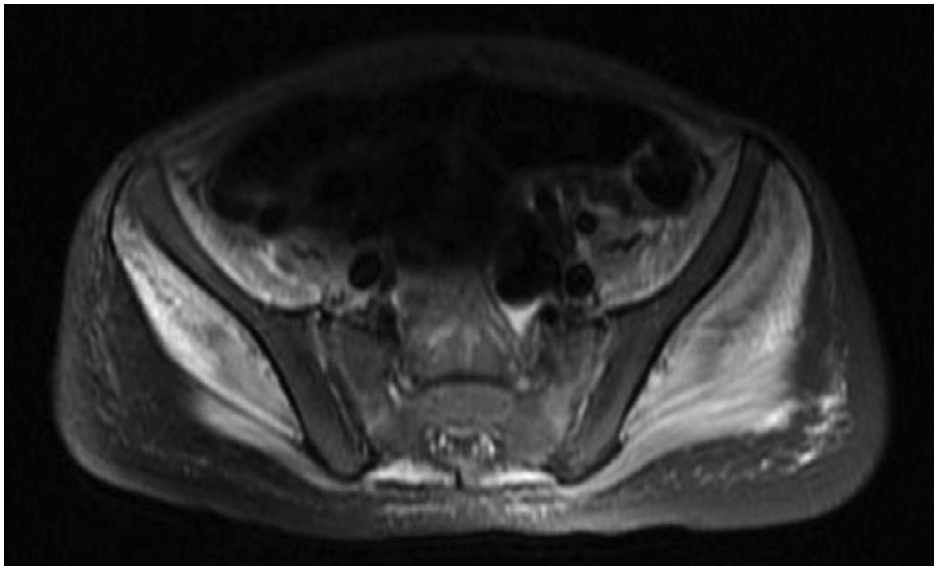

FIGURE 2

Without treatment, mortality can reach 30% and there is significant morbidity, whereas with prompt treatment, mortality is less than 2%.[1] With early treatment, patients with JDM can make a full recovery. It is also possible to develop an intermittent relapsing or chronic pattern of symptoms, with up to 50% needing treatment for longer than 2 years.[4]

Conclusion

Juvenile dermatomyositis is a treatable idiopathic inflammatory myopathy that causes progressive proximal muscle weakness and characteristic dermatologic phenomena. Prognosis is significantly improved by early diagnosis and treatment, and delay can result in severe manifestations of myopathy such as global hyporeflexia, as in our patient. ✶

TAKE-HOME POINTS

- Juvenile dermatomyositis is a reversible cause of progressive proximal muscular weakness.
- Juvenile dermatomyositis does not typically affect the nervous system. However, with advanced disease, profound myopathy can result in hyporeflexia.
- Diagnosis of juvenile dermatomyositis is by clinical exam, muscular enzyme elevation, and MRI demonstrating inflammation of the proximal muscles.
- Untreated JDM can result in up to 30% mortality.

References available online

EMS Ride-along Etiquette

DO'S AND DON'TS

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*The day starts at 8 am. It feels strange as you enter the station, as if you're entering someone else's home rather than their job. The paramedic and emergency medical technician (EMT) quickly greet you and are enthusiastic that you want to see their work. They tell you to make yourself comfortable and to ask questions when you have them. You store your belongings in an extra room and mention you brought bagels, which is met with excitement. It's not long before an alarm bell sounds, and a disembodied voice says, "*Engine 501, Medic 505, K Deck 5, Full Code, 1234 E. Main St.*" Before you can ask what a "K Deck" is you find yourself in the back of a bouncing ambulance, wondering what awaits you at the scene.*

In the cab of the ambulance, the EMT says something into the radio while the medic sits quietly tapping on an industrial-looking laptop. Now you start to get nervous. There's no team of nurses and doctors awaiting at your scene. There are no consultants, no CT scanners, no labs, nothing but what is kept in this surprisingly cramped vehicle. All that your future patient has is the EMT's and medics who were earlier so excited about your bagels. But there's no anxiety shown from the two sitting up front. As the sirens begin to wail and you can see the blue and red lights bounce off passing buildings, you wonder, "How can I be helpful? What am I allowed to do? Where do I even stand?"

How to Succeed as an EMS Observer

A certain level of uncertainty is understandable on one's first ambulance ride-along shift. The EMS setting is unpredictable and uncontrolled and thus represents an opportunity to learn medicine in a unique environment — for those prepared to make the most of the ride-along and not get lost in the chaos. It's also a privilege; a poor observer can be a drain on a crew's shift, but a good observer can be revitalizing to EMS personnel. **Here are the Do's and Don'ts to be an observer whom a crew would be happy to invite back to their shift.**

DO

1. ***Stay safe.*** From Day 1 of EMT school the question "*Is my scene safe?*" is drilled into every student's head. From environmental hazards to those presented by standing on the side of an active highway to the dangers of a domestic violence situation, EMS work carries some risk. As an observer, maintain situational awareness and follow the guidance of the crew.
2. ***Show gratitude.*** When you embark on a ride-along you are entering that EMS crew's home. As you would if you were a guest in anyone's home, make sure to respect the rules and traditions of your host. Introduce yourself to every member of the crew, ask what you can do to help with station duties, and chip in for meals (or at least offer). Many firehouses buy food in the morning and then cook lunch and dinner, so bringing some cash to contribute can be helpful. Bring bagels or doughnuts to your ride-along if it's within your means. It is by no means expected, but always appreciated.
3. ***Show interest.*** Most crews like answering questions about their jobs. An engaged observer can be a revitalizing presence, helping to combat burnout or exhaustion in even the most jaded EMT. Be engaged and genuinely interested in the calls your crew gets dispatched to handle. You might know the medicine from the ED perspective, but prehospital medicine is different, and the nuances can be valuable to learn. Just do as you would with an attending during hospital rounds, and be mindful of interrupting. Try to ask questions when you're between calls, rather than on scene, when the EMS providers are more focused on patient care or charting their call.
4. ***Know the rules.*** Depending on where one is in their training/education, your role will likely change. It's important to establish before you ever accompany a crew on a call what it is you can and cannot do as a student. Are you a pure observer? Can you assist with patient care? Can you do an H+P? Speak with the crew supervisor and the crew about this and adhere strictly to the rules that are set. If you aren't sure what you're allowed to do, just stay hands off. Sometimes malpractice and liability issues make direct patient care difficult outside of the hospital.

DON'T

1. ***Be a know-it-all.*** You might be a stellar ED resident, or an MS4 with lots of time in the ED. You may feel confident about your knowledge and abilities in emergency medicine. You should appreciate that some of this may translate to EMS — but a lot of it will not. Unless you have an EMS background, it's important to recognize that you're in unfamiliar territory as an observer. Most crews will be put off if their observer is constantly referencing "Well this is how *we* do it…" The desire to relate may be well-intentioned, but it can come across as arrogant if you haven't spent time working an unstable trauma in the back of a moving ambulance. The ED and EMS work closely together and their roles are intertwined. However, from the perspective of the ED you only get to see a few moments of an ambulance crew's work, which doesn't provide much insight into their role. As an observer, your goal should be to gain understanding about their role in caring for the patients you see. Humility is important in any learning setting, and a ride-along shift with an EMS crew is no different.
2. ***Be a fly on the wall.*** While this goes along with "show interest" and staying engaged, it bears further mentioning. To some, stepping into a fire house or an EMS station can be intimidating. It's like stepping into a stranger's home where everyone knows each other very well, and it can be easy to feel out of place. But don't let this discourage you. In the past, students have told me they have felt uncomfortable to the point they just held back on calls and kept to themselves. This is one of the worst things you can do on a ride-along shift! Crews are receptive to those who want to learn.

Riding along with an ambulance crew is a great way to see the side of emergency medicine most ED-based physicians don't get to see. Every paramedic is required to spend a number of hours in the ED during training; seeking out opportunities to see the prehospital side of patient care is a chance to increase your understanding of everyone's respective roles. The culture of EMS can be difficult to adjust to, but if you follow the do's and don'ts here, you'll be welcome as an observer and you'll gain a valuable learning experience. ✶

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Why You Should Participate in Quality Improvement

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What is Quality Improvement?

