

# EM Resident

Official Publication of the Emergency Medicine Residents' Association

April/May 2018

VOL 45 / ISSUE 2

## EMS Helicopter Safety



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# Letter from the Editor

**Tommy Eales, DO**

*Editor-in-Chief, EM Resident  
Indiana University  
@tommyeales*

**A**s we look forward to National EMS Week in May, it's worth pausing to consider that emergency physicians have a lot in common with our Fire/EMS colleagues.

If there's one thing we share, it's dedication to a difficult job.

No one else fully understands what it's like to do what we do. Held to the highest standard despite limited time and resources, we are the experts at resuscitation and stabilization of critically ill, undifferentiated patients. On a daily basis, we come face-to-face with many of the greatest challenges of our society — from drug abuse and violent crime to poverty and mental illness. As the safety net for our communities, we operate on the frontlines of a broken health care system. Above all else, we share the common mission of providing the best possible care to our patients.

Like many emergency physicians, I started off as a firefighter.

Sometime during my first year out of the fire academy, I remember walking upstairs to the station locker room on a slow morning. We had only run one call that day, so I planned to get a head start on the daily chores. I stopped by the utility closet looking for the broom and mop, only to discover that someone else had already taken them. I continued into the locker room to find the department's senior firefighter with the mop in his hand. He was just finishing up with the last section of the floor. I quickly offered to take over, but he just smiled and shook his head. "I've got this," he said.

It was a simple gesture, but that moment helped me understand the importance of pride and how it can shape the culture of an organization. Despite being one of the most experienced and respected members of the department, our highest-ranking firefighter demonstrated that he was willing to help with whatever needed to be done — even

if it meant mopping the floors.

It became apparent to me early on in my career that the culture of the fire service is one of respect, dedication, and hard work. Firefighters report for duty every day knowing well that at a moment's notice, their lives may depend on the person sitting next to them at morning changeover. This sobering reality is reflected in nearly everything they do.

Despite our many similarities, Fire/EMS operators have a superpower that far too often is severely lacking in the ED: taking pride in every aspect of their work — whether that's rescuing someone from an active fire or mopping the floors at the station.

No matter the setting, respect is earned through hard work and dedication. This holds true across every rank and experience level. If someone is willing to go the extra mile to do a great job on a small or minor task, that speaks volumes about their pride for their work. In this, we can all learn something from our Fire/EMS colleagues. ★

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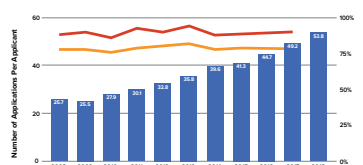


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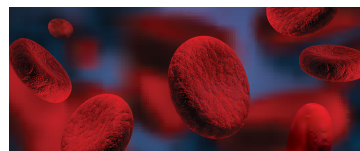


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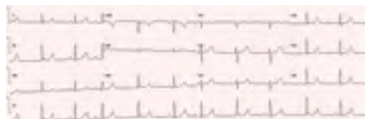


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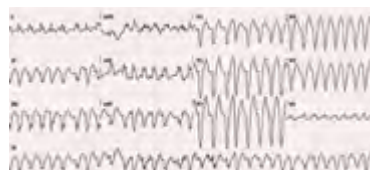
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- April 5:** *EM Resident* Articles Due
- April 22–25:** EMRA Events @ CORD Academic Assembly in San Antonio
- May 5–8:** ABEM Spring Oral Certification Exam
- May 15–18:** Essentials of Emergency Medicine — discount for EMRA members! The Cosmopolitan, Las Vegas
- May 20–23:** ACEP Leadership & Advocacy Conference
- June 1:** Registration opens for ACEP18, the 50th anniversary meeting!
- June 7-9:** AMA RFS Interim Meeting, Chicago
- June 19-22:** Resuscitation 2018 — discount for EMRA members! Baltimore, MD
- Aug. 5:** EM Resident Articles Due
- September (all month):** EM Day of Service
- Sept. 28-Oct. 4:** EMRA Events at ACEP18, San Diego
- Oct. 13-16:** ABEM Fall Oral Certification Exam
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## Congratulations to the Medical School Class of 2018