Quality improvement is "systematic and continuous action that leads to measurable improvement in health care services and the health status of targeted patient groups."[1] While it is not a novel concept in the health care industry, quality improvement is now becoming an integral component of medical education and training. In 1999 the U.S. Institute of Medicine released the landmark report "To Err is Human: Building a Safer Health System," which was followed up 2 years later with "Crossing the Quality Chasm: A New Health System for the 21st Century."[2,3] These reports discussed the thousands of preventable medical errors that occur annually and proposed a framework for improvement. They are credited with creating awareness and have served as the foundation for work done today.[4,5] Given all the demands on time, quality improvement can seem obscure and prove challenging to incorporate into your training in a meaningful way. So, here are 5 reasons why you should participate in quality improvement during residency.

❶ *Diversity of projects*

When choosing a quality improvement project, you can focus on anything that interests you. Examples of projects we have seen through our own training or through the Quality Improvement and Patient Safety (QIPS) section of ACEP include improving sepsis metric compliance, ordering of second dose antibiotics (for boarding patients), point of care ultrasound labeling, pain control in the waiting room, improving patient satisfaction, and addressing second victim syndrome. You can identify and prioritize any area of improvement and use established quality improvement models to guide your work. Here are a few examples of quality improvement models:

- Plan-Do-Study-Act (PDSA) cycles to model improvement with small tests of change.[1]
- Six Sigma to eliminate defects and minimize variability.[1]
- Lean Methodology to eliminate waste.[1]

In short, the breadth of topics is amazingly broad and dynamic. So, identify your interest, become a content expert, and work to make things better!

❷ *Publishing Opportunities*

Time and effort put into quality improvement work should be recognized, celebrated, and shared. Sharing your success, and even your failure, may benefit not only patients but the healthcare sector as a whole. There are multiple publication platforms available for disseminating your work; but if writing a manuscript seems daunting to you, don't worry! You can present quality improvement projects as posters or oral presentations at your institution, or at regional and national conferences like ACEP and SAEM. While you are writing up your project, keep in mind that the QIPS section is always looking to highlight projects and circulate practical information to readers and they would love to highlight your projects! Find them at https://www.acep.org/how-we-serve/sections/quality-improvement--patient-safety.

❸ *Great Addition to Your CV*

Regardless of your practice setting after residency, there will be operational inefficiencies and patient care concerns that require action. Therefore, employers are continuously looking for champions of change. Going through the quality improvement process during residency will help you acquire skills that you can lean on during your career in emergency medicine. Presenting a quality poster, oral presentation or written publication is an excellent addition to your Curriculum Vitae. It demonstrates your understanding of the process, while simultaneously reflecting your innovation and personal interest. This will help you stand out when it comes time to explore job opportunities.

❹ *Improves Patient and System Outcomes*

Quality improvement work often leads to meaningful change in clinical practice, or at least identifies core issues not otherwise well studied. There was a time when stroke, myocardial infarction and trauma care was heavily fragmented. Through scientific study and quality improvement work, such diagnoses have been better integrated into highly reliable systems.[6-8] Another example, from the University of Pennsylvania, was the changing of default prescriptions from brand names to generics whenever appropriate. The result was tens of millions of dollars in savings for patients.[9] Ideas like this may seem simple, but can lead to a substantial reduction in health care disparities and improvement in patient outcomes!

❺ *Expectation of the ACGME*

The ACGME expects resident participation in scholarship activities, one of which is quality improvement. Their guiding philosophy is "all physicians share responsibility for promoting patient safety and enhancing quality of patient care…Graduating residents will apply these skills to critique their future unsupervised practice and effect quality improvement measures."[10] Even though involvement in scholarly work like quality improvement is a requisite during residency training, it can still be interesting and individualized.

So, now that you know a little bit more about quality improvement, we hope you think about getting involved. Reach out to your department leadership and see what opportunities are out there. Remember, quality improvement is a broad term, so there is sure to be a project that aligns with your interests, and there is always a need for more resident involvement! ✶

THE DUAL DEGREE DEBATE

What to Consider Before Getting an MBA as a Physician

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When people find out I have a dual MD/MBA degree, their first question is usually about what I intend to do with an MBA. When pinched for time, I usually say something glib, like "have my own parking spot." In reality, the utility of the MD/MBA dual degree is difficult to explain quickly. Moreover, the factors that go into the decision to get a second degree can be numerous, complex, and individual. I started my MBA at the end of my first year of medical school and finished it during interview season. Fortunately, I was able to pursue both degrees concurrently and finish both in 4 years. However, not all programs nor all students are the same. My path and my perspective on the dual program may not be identical to another MD/MBA, but I hope that this article can help those thinking about pursuing a concurrent business degree.

References available online

An MBA will...

Expand your knowledge base

Having a background in business or economics is not mandatory for pursuing an MBA. However, you will inevitably learn a lot more about these and related topics while pursuing the degree. As physician learners, pursuing an MBA also helped me experience a break from the science-heavy subjects and was a refreshing way to gain some knowledge about the business world. You will work with classmates who have no idea what a Kreb even is, but have a wealth of non-health care industry knowledge that is both illuminating and interesting. You will also study topics that cover a broad range that encompasses economics, design, and marketing. Your intellect will truly grow and help you understand the world outside of the hospital and textbooks.

Provide experience in the non-medical world

You won't necessarily be "interning" on Wall Street, but some aspect of your MBA training will have you working on a consulting project or similar. You will experience some semblance of what day-to-day life looks like for people who, for instance, don't live in hospital call-rooms.

Help you become a better leader AND team player

Many of the MBA classes are a combination of small-group and individual work. While basic prerequisite classes like statistics are heavily skewed toward the individual side, the vast majority have some group project or team deliverable. In the medical field, we tend to be the kids who still have traumatic memories of having to do all the work in school projects, as we are inclined toward perfectionism. The same holds true for the kinds of individuals who decide to pursue MBAs. Having so much group work, you really learn how to step up when things are a bit disorganized, or when your particular strengths and expertise are needed. At the same time, there are occasions when you have to fall back and allow others to lead and contribute. This is very true for teams in both medicine and business. However, whereas in medicine you almost expect a similar background and a well-established hierarchy, in business school, the teams are so varied and diverse, that you really have to hone both your team-building and team-leading skills.

Broaden your network

Someone once told me an MBA is all about learning how to shake hands. That's true, to an extent. For instance, during my MBA training, I had the opportunity to be in groups with and get to know attendings from my own medical school, administrators from nationally recognized companies, and even a producer from a well-known TV studio in NYC. You learn how to present yourself professionally and with confidence. You can no longer hide behind the short white coat saying, "I'm just a medical student, I don't know yet." You learn how to mingle and foster a sense of familiarity with new acquaintances. All of these are really great skills to have as a physician, particularly in a field where you have such a short period of time to build a connection with a patient. So, while part of it is the simple action of shaking hands and trying not to focus so hard on the shaking that you forget the person's name, the most important aspect is learning the skill of relationship and network building.

Teach you a new vocabulary

"Enterprise solutions," "momentum," "synergy," "risk management," "strategy," "growth," "leverage." Pepper a few of these puppies into regular conversation and you're basically a CEO. Realistically, you have to know the language to establish some baseline from which the team, unit, or company operates. We have our own medical lingo. "Pt. was NPO at MN 2/2 N/V/D s/p KUB and labs. W/u WNL. IVF d/c'd. WBAT." (Try saying that at a sales meeting and not being carted away in an ambulance for a stroke evaluation.) Memorizing MBA lexicon won't teach you all there is to know about business or leadership, just like knowing medical jargon can be accomplished by TV script writers without any actual medical knowledge. However, it WILL teach you the vocabulary to contextualize your formal and real-world learning.

Give you "street cred"

OK, maybe not *street* cred per se. However, having an MBA really shows that you mean business (sorry, I had to use that pun somewhere). It shows not only a recreational interest in administration or business, but a true dedication to the field. When I went to my hospital system's CMO as a 4th year med student and asked to follow her around for 4 weeks, she knew I wasn't looking for a vacation block. As a result, she created a 4-week, comprehensive crash course for me to learn many different aspects of hospital administration. It was a once-in-a-lifetime opportunity that would not have been granted to me had I just shown "interest" in administration without the 3 letters to back it up. A few attending-level physicians have told me that having those extra letters has helped them advance the nonclinical parts of their career as well. Many doctors are reluctant to go into administration. The concept of medicine as a business is offensive to many clinicians, but it's also our reality. If you look at the administrators at your hospital, I bet there are just a few MDs (with the majority in middle management) and mostly RNs, MHAs, CPAs, etc., in the highest positions. So, when doctors go that extra step to obtain a business degree, it shows their commitment to and interest in nonclinical aspects of the health care system, contrary to the common assumption that doctors have no desire to step into that world. It effectively puts them in the game as well.

Provide you with a plan "B"

No one goes to medical school thinking they will go through the rigors of medical training and residency only to burn out 5-10 years later and never wanting to see a stethoscope ever again. However, physician burnout is an undeniable problem. Emergency medicine is notorious for burnout, even before you finish residency. While it's healthy to be optimistic and positive about our futures, we also need to be aware of this reality. We can't expect to want to switch days and nights every few shifts and be on our feet for 12 hours for the rest of our careers. We also cannot predict what will happen to our bodies, emotions, and life situations later in our careers. That's why you need a backup plan to protect that huge investment in time, money, and youth you made by going into medicine.

An MBA will NOT...

Make you a financial guru, venture capitalist, stockbroker, etc.

For some reason, the minute you graduate from medical school, everyone wants your medical advice. As if, suddenly, having a diploma in your possession is what gives you all that knowledge you've spent years acquiring. The same thing happens when people learn you have an MBA. I honestly still have just rudimentary knowledge of how stocks work and have a negative net worth to invest into startups. If that's your thing and you put time and interest into those skills, by all means, go for it. However, don't expect an MBA to make you into a "finance" person. Sure, basic finance is part of the training, but people go to years of business school specially dedicated to finance or investing or have decades of experience before they can even be a viable player in the financial world.

Give you a parking spot and C-suite... yet

You don't earn the 2 letters after your name without blood, sweat, tears, or other bodily fluids which may or may not belong to you. Similarly, no one will make you a CMO/CEO/CIO/C-whatever-O the second you get your MBA. You still have to work hard to advance that part of your career. You have to climb the ladder, establish yourself, and prove your skills just like you do in medicine. The same way you cannot walk into a hospital with your med school diploma and expect to be an attending, you can't get a C-suite office as an MBA graduation gift either. It is, however, a nice conundrum to have when you have to figure out for the first time how to arrange MD, MBA with all the period and comma glory at the end of your signature.

Eliminate the need for doing a residency

The most uniform, consistent piece of advice most physician leaders, MD/MBA or otherwise, have given me is: "You have to be a good clinician first." This is the difference between being called "doctor" vs. being called "Mr./Mrs.," or worse, a "suit." Inherent to being a physician leader is being a physician. Inherent to being a leader is that you, usually, have to be good at what you do, or at least be respected for what you do. You have to do a residency and spend that time being the best doctor you can be. Unless you're over the whole doctor thing and want to be a "consultant" forever, you cannot forego residency training. Even IF you want to be a consultant, and you are not residency trained, you might regret it when no one takes your medical training seriously. Residency does not only train you in your specialty, it trains you to understand both the healthcare system at large and microsystems unique to the institution. It helps you understand what the flow is really like, and what problems clinicians actually (vs. theoretically) face. It also gives you a huge set of colleagues and friends who can contribute to your knowledge, career, and influence. Moreover, finishing residency isn't just a checkbox to complete so you can move on to bigger and better things. Residency is where you hone your doctoring, leadership, and management skills. You have to be a good doctor to earn respect as a physician and as a leader.

Teach you all there is to know about business

Did medical school teach you all there is to medicine? Probably not. The same applies for business school. You still have to research and read up on topics when you lack full understanding, and you have to go through some on the job training as well. There will be MANY such topics because both fields are very broad and are impossible to condense into a finite amount of education.

Guarantee you a high-paying job after school or residency

This one goes with the parking spot statement, with a little qualification. It can be tempting to look at the salaries of the CMOs and other MD/MBA big shots. Not only are the salaries enough to make you want to Scrooge McDuck into a fantasy pool of money, but the benefit packages induce immediate salivation. But in reality, everything is a bell curve; we just don't often hear about those on the lower end of the curve. While on average, an MD/MBA degree can lead to a higher salary, that higher salary is not because you happen to be the doctor with an extra degree and more student loan debt, but because the degree, along with a lot of hard work and experience, allowed you to attain a position that pays a higher salary. Therefore, some of the statistics might make you think that you will earn more than average just because you have more education. In reality, you still have to get a job, perhaps one that uses your MBA, in order to get that higher salary.

Factors to consider before making a decision

Burden

Getting a second degree requires sacrifice in terms of money, time, energy, family, etc. Whether you are getting it during, before, or after medical school, these demands are often concurrent and, therefore, double. During my MBA training, I had online classes every Thursday evening, meaning my entire family had to coordinate and take turns entertaining and keeping my very loud toddler — who had the sole life mission of breaking into the bedroom and ripping out my headphones - quiet. Their Thursday (and many other) evenings were captive to my MBA work, online team meetings, and powerpoint recordings in addition to the rigors and demands of having a wife, mother, and daughter in medical school. While I could sometimes work out scheduling issues between med school and my MBA program, I had multiple really tough moments that required an energy level and insomnia that bordered on mania. I still

get tachycardic and sweaty remembering how I had to do a week-long, on-campus boot camp for the MBA while studying for Step 2 (which was 1.5 weeks away), with my usual childcare provider being out with a broken hip. Additionally, it is difficult to make assumptions about how flexible individual residency directors can be with even online classes, let alone the in-person activities some MBA programs demand. You also have to consider whether you can truly sacrifice all of your post-shift or personal time and intellectual real estate to crack open yet more textbooks and participate in class. In residency, some portion of that time should be spent studying medicine and preparing for your boards, which can be difficult if you have a whole other degree for which to study. In terms of the financial sacrifice, as a person who avoids looking at their loan statements to ward off nervous breakdowns, I can tell you it's not small. My medical school offered an alumni discount (very tiny but not negligible) as well as forgiveness of 25% of the required MBA credits because of the dual program, which helped a little. However, my debt is still the size of a small private island that keeps magically growing while I sleep. Some universities offer discounts for staff and faculty as well. You have to do the math to figure out what is more fiscally realistic for you and what kind of discounts and offers you can take advantage of. This might make a difference in your loan repayment or deferment, as well as the timing for when you would enroll in classes to maximize your tuition savings.

Timing

I knew that after medical school, I would probably never want to go to any kind of school ever again. That's why I decided to suffer once and get the degrees done together. As a resident, I cannot imagine factoring in an MBA degree right now, but it may not be impossible, depending on your program and schedule. Waiting to be done with residency to do the MBA is another option, one that depends on your post-residency plans and life stage. For some, this may be the optimal timing, since your schedule may finally have some flexibility. As mentioned previously, be aware that timing may also impact what kind of financial incentives you have for doing the MBA. For instance, some jobs may offer to pay for a portion of your classwork, while your alma mater may provide tuition discounts. You may potentially get better loans and scholarships before you start making that sweet attending money. Depending on the program, there may also be limits on how much time you have to complete the degree. If you pursue both concurrently, you may have additional time to space out your learning, given that you might have conflicting schedules and demands from both degrees. If your program sets a cap of 18 months for example, and you still have to pass your Boards and move across the country, and you're also about to become a parent, those 18 months creep up very fast.

Post-training career aspirations

If you never want to do anything outside of the department, especially anything that requires attire other than scrubs, why put yourself through an MBA? An MBA, or any degree other than a medical one, makes sense only if you are interested in that field of study and/or that field of study will advance your career. If you're getting a business degree just for the knowledge, there's plenty of sources that are several tens of thousands of dollars and years of your life cheaper. Conversely, if you're getting an MBA just for bragging rights, there are better ways to spend 1/3 of your mortgage and 1/2 of your free time. Additionally, with the rise of the Administrative Fellowships, there is a built-in degree that you complete while you do the fellowship. Whether that degree is an MBA or MHA or MPH varies by institution. However, if you are planning on looking for a fellowship opportunity to pursue business, policy, or administrative training, this is a viable option to consider.