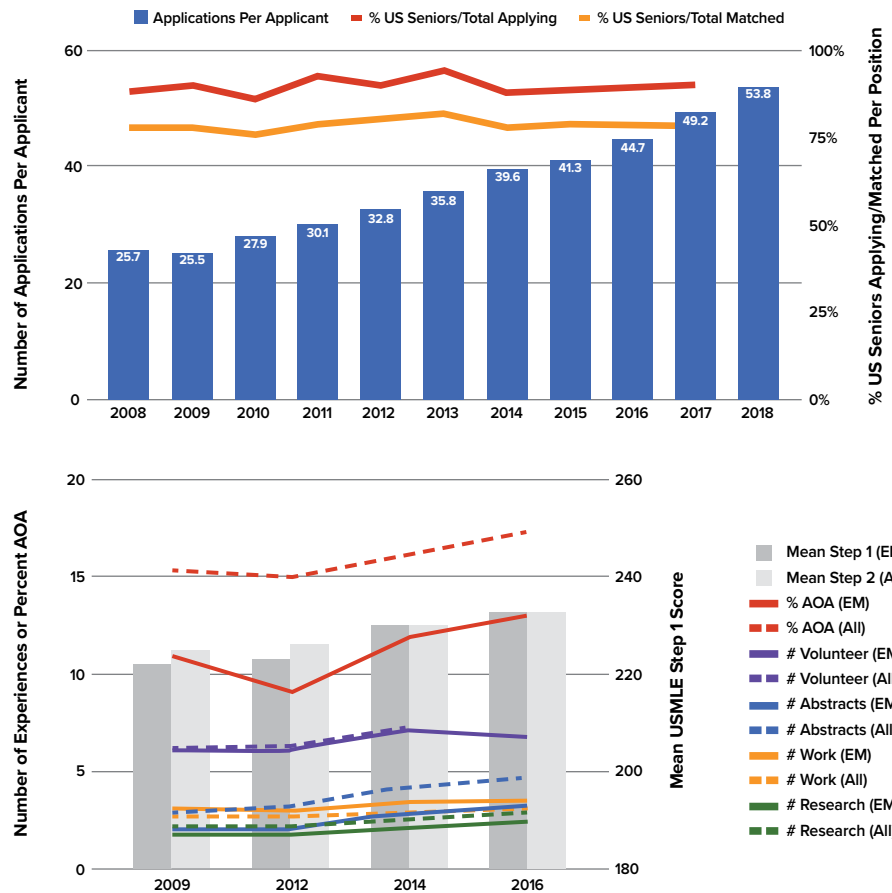
Every year on the third Friday of March, medical students across the country open their “Match Day” envelopes and (hopefully) breathe a sigh of relief to discover they will be training at one of their top-choice programs. Across all specialties, half of all U.S. seniors will end up with their top-ranked program, and three-quarters of seniors will end up with one of their top three choices.

### Over-application & Filters

The percentage of students matching at their preferred programs has remained very stable over the past several years. Unfortunately, a number that has NOT remained stable is the number of emergency medicine programs that students are applying to. Today, students are applying to **twice the number of residency programs** than they were a decade ago: 53.8 in 2018, compared to only 25.7 in 2008. And while it's not entirely clear what's driving this phenomenon, program directors — now inundated with more applications than ever before — are forced to resort to filtering applications, and new tools like the AAMC Standardized Video Interview are being piloted to help select applicants with the right skill sets to become excellent emergency physicians.

### EM Is Average

Why are students doing this? The most common response I hear is “because emergency medicine is becoming more competitive.” However, I have yet to find any convincing evidence that this widely held belief is true. Approximately 4 out of every 5 EM residency spots continues to be filled by U.S. seniors each year, and over the past decade, the four NRMP “Charting Outcomes in the Match” reports that have been released show that mean Step 1 scores and number of volunteer and work experiences for EM applicants remain average. In fact, EM consistently falls below average in terms



of the percentage of Alpha Omega Alpha members matched and the average number of research experiences and abstracts/posters presented.