Personality/interest

For some people, the thought of meetings, Excel spreadsheets, and talking to a bunch of suits is one of Dante's rings of hell. For others, it's a necessary evil to exact change and make important decisions. It all boils down to what is interesting, natural, and stimulating for you. We have all seen that person in med school who dropped out in the first few years, or even the one who trudged through the preclinical stuff until they got to OB and bailed. They were miserable from the onset, and everyone saw it coming from a mile away because they were neither interested nor built for medicine. The same is true for business administration. Either you're the type who digs this stuff and has the right disposition for it, or you're not. It may be hard to tell if you've only thought about it theoretically and have no real-world experience. My suggestion is to do what wannabe doctors do to figure it out — shadow in some capacity before you take the plunge. I got my taste for business administration while inadvertently falling into that role in my job before med school, which made me realize that I was both cut out for and into this whole business thing. Some residencies and even med schools offer rotations or electives for you to get a taste of the admin world for yourself. Alternately, you can seek out a practicing MD/MBA or administrator who happens to also be a physician and ask them about their experience or see if you can tag along and observe what they actually do.

Other types of degrees

I don't have much experience with MHAs, PhDs, or MPHs to be able to offer any sensible advice on why you should or shouldn't pursue those instead of an MBA. Undoubtedly, there are specific differences in what the various degrees focus on from a learning perspective and what kind of opportunities they present later in your career. An MBA is usually the most general "administrative" degree and can be applicable outside of health care. With that, however, you sacrifice a health-care-focused curriculum during your studies. If you think another type of degree is better suited to your needs and aspirations, do some research to see what the other degree can offer you that the MBA cannot (and vice versa). It always helps to talk to someone who has pursued that degree and get a sense of their experience. Getting real insight from someone who has both talked the talk and walked the walk is crucial.

Hopefully, this information will help you decide whether a dual MD/MBA degree is right for you. You may wonder why I

would (as the business-savvy doctor that I am) encourage more doctors to become my competitors in the power struggle to be the biggest fish in the health care admin pond. The reality is that there are many, MANY ponds and they are struggling. To overuse the common parlance, the health care landscape is changing, and unfortunately not for the better in most cases. Our systems, our specialties, our patients, and our colleagues need us. They need us to innovate, cut costs, protect our coffers while saving lives, provide a non-whitecoat perspective, and be the bosses that we know we are. And, if you're the right kind of fish for this kind of work, there's plenty of pond to share. ✶

Suggested Reading

1. This article in the Harvard Business Review from Lisa S. Rotenstein, MD (a fellow MD/MBA and resident), Raffaella Sadun, and Anupam B. Jena about why we need better leadership training during our pre- and postgraduate medical education: "Why Doctors Need Leadership Training."
2. A really comprehensive piece by Vidya Viswanathan in The Atlantic that holds true to a lot of my personal experience and that of my MD/MBA colleagues: "The Rise of the M.D./M.B.A. Degree."
3. The Association of MD/MBA programs: www.mdmbaprograms.org. This website has a fairly up-to-date list of current MD/MBA programs. Most of the programs listed have a link where you have to dig a little deeper for program specifics but this is a good place to start.

Title X Changes Egregiously Harm Women's Access to Comprehensive Medical Care

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On Feb. 22, 2019, the Trump administration announced sweeping changes to Title X,[1] the federal program that provides funding for reproductive health services, including STI treatment, cancer screening, and contraception to millions of women. These alterations ultimately serve as a gag rule for physicians, as under the Trump administration's changes, any physician or organization that provides or refers patients to abortion services are no longer eligible for Title X funding.[2] Put plainly, any referral or performance of abortion services would limit funding that is used for preventative cancer screenings, contraception, and treatment of STIs. The main beneficiaries of Title X funding are predominantly low-income and minority patients; therefore, the cessation of these services threatens to further widen health inequalities. These political decisions will affect not only women's ability to access health care, but also physicians' ability to offer comprehensive services.

What is Title X?

Title X was enacted in 1970 with unanimous support in the Senate and an overwhelming majority in the House. Title X was considered powerful bipartisan legislation meant to support culturally sensitive, high-quality family planning services for low-income uninsured or underinsured populations.[3] Throughout the 1980s, the Title X program survived attempts to dismantle it, though the total funding for the program was reduced.[4,5] Since its inception, Title X has served as a critical facet of the American health care system. In 2017, Title X clinicians served more than 4 million patients and provided 2.8 million patients with contraceptive services. The majority of these beneficiaries reported family incomes at or below the federal poverty level. Half of these patients were uninsured, and two-thirds were men or women of color.

To be clear, Title X has never been used to finance abortions. The statute governing Title X outlines that funds may only be used where abortion is not a method of family planning. However, until this point, health care entities have maintained the ability to utilize Title X funding for contraception as well as STI screening and treatment, as well as other preventative services within clinics that also provide abortions, so long as abortions were financed separately from Title X funds. Therefore, the money that funds Title X has been used exclusively for other sexual and reproductive health care needs. However, the new regulations prohibit any Title X funds to support any health care site that also provides abortions, regardless of the fact that Title X funding would not – and has never – been used to finance abortions. Furthermore, the new regulations would threaten to remove Title X funding from any organization or provider that promotes or refers patients to abortion services.

How will Title X changes affect patients and providers?

Regardless of your political or social views on reproductive health care options, any policy that threatens our ability as doctors to provide and counsel our patients with comprehensive health care options should be opposed by all physicians. As physicians, we are bound by our profession to care for our patients

and to provide the most comprehensive health care options available. The limitation of discussing or referring patients for abortion services acts as a slippery slope. Taken further, it is not difficult to imagine these limitations as the precedent for additional governmental gag rules, which could later be expanded to other issues or medical services.

The Title X changes hold numerous possible downstream consequences. As discussed by the Kaiser Family Foundation,[2] potential side effects include limiting the number of family planning providers, limiting low-income women's access to contraception, compromising the quality of family planning care available, placing individual providers at increased medical liability, adding administrative burdens to clinics receiving Title X funding, and inadvertently promoting organizations that offer only "natural" family planning and abstinence. As Title X funding most directly benefits low-income women, the new rules would only serve to further health disparities that exist based on race, income, and geography.

The effects of these changes are not limited to the outpatient setting. If hospitals and health care systems receive Title X funding, these changes could severely alter the funding that individual hospitals receive. The reduction of Title X funding to outpatient clinics will undoubtedly decrease the number of available family planning practitioners. This, compounded by the limitation of referring patients to abortion providers, will severely hinder our referral practices in the emergency department, creating barriers to access for our most vulnerable patients. Additionally, the administrative burdens for tracking the complexities of Title X funding under new restraints may cause organizations to make blanket changes to their policies to prevent potential violations, even for allowable services and referrals.

There are also immediate effects that may affect both patients and providers in the emergency department. The potential decrease in family planning clinics will reduce the number of STI screening and prevention, potentially increasing the prevalence of STIs within communities and emergency department visits for STI-related complaints. This could lead to potential outbreaks in STIs, as was seen in Indiana when access to comprehensive family planning centers was obstructed and the state underwent one of the largest HIV outbreaks in the 21st century.[6] Perhaps if comprehensive health centers remained unencumbered, they may have performed HIV testing that could have detected the outbreak sooner and reduced its severity.

With a limited number of outpatient abortion providers, women may turn to unsafe termination practices. This would undoubtedly increase the number of presentations to the ED for complications associated with these dangerous procedures. Lack of access to safe abortions, predominantly in low-income minority women, led to more than 15,000 abortion-related deaths in the 1920s. The year after abortion was legalized in New York state, the maternal mortality rate dropped by 40%.[7] To say that limiting the ability of physicians to perform or refer patients for abortion will limit the number of abortions is unfounded. What will be reduced is the number of safe abortions, but with this the number of unsafe abortions will rise. Patients who lack access to safe and affordable care may resort to extreme means, such as at home terminations, mail-order medically induced abortions, and unsterile practices, thereby increasing the rates of morbidity and mortality.

Summary

As Dr. Leana Wen, emergency physician and former president of Planned Parenthood, said:

> *As a doctor, this compromises the oath that I took to serve my patients and help them with making the best decision for their own health.... My patients expect me to speak honestly with them, to answer their questions, to help them in their time of need. It's unconscionable and unethical for politicians to restrict doctors like me from speaking honestly to our patients.*[1]

Days after this statement, Planned Parenthood, one of the nation's largest recipients of Title X funding, stopped accepting any Title X funding, as they refused to allow their ability to care for women and the public health to be in any way infringed upon by policy debates.

EMRA's official policy statement on Title X emphasizes the importance of protecting women's health care, as well as patients' right to information and access:

These changes aim to block federal funds from family planning providers that provide abortion services, counseling, or referrals - effectively decreasing the network of clinics and scope of family planning services offered to women, especially low-income and uninsured patients. EMRA believes in protecting access to women's health care. Our patients, especially vulnerable populations who depend on Title X funding, deserve full information and referrals. EMRA opposes the proposed Title X rule on the basis that they will detrimentally decrease patient access to women's health care including reproductive care.[8]

The changes to Title X threaten to reverse 40 years of advancement in women's health care. It places us in a world where patients not only lack access to STI testing, sexual education, contraception, and cancer screening, but also limits physicians from fully discussing known medical treatments with our patients. If we allow the Title X changes to limit what we as physicians can tell our patients, where does it end? At what point will we tolerate governmental restrictions in our discussions with patients? Join EMRA, the AMA, and other organizations in opposing the Trump administration's efforts to reduce access to comprehensive family planning solutions and women's health. Don't let politics get in the way of providing patients with evidence-based medical care. ✶

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References available online

The Heart of EM

Protecting Your Greatest Investment

James M. Dahle, MD, FACEP
Founder of The White Coat Investor

I was recently approached by a physician who wished to thank me for telling him to buy disability insurance. Since I tell every non-financially independent doctor to buy a policy, I did not think the advice was particularly remarkable. His story and the timing of his purchase, however, were remarkable.

We share his story (anonymously) as an example of the value of preparedness. This could have happened to any one of us.

In His Own Words

On April 21, 2019, I was caught up in the excitement of an upcoming medical mission trip to help the people of Madagascar. I had spent the past 6 months working tirelessly planning, fundraising, and acquiring supplies in preparation for this special trip. It was now only weeks away, and other than a few minor details, we were ready.

At this time, I was approaching the end of my second year as an emergency medicine resident. My confidence was building, and I had a number of exciting things going on professionally and personally. Life was really good.

I was in the middle of a toxicology rotation for which I commuted by train each day. On April 22, as I was getting off the train, I noticed my left leg felt "asleep." I just shook it off and went on with my day. However, the feeling never went away. The next day when I got on the train the strange sensation persisted in my left leg, and now I noticed a similar feeling on my left flank. Later that day, I felt it on the left side of my face. At this point I wasn't sure if I was crazy or if something was actually going on. I started calling all my doctor friends and colleagues. We went through the differential ad nauseum, but given the lack of any objective abnormalities I decided to just watch and wait.

On April 24, I was awake at 5 am and ready to catch my train, but now the entire left side of my body from head to toe had a tingling sensation. At this point I was pretty sure I wasn't just crazy, so I went to the emergency department where I worked. I was greeted by nurses, staff, techs, co-residents, and attendings whom I knew well from my previous 2 ½ years of training. I received exceptional care from concerned friends and colleagues. I had developed very mild weakness in my left leg, and I got a full stroke work-up. By the end of the day I had an MRI, which showed a small lesion on my right pons. To complete my work-up and manage unforeseen complications, I spent 5 days in the hospital.

I'm an avid rock climber, biker, outdoors enthusiast, and I've never had anything close to a major medical problem. I'm young and healthy, careful about my eating and exercise habits. ***I've tended to countless patients as a resident and medical student but never thought I would be the one on the stretcher. I'm so grateful for what I learned from the time I spent as a patient.*** *However, this article isn't about my medical journey, but recognizing our mortality and preparing for our future.*

Serendipitous Knowledge

Just 2 weeks prior to this event, I had begun reading Dr. Dahle's new book,

The White Coat Investor's Financial Boot Camp. *During my 1-hour commute each way by train I would study and take notes on this book. The first chapter is about long-term disability insurance, and the second chapter discusses life insurance. Dr. Dahle emphasizes how important this insurance is for protecting ourselves and our loved ones from catastrophe. I already had a sufficient life insurance policy, but long-term disability insurance was much more complicated and expensive.*

After reading this book, I knew I needed the financial protection of disability insurance. Like many others, I did have a small amount of long-term disability insurance through my hospital as a resident, but the more I read the more I realized this coverage was desperately insufficient. I organized my notes and called my insurance agent. I was able to approach this phone call with educated questions and the basic idea of what I needed to protect myself as a physician, father, and husband. Thanks to what I had learned from that first chapter I felt confident the new policy I purchased would provide appropriate coverage at a fair cost. In just a few short days I had confirmation that my policy was active. I paid my first premium for this policy on April 24, which happened to be the day I began having symptoms.

Unfortunately, once you need to make use of disability insurance it's too late to purchase it. *If I tried to apply for coverage today, I would likely be denied or wouldn't be able to get the coverage I needed for a premium I could afford. I recognize I was extremely blessed to have purchased a policy when I did. Life is full of the unexpected, both good and bad. We won't know the future, but we can take steps to prepare. Each of us has family, friends, and/or have personally experienced tragedy that has impacted their ability to financially succeed and provide.*

I'm extremely humbled to have the opportunity to team up with Dr. Jim Dahle, founder of The White Coat Investor*, to share my story and provide you with the tools you need to protect yourself, your loved ones, and your future. We have invested so much and sacrificed even more in our calling to provide care for those in their worst possible moments. However, we rarely stop to think about who will care for us or our loved ones if tragedy strikes. The truth is that no one will do it for you, and it's your responsibility to provide this necessary protection.*

How to Protect Your Greatest Investment

If you have enough money to live and provide for your dependents for the rest of your life, you likely have no need for disability or life insurance. However, that's not the case for most people who work for a living. Young physicians need to buy disability insurance, and if anyone else also depends on their income, term life insurance. Physicians, including resident physicians, become disabled all the time. Sometimes the disability is only partial or temporary, but other instances may be complete and permanent. Either way, protecting your decade-long investment in medical knowledge and skill is one of the most important pieces of your financial plan. Think about how much money is at risk. **If the average emergency physician makes $375,000 per year for 30 years, a disability could cost you more than $11 million!**

Understand Your Options

Compared to life insurance, disability insurance is complicated; life and death is black and white. Sure, as emergency physicians we know there is a little gray there, but it generally only lasts a few minutes. That is not the case for disability. Thus, you want a disability insurance contract that has the strongest possible definition of disability, with the least amount of wiggle room for the insurance company. For a doctor, that means a true own-occupation, specialty-specific policy from a company such as Mass Mutual, The Standard, Ameritas, Guardian, or Principal.

Besides a strong definition of disability, you also want to make sure the policy has a partial/residual disability rider that will cover partial disabilities and continue to pay benefits as you recover.

Young physicians, particularly residents, should also consider buying a Future Purchase Option Rider (so you can buy a larger benefit later even if you develop a pontine lesion or take up rock climbing) and a Cost of Living rider, so your benefits will go up with inflation over the years in the case of a long-term disability.

Disability insurance, because it is frequently used, is not cheap. Expect to pay between 2% and 6% of the monthly benefit amount in premiums (ie, $100-300/month for a $5,000/month benefit). **However, it will never be cheaper for you than right now when you are young and healthy.** While it is true that you are less likely to become disabled now, when you are young and poor a disability is much more of a financial catastrophe than it will be later. The consequences (and thus the benefits of a lifelong stream of income despite being disabled) are simply larger at this stage of life. It is best to purchase this policy from an independent agent who sells hundreds of them each year. They can help you understand what you are buying and provide the best possible recommendation for your age, gender, state, specialty, medical condition, and hobbies.