### Apply Smarter, Not Harder

While the NRMP’s “Charting Outcomes” helps students predict their likelihood of matching based upon the number of programs on their rank list, it doesn’t help students understand the number of programs they need to apply to in order to secure enough interviews to build a rank list that will guarantee a match. This is where the AAMC’s new **Point of Diminishing Returns** figures come into play. For example, students with low Step 1 scores (<=215) applying to EM will experience diminishing returns after 32 programs, while students with Step 1 scores at or above the national average (>=234) will experience diminishing returns after only 18 programs. Students should seek the advice of a trusted advisor to fine-tune their application strategy,

based upon their individual goals and competitiveness, rather than scatter-shooting for residency positions.

### EMRA Match for Clerkships

For students heading into their final year of medical school, spring is the season to secure away rotations. Approximately 20% of career-track, adult EM away rotations are not listed on VSAS. Fortunately, EMRA is here to help with the launch of our new **EMRA Match for Clerkships**, a collaborative, filterable, crowd-sourced clerkship directory created with our friends at CDEM, CORD, and ACEP. Students will also be able to learn:

- Which sites offer subspecialty rotations or diversity externship scholarships?
  - Which rotations require Step/COMPLEX?
  - Is a car recommended?
  - Is there an on-site housing option?
- Easily sort through hundreds of opportunities to find the one that’s right for you! ★



# Out of the Blue

## Management of Methemoglobinemia

**Joshua Glick, MD**

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**A** 56-year-old male presented following an intentional overdose of an unknown amount of dapsone. The patient was initially confused and vomiting, with an exam notable for an oxygen saturation of 80% with peripheral and perioral cyanosis. During peripheral IV insertion, the nurse noted his blood was chocolate-brown in appearance and his initial venous blood gas demonstrated a methemoglobin level of 58%.

### Pathophysiology and Causes

Methemoglobinemia is characterized by an alteration in hemoglobin in which iron molecules are oxidized from the ferrous to the ferric form, ultimately leaving them unable to reversibly bind oxygen. Methemoglobin also causes a “left-shift” on the hemoglobin-dissociation curve, in which the affinity of oxygen to the hemoglobin molecule is increased and peripheral dissociation is decreased. The net result of these effects is a profound functional anemia.

In healthy humans, red blood cells are continually and spontaneously oxidized into a methemoglobin state as a result of normal metabolism. Two naturally occurring enzymes, NADPH reductase and CYP-5 reductase, typically reverse

this process and maintain levels at less than 1% in an unstressed state. When either of these enzymes is overwhelmed or functionally deficient, methemoglobin levels increase at an uncontrolled rate.

Methemoglobinemia can be attributed to either acquired or hereditary causes. Hereditary causes are quite rare, but include cytochrome b5 reductase deficiency and hemoglobin M disease. Most instances are due to an acquired state, which can be caused by a wide variety of chemical and pharmacologic exposures. The most common pharmacologic causes include dapsone, topical anesthetics (benzocaine, lidocaine, and prilocaine), amyl nitrate, nitroglycerin, phenazopyridine, rasburicase, and sulfonamides. Other chemical exposures include aniline



dyes, antifreeze, hydrogen peroxide, naphthalene, paraquat, and benzene derivatives.

### Clinical Presentation and Diagnosis

Symptoms of methemoglobinemia largely depend on the severity of poisoning and ultimately stem from a lack of effective oxygen delivery throughout the body. Methemoglobin levels less than 20% are usually asymptomatic unless underlying cardiopulmonary diseases are present, although subtle cyanosis may be present. As levels rise between 20-50%, symptoms include blue-grey discoloration of the skin and mucous membranes, nausea, vomiting, light-headedness, headache, fatigue, lethargy, and mild dyspnea. Levels between 50-70% are more life-threatening and can present with respiratory depression, altered mental status, coma, and seizures. Levels higher than 70% are typically fatal.