A Note About Life Insurance

Compared to disability insurance, buying life insurance is easy. Most young doctors with dependents should buy $2-5 million in 20-30 year level premium term life insurance from a reasonably reputable company. You can usually use the same agent you bought disability insurance from. If they recommend whole life insurance to you as a resident or young physician, get a new agent. The good news is term life insurance is much cheaper than disability insurance. A 30-year-old healthy female can buy $1 million of coverage for just $520/year. ✶

TAKE-HOME POINTS

- Disability insurance will never be cheaper for you than it is right now.
- By the time you need this type of insurance, it's too late to purchase it.
- Doctors wouldn't think of going without malpractice coverage, but far too many fail to insure themselves against an even more expensive risk. Do yourself and your loved ones a favor and get your disability and life insurance in place STAT!

Premeditated Compassion

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My second shift as an intern was a long haul. Swamped with tasks, the learning curve I was on felt more like a wall. So it was with relief that I noticed I was 20 minutes away from the time at which I wouldn't pick up any new patients. Two people were still in my care and I would likely not be signing out anyone to the next shift.

Then, looking at the board once more, I saw the little red box pop up: a new patient. The conflict ate at me. I wanted to cross the finish line that was just 20 minutes away. I also wanted to be the intern who never let a patient sit on the board. I saw my co-residents fighting the same battle as they hovered their mouses over the "Assign Me" button.

My patients happened to be more tidied up at that point, so I took the plunge, picked up the patient, and went to meet the EMS squad. They waited in a hallway tucked between the main patient care areas. This hallway was where cramped rooms begged for simple encounters and quick dispositions. The stretcher clogged the space, making me squeeze between it and the linen basket to get to the foot of the bed. That squeeze reminded me of what I had been feeling the entire shift: simple things being complicated by unrelated demands. Finally, I found my mark and I asked the paramedic for the story.

"Doc, we got a call because he all of a sudden felt weak in both of his legs and couldn't move them."

As the monologue progressed the patient was whirled into the room and hooked up to the monitor. I heard the nurse say, "When did you have that aortic sleeve placed?" That question pulled me into the room. Entering it felt like the start of a simulation case. The diaphoretic man before me was groaning in pain, yelling for us to do something. The vitals on the monitor were ominous.

Fast-twitch decisions started to happen. I had rehearsed these. EKG ordered. Access obtained. My fourth-year resident brought the ultrasound to the bedside.

"Doc, just put me down already. I can't take this pain!"

The melodrama of it shook me. I remember freezing for a moment and thinking "Is this a simulation?" As in any simulation, we continued to act quickly. Attending to the bedside. No pulses on Doppler. CT angiography of the chest, abdomen, and pelvis with runoff. STAT read requested. Vascular Surgery paged.

***CT Read**: "Complete occlusion of the aortobifemoral bypass graft due to occlusion at its proximal end with poor renal enhancement and no distal flow"*

The patient was handed off to the resident covering the Shock Resuscitation Unit and I finished my night.

Reflection and Review

Running back the "tape" of my first critically-ill patient felt like those painful moments when I watched the recordings of standardized patient encounters as a medical student. I saw myself standing in the radiology room as the patient was transferred to the scanner. I was saying, "We need CTA with runoff to the toes. He has an aortic graft and it looks like there are no pulses in his feet." The patient's hands were secured above his head. He lay there naked, while I talked loudly with the radiology technician about the protocol. The tech now knew more about my patient's condition than the patient. My sense of urgency had led to disregard; I had failed to inform my patient what was going on.

In replaying this scenario, I realized that I almost never spoke to the patient, explained what was happening, or addressed what we were doing. He was a mannequin sitting on a stretcher with mechanical pupils and there was an attending behind the glass screen telling me how bad his pain was. The lack of consideration I showed my patient was counterbalanced only by my ability to manage the encounter clinically.

Simulation Plus

After this case, I would never question the value of training with simulation. This case forced my mind to move down clinical pathways that have been worn deeply into my brain via simulated cases. What became clear is that committing to a clinical pathway is not sufficient to treat our future patients; we also must engage with them compassionately. In this case, it is clear, my medical decision-making outpaced my ability to care for this patient with compassion.

This subordination of compassion to medical decision-making does not surprise me. Saving a life is more important than tripping over words. Yet, I can't help but wonder: if my care saves the patient but leaves their mind wounded, have I fully cared for them? The tension between compassion and critical care arises because the former requires complex cognition that is nearly impossible to muster when every neuron is being utilized to manage a situation clinically. This is why our training for compassionate care should not be reserved for controlled conversations about how to deliver cancer diagnoses; it must include acute care situations. Our patients need this, as the compassion we show in critical moments is directly related to the number of patients leaving our care with PTSD.[1]

If I could go back to that evening, the compassionate care that I wish I had offered would be premeditated; not robotic, contrived, or fabricated compassion, but deliberately practiced. Maybe then I would have used the 30 second trip to the CT scanner to tell my patient about what was happening to him.

Take-Away

We all desire to show our patients compassion. Adopting a practice of premeditated compassion would allow us to do so reflexively even as we develop the tunnel vision common to acute care situations. Working through this deliberate practice shows the value we give to others as we sacrifice time to prepare for their worst days. ✶

#MedMissionHacks

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The global hyper-awareness from exposure to 24/7 news and social media has left many people wanting to get more involved in medical missions. How can you make the most of a medical mission?

Kent Hospital EM residents Landon Wood, DO, and Timothy Bikman, DO, have devoted much of their free time during the past 6 years to organizing an EM-focused mission project in Madagascar, an island off the southeastern coast of Africa. They are sharing their top 10 #medmissionhacks based on their Madagascar experience.

Ultrasound: Ultrasound has become an essential tool in the ED. In an environment where labs and imaging are limited, its value increases exponentially. But access to this technology is extremely limited. **Sonosite's Global Health Loaner Pool** program can help secure a portable ultrasound machine that can be used for free during your medical mission work. Details: www.sonosite.com/about/global-health-loaner-pool. The disrupter **Butterfly IQ** is a single probe that connects directly to portable digital devices and costs only $2,000. Details: www.butterflynetwork.com.

Education: Too often as medical mission team members return home from 1-2 weeks of service, they are left asking themselves, "What was the real impact we made?" Education is the most powerful tool to effect long-term change. This should be a primary focus for any medical mission project. Host workshops on dental hygiene, clean water preparation, hand hygiene, nutrition, medical procedures, and more. We offered POCUS training, for example.

Doximity Foundation: If you are a physician in training with a Doximity account and are planning to do international medical mission work, you might qualify for a free flight. The Doximity Foundation is a nonprofit arm of Doximity that provides travel grants for medical mission work across the world. Details: https://foundation.doximity.com.

Prevention and general health tips: Review CDC and WHO guidelines for the country at least 3 months before leaving. You will find very valuable information on travel alerts, necessary vaccines, health care-related outbreaks/crisis, active WHO programs in that area, appropriate malaria prophylaxis, and more. Details: www.who.int and wwwnc.cdc.gov/travel/destinations/list.

Domestic support: Your local hospital and EMS stations can be powerful allies in helping organize supplies and fundraising. Reach out to your hospital system and your community. It's amazing how this type of work has the ability to ignite a special flame within your community.

Social network and fundraising: Social media can raise awareness and funds for your medical missions. Don't be afraid to launch a fundraising campaign. Just remember this is charitable money and should be used responsibly, for the direct benefit of the people you plan to serve. Document on social media the impact that money makes. Be creative in your fundraising efforts. For example, we created a kickback incentive program: individuals who donated at least $25 received fresh Madagascar vanilla bean and those who donated at least $50 received fresh vanilla bean and chocolate.

Supplies: Around the world, countless people suffer or die because of treatable illnesses. Domestically, our health care system produces countless tons of medical waste yearly. Programs like **Mop.americares.org** and **Medshare.org** help divert

these potentially wasted medications and medical supplies from landfills into the hands of medical mission teams at a greatly reduced or no cost. Details: https://mop.americares.org/MOPHome/index.jsp and www.medshare.org.