The diagnosis is typically made based on a clinical presentation of acute cyanosis and hypoxia that does not improve with increased administration of oxygen, in the setting

of a known pharmacologic exposure. Chocolate discoloration of the blood is a classic exam finding, and the level of discoloration has been shown to correlate to the degree of poisoning. Of note, peripheral pulse oximetry is inaccurate and classically displays values between 80-85% because of impairments in wavelength interpretation. The diagnosis is ultimately confirmed by measurement of methemoglobin level on blood gas analysis, and is generally recorded as a "MetHb" percentage.

### Treatment

Methylene Blue is the standard first-line antidote for methemoglobinemia. The drug acts as a cofactor for NADPH reductase and ultimately increases the rate of conversion of ferric methemoglobin to ferrous hemoglobin. It is dosed at 1-2 mg/kg given as a slow push over 5 minutes, with repeat dosing every 30-60 minutes until clinical improvement is observed and methemoglobin levels are corrected. The maximum recommended daily dose is 7 mg/kg. Methylene blue is not recommended in patients with G6PD given the possibility of hemolysis.

There are several additional treatment strategies that have been described in the literature as adjunctive therapies to be considered if methylene blue is not available or is contraindicated. Vitamin C (ascorbic acid), given as a high dose infusion, has been shown to correct methemoglobinemia, but is generally not recommended as a first line agent given its slower mechanism of action. Exchange transfusion with packed red blood cells has been shown to help in poisoned patients who cannot get methylene blue, but does not ultimately reverse the underlying physiologic defect. Finally, there have been several case reports of methemoglobinemia treated with hyperbaric oxygen therapy, but there is no currently accepted course and treatment may ultimately require transfer to a tertiary care facility.

### Case Conclusion

The patient was immediately given methylene blue (2 mg/kg) with improvement in his confusion, vomiting, cyanosis, and pulse oximetry within 20 minutes. He remained stable and was discharged to a psychiatric facility one day later. ★

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# Nitrous Oxide Toxicity

## A Rising Concern as Street Use Surges

**Steven W. Flynn, MD**

*University of Chicago*

**Navneet Cheema, MD**

*Northwestern University*

**N**itrous oxide (N<sub>2</sub>O) toxicity is an uncommonly encountered patient complaint in the emergency department; however, it is a diagnosis the emergency physician should remember, as it could be responsible for unexplained neurological symptoms. N<sub>2</sub>O was first developed in 1775 by Joseph Priestley after combining nitric acid with iron shavings. Its potential medical use was later noted by Sir Humphrey Davy who, after inhaling the gas, noted the qualities of euphoria and anesthesia and suggested its use for surgery.

Similar to today, the potential for abuse was quickly recognized, and soon the British elite were holding “laughing parties” where they inhaled the gas for entertainment.<sup>1</sup> Since its discovery N<sub>2</sub>O has been used in various ways, including general anesthesia, pain control, food preservation, air compressors, additive to fuel to enhance combustion, and for the treatment of withdrawal of nicotine, opioids, and cocaine.<sup>2</sup> In the 1980s, N<sub>2</sub>O became a popular street drug and is commonly known today as laughing gas, hippy crack, and whippets.

### Uses/Abuse

One reason N<sub>2</sub>O is so commonly abused is the ease with which it can be obtained. It is sold as whippets, which



are small containers of N<sub>2</sub>O intended to be used for refillable whipped cream containers or to inflate balloons. Recreational use of N<sub>2</sub>O varies across the world, with the United Kingdom logging the highest prevalence of use. Within the U.S. the lifetime use prevalence is approximately 29%.