Volunteers: When it comes to the human resources needed for a successful medical mission, doctors make up only a small proportion. Invite a full team: advanced practice practitioners, nurses, EMTs, paramedics, pharmacists, medical students, and non-medically trained individuals. Each person adds valuable skills in planning and carrying out the mission.

Have fun: This work takes a significant amount of time, energy, and money. It can be physically and psychologically demanding. Take time to embrace the people, culture, food, and geography. Let this work transform you and find joy in the journey. ✶

Spring 2020 Award Recipients

EMRA is proud to offer a robust array of awards, scholarships, and grants every year - recognizing outstanding work and helping to foster excellence among clinicians, educators, and leaders in EM. Please join us in congratulating the EMRA 2020 Spring Awards recipients.

Academic Excellence Award
CAPT Rachel Bridwell, MD, Brooke Army Medical Center Emergency Medicine, USUHS

Dr. Alexandra Greene Medical Student of the Year Award
Jeremy Towns, University of South Alabama College of Medicine

Chief Resident(s) of the Year: Indiana University School of Medicine Chief Residents
Kimi Chernoby, MD, JD
Carter Duggan, MD
Andrea Purpura, MD
Devin Doos, MD
William Martin, MD
Emily Wagner, MD

Fellow of the Year
CPT Justin Grisham, DO, Madigan Army Medical Center

Resident of the Year
Ynhi Thomas, MD, MPH, MSc, Baylor College of Medicine

Jean Hollister Contribution to Pre-Hospital Care Award
Faroukh Mehkri, DO, University of Connecticut Hartford Hospital

Rosh Review "One Step Further" Award
Corlin Jewell, MD, University of Wisconsin

ACEP CORD Teaching Fellowship
Kathryn Fisher, MD, Baylor College of Medicine

FACULTY AWARDS

Residency Director of the Year
Scott Johnson, MD, FACEP, Stony Brook University

Associate Residency Director of the Year
Kristen Kann, MD, Brook Army Medical Center

Residency Coordinator of the Year
Kim Watkins, North Florida Regional Medical Center

TRAVEL SCHOLARSHIPS

ACEP *Scientific Assembly*
Kellan Etter, Des Moines University

Critical Care Medicine Conference
Shyam Murali, MD, Mercy St. Vincent Medical Center

EDDA
Barron Frazier, MD, Vanderbilt University Medical Center
Daniel Novak, DO, Maimonides Medical Center
Nicholas Stark, MD, UCSF
Vir Singh, MD, MBS, University of Central Florida
Phillip Tseng, MD, MBA, MEd, Ohio State University Medical Center

EMBRS — EM Basic Research Skills Conference
John Campo, MD, Harbor–UCLA Medical Center

EMRA Congressional Health Policy Fellowship
Owais Durrani, DO, UT Health San Antonio

FemInEM FIX19
Jessica Barlow, MD, Brooke Army Medical Center

SAEM
Brittney Mull, MD, Harbor–UCLA Medical Center

Join Our New Cast of EMRA*Cast

Have you heard an episode of EMRA*Cast and thought, "I'd like to do that!"? Are you interested in interviewing leaders in emergency medicine? If so, we want to hear from you! We are looking for 2 residents to further cultivate the voice of our specialty in a podcast "By residents, for residents." This opportunity is a 2-year time commitment and open to any active EMRA Resident member.

No podcast experience? No problem. That's what EMRA*Cast is envisioned as — a chance for anyone who wants to give it a shot. We have mentors ready to work with you if you are selected. Applications are open until April 30. ✶

ABEM Resident Ambassador

EMRA is seeking applicants for a new position: the American Board of Emergency Medicine Resident Ambassador Panel. EMRA will forward 2 nominees to ABEM, which will appoint 3 residents from among those nominated to serve as ambassadors. Terms are 2 years (July 1, 2020–June 30, 2022) and must be encompassed while in residency. The member must be at the PGY-2 training level or higher at the start of his/her term. Members of the Resident Ambassador Panel will participate in ABEM projects such as resident-related content on ABEM's website or presentations. **Apply by April 10 at 5 pm Central**. Details can be found by searching "ABEM" on emra.org. ✶

ABEM Elects First-Ever Public Member Director

The Board of Directors of the American Board of Emergency (ABEM) recently elected 3 new members: James D. Barry, MD, and Suzanne R. White, MD, as physician directors, and Hala Durrah, MTA, as its first public member director.

Dr. Barry is Clinical Professor, Department of Emergency Medicine, University of California, Irvine, and practices clinically at the Long Beach VA Medical Center.

Dr. White is Chief Medical Advisor for the City of Detroit, and Regional Chief Medical Officer, Bayfront Health Community Health System, Florida. She practices clinically at the John D. Dingell VAMC Emergency Department, Detroit.

Ms. Durrah was elected as the Board's first public member director. She is a national speaker, patient/family-centered care consultant and advocate. She serves on a number of national committees, including the American Academy of Pediatrics Family Partnerships Network Executive Committee, the CMS Executive Leadership Council of the Strategic Innovation Engine, the Patient-Centered Primary Care Collaborative Care Delivery and Integration Cabinet. ✶

Plan Ahead for Residency Fair and Job Fair

EMRA's vaunted Residency Program Fair and Job & Fellowship Fair are on the calendar! Plan to attend these events, both held in conjunction with **ACEP20** in Dallas.

EMRA Residency Program Fair

1:30–3:30 pm Sunday, Oct. 25

Kay Bailey Hutchison Convention Center, Hall F

Early-bird registration deadline: Aug. 1

More than 1,000 U.S. and international students flock to the EMRA Residency Program Fair each year, eager to explore their options for emergency medicine training. Make sure your program is represented! *Thanks to our sponsor, Laurel Road.*

EMRA Job & Fellowship Fair

5-7 pm Monday, Oct. 26

Kay Bailey Hutchison Convention Center, Hall F

Early-bird registration deadline: Aug. 1

EMRA's Job & & Fellowship Fair delivers one-of-a-kind networking opportunity with 2,000+ job seekers, including graduating residents and EM physicians looking for new opportunities. This is the largest recruiting event in the specialty, and we look forward to seeing you there. *Thanks to our sponsors, TeamHealth, emCareers.org, Vituity, and Laurel Road.* ✶

ECG Challenge

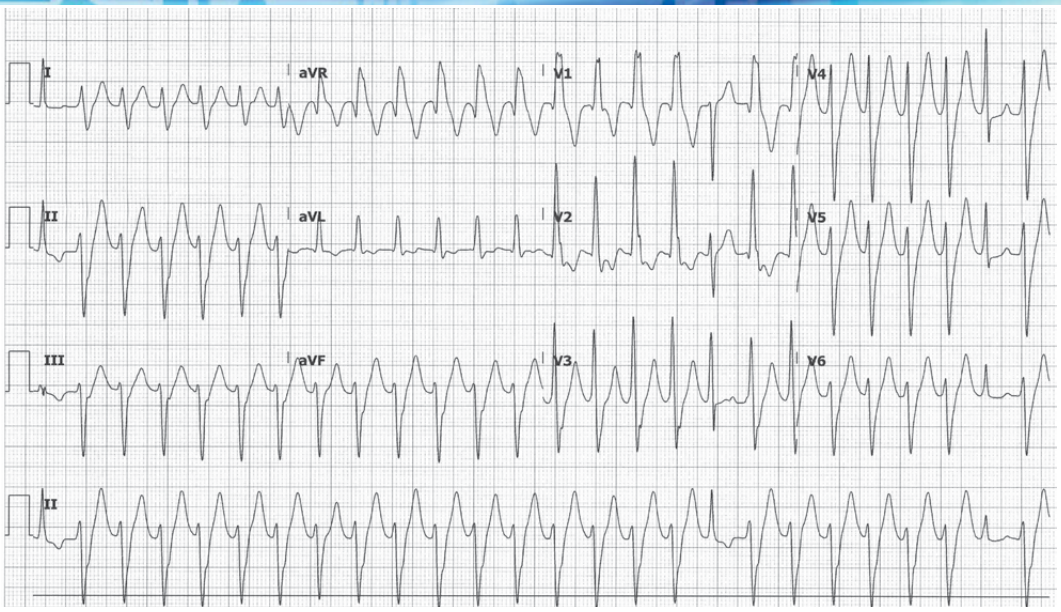
Danielle Miano, MD
ChristianaCare
@dimian0
Greg Steinberg, DO
ChristianaCare

Jeremy Berberian, MD
Associate Director of Resident Education
ChristianaCare
@jgberberian

CASE.
A 33-year-old male presents to the emergency department because of palpitations. He reports a history of similar episodes in the past that always respond to adenosine.

What is your interpretation of the ECG and what would you do next?

See the ANSWER on page 48



ECG Challenge

This ECG shows a regular wide complex tachycardia with a ventricular rate of 155 bpm, no visible P-waves, northwest/extreme axis deviation, prolonged QRS duration of 130 ms with a RBBB morphology, ST-segment and T-wave discordance, and 3 narrow complex beats. The differential for a regular WCT includes:

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

The key to interpreting this ECG is beats 1, 18, and 25. These narrow complex beats are suggestive of capture and fusion beats, which are considered pathognomonic for ventricular tachycardia.

This patient was known to have idiopathic fascicular ventricular tachycardia (IFVT), also called verapamil-sensitive VT. In this patient's case, the VT does break with adenosine, but this variant of VT does not reliably respond to adenosine.

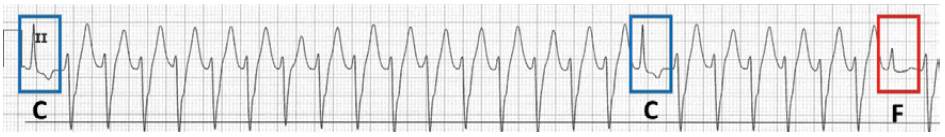
Basics of VT

Ventricular tachycardia is defined as a cardiac arrhythmia of ≥ 3 consecutive beats that originate in the ventricle with a wide QRS complex ≥ 120 ms at a rate >100. Sustained VT is defined as lasting ≥ 30 seconds or causing hemodynamic collapse regardless of duration. Non-sustained VT is defined as lasting < 30 seconds with no hemodynamic compromise. VT can be further categorized into monomorphic and polymorphic. Risk factors include structural heart disease and scar tissue from prior ischemia/infarct.

ECG findings that are considered pathognomonic for VT include AV dissociation, fusions beats, and capture beats. A fusion beat is a complex between a supraventricular impulse that originates above the AV node which fuses with an impulse generated in the ventricle (red box marked with F in Figure 1). Capture beats happen when a supraventricular impulse conduct normally through the AV node amid AV dissociation (blue boxes marked with C in Figure 1).

Management of VT

Always start with the ABCs, ensure you have adequate IV access, place the patient on a continuous ECG monitor, and place your cardiac pads in the anterior/posterior positioning.



Treatment options for stable patients include:

- Amiodarone 150 mg over 10 min followed by a drip at 1 mg/min for 6 hrs
- Lidocaine 1–1.5 mg/kg
- Procainamide 20–50 mg/min until VT terminates, or a max dose of 17 mg/kg

Treatment options for unstable patients include:

- Synchronized cardioversion at 100 J with increasing energy for each subsequent shock (note that this is also an option for stable patients)

Idiopathic Fascicular Ventricular Tachycardia

Idiopathic ventricular tachycardias represent ~10% of all ventricular tachycardias, arise spontaneously in the absence of structural heart disease, and are generally seen in young, healthy patients. ~10-15% of idiopathic VTs occur in the left ventricular conduction system, called idiopathic fascicular ventricular tachycardia, with the remainder occurring in the right ventricle.

Idiopathic fascicular ventricular tachycardia (IFVT), also known as verapamil-sensitive VT, idiopathic left ventricular tachycardia, and Belhassen VT, is a re-entry tachycardia from an ectopic focus in the left ventricular conduction system. The ECG will show a RBBB pattern with a relatively mild prolongation of the QRS interval (120-140 ms), as seen in this patient's ECG, when compared to other VTs.

IFVT can easily be confused with supraventricular tachycardias with aberrant conduction, however the presence of capture beats, fusion beats, or AV dissociations can help distinguish IFVT from SVT. Unlike SVT, adenosine and vagal maneuvers are not reliably effective in treating IFVT. Additionally, unlike traditional ventricular tachycardias, Class I antiarrhythmics such as lidocaine and procainamide are also not effective. As the name verapamil-sensitive VT suggests, this rhythm is very responsive to verapamil at a dose of 2.5–5 mg given IV every 15–30 minutes.

LEARNING POINTS

Monomorphic VT

- ≥ 3 consecutive, regular, wide complex beats with rate > 100
- Non-sustained: < 30 sec duration with no hemodynamic instability
- Sustained: ≥ 30 sec duration OR causes hemodynamic instability

ECG features that increase the likelihood of VT in a WCT

- QRS > 200 ms is almost always VT or hyperkalemia with aberrancy
- AV dissociation (ventricular rate > atrial rate)
- Positive or negative QRS concordance in leads V1-V6 (entirely or predominantly positive or negative QRS complexes from V1 to V6)
- Extreme axis deviation ("northwest axis")
- If BBB pattern is present, the absence of typical RBBB or LBBB pattern suggests VT (ie, normal RBBB or LBBB pattern makes SVT with aberrant conduction more likely)
- Fusion beats — hybrid QRS complex formed by fusion of a supraventricular impulse and ventricular impulse
- Capture beats — sinus QRS formed by transient normal conduction amid AV dissociation
- Brugada's sign — time from the onset of the QRS complex to the nadir of the S-wave is > 100 ms
- Josephson's sign — notching near the nadir of the S-wave

Case Conclusion

This patient was known to have IFVT diagnosed on prior EP studies. He was treated with adenosine, which terminated the VT. In this patient's case, the VT did break with adenosine, but it important to recognize that this variant of VT does not reliably respond to adenosine. ✶

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 4-year-old girl is brought in by her parents because she has foul-smelling vaginal discharge mixed with blood. She has no history of recent fever or UTI. Detailed questioning of the patient and her parents eliminates concern for sexual abuse. What is the expected finding on vaginal examination??
   A. Normal anatomy
   B. Retained foreign body
   C. Urethral prolapse
   D. Vaginal neoplasm

2. Which of the following is an indication to use a factor Xa inhibitor?
   A. Presence of a mechanical valve
   B. Prevention of atrial fibrillation
   C. Thrombolysis in ischemic stroke
   D. Treatment of DVT

3. What is the most common cause of death from complications of a Bordetella pertussis infection?
   A. Diaphragm rupture
   B. Pneumonia
   C. Pneumothorax
   D. Seizures

4. A 30-year-old woman presents by ambulance from an outpatient surgical center with CPR in progress. She received an accidental intravenous injection of bupivacaine and suffered a convulsion, followed by cardiac arrest. In addition to standard resuscitation measures, intravenous administration of which agent should be strongly considered?
   A. Fat emulsion
   B. Hydroxocobalamin
   C. Pyridoxine
   D. Sodium nitrite

5. Which statement about the management of traumatic pulmonary contusions is correct?
   A. CT findings reliably estimate the severity of pulmonary contusions
   B. Management of hemoptysis is rarely a concern in pulmonary contusions
   C. Respiratory support with intubation and mechanical ventilation improves outcomes
   D. Restricting intravenous fluids can help prevent the need for positive-pressure ventilation ✶

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

EMRA Emergency Medicine Residents' Association

The Emergency Medicine Residents' Association is the voice of emergency medicine physicians-in-training and the future of our specialty. With a membership of over 16,000 residents, medical students, and alumni, EMRA provides a like-minded community of your peers for a lifetime!

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The George Washington University Medical Faculty Associates, an independent non-profit academic clinical practice group affiliated with The George Washington University, is seeking full-time academic Emergency Medicine physicians. The Department of Emergency Medicine (http://smhs.gwu.edu/emed) provides staffing for the emergency units of George Washington University Hospital, United Medical Center, the Walter Reed National Military Medical Center, and the Washington DC Veterans Administration Medical Center. The Department's educational programs include a four-year residency program and ten fellowship programs.

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

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

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For further information, please contact Suzanne LeCroix at (256) 265-9639 or suzanne.lecroix@hhsys.org

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