A single bulb is approximately equal to a single hit. After inhalation, symptoms of euphoria and sometimes visual hallucinations ensue within seconds and then dissipate after several minutes.<sup>2</sup> Adverse events are related to the number of hits per session and concomitant use of another drug or alcohol. Typical adverse events include fainting, nausea, and accidental injury. At higher doses (about 27 hits/session), accidental injury has been shown to be common, whereas at lower doses (14 hits/session), nausea was the prevailing symptom.<sup>1</sup> The typical individual will do fewer than 5 hits in a single session. Patients presenting with neurologic complications typically do 10-100 hits in a session over a long-term course.<sup>1,3</sup>

### Mechanism of Toxicity

There are several different biochemical pathways that contribute to the toxicity of N<sub>2</sub>O. B<sub>12</sub> is an essential vitamin that is needed for brain and nervous system. Nitrous oxide oxidizes the cobalt moiety on B<sub>12</sub> and renders it inactive. The downstream effect of this is that methionine synthase — which requires active B<sub>12</sub> to function — is irreversibly blocked. This leads to depletion of methionine and tetrahydrofolate, which are required for DNA synthesis and myelin production. In turn, this depletion results in a clinical picture that resembles the characteristic findings of pernicious anemia (bone marrow depression and polyneuropathy).<sup>4-6</sup> This demyelination can be seen on MRI and is diagnosed as Subacute Combined Degeneration (SDG).<sup>7</sup>

### Symptoms: Hematological and Neurological

Side effects of long-term nitrous oxide use vary. The most common side effects that may be encountered in the ED are peripheral neurologic disorders. As discussed earlier, N<sub>2</sub>O has a large impact on myelin resulting in a sensorimotor polyneuropathy, often with combined signs of lateral and posterior cord involvement. Patients present with numbness, tingling, weakness, and truncal ataxia. These clinical symptoms are indistinguishable from SDG seen with B<sub>12</sub> deficiency. Bone marrow suppression with a macrocytic anemia can be seen on lab testing.

Other less common manifestations of toxicity include frostbite of the mouth (secondary to breathing it directly from pressurized tanks), urinary retention, painful erection, decreased libido, delusions, paranoia, anxiety, and even death.<sup>2,4</sup> Asphyxiation is seen in patients who inhale in closed spaces such as cars, face masks, or bags over the head resulting in a low partial pressure of oxygen.<sup>2</sup>

### Testing and Diagnostics

There are no ideal lab tests to evaluate for nitrous oxide toxicity in the ED because B<sub>12</sub> levels may be low, normal, or even high, especially if patients are taking B<sub>12</sub> supplements at home. Typically, methylmalonic acid (MMA) and homocysteine levels will be elevated; however, these tests are not routinely performed in the ED.<sup>8</sup> In patients with more severe symptoms (incontinence, paresthesias, and motor deficits) an MRI may be obtained. The distribution of the demyelination may not always correspond to the symptoms being demonstrated.<sup>7</sup>

### Treatment

Treatment is aimed primarily at stopping the exposure after other potential etiologies have been ruled out,

such as syphilis, HIV, mercury toxicity, copper deficiency, cord compression, and electrolyte deficiency. Afterward, B<sub>12</sub> can be administered as IM shots. There is no standardized B<sub>12</sub> regimen, but a commonly accepted plan is 1000µg IM for 5 days and then intermittently until symptoms resolve. An alternative plan is 1000µg-2000µg PO daily for 1-2 weeks and then 1000µg daily.<sup>7</sup>

When the presenting symptoms are minor, such as decreased vibration sense or decreased peripheral sensation, it may be acceptable to discharge these patients with a prescription for PO B<sub>12</sub> and close outpatient neurology follow-up. In patients exhibiting more severe symptoms, such as ataxia and motor deficits, admission for imaging and further evaluation may be warranted. Even with B<sub>12</sub> replacement and cessation of use, patients may never return to their baseline and may have permanent neurologic symptoms.<sup>8</sup>

Patients can be given basic information on safe practices of recreational N<sub>2</sub>O. These basic practices include avoiding use with other drugs or alcohol, using while seated to prevent falls, following up early with a doctor if peripheral neurological symptoms occur, using in ventilated spaces, and avoiding placing bags of N<sub>2</sub>O over the head do not put bags of N<sub>2</sub>O over the head.<sup>1</sup>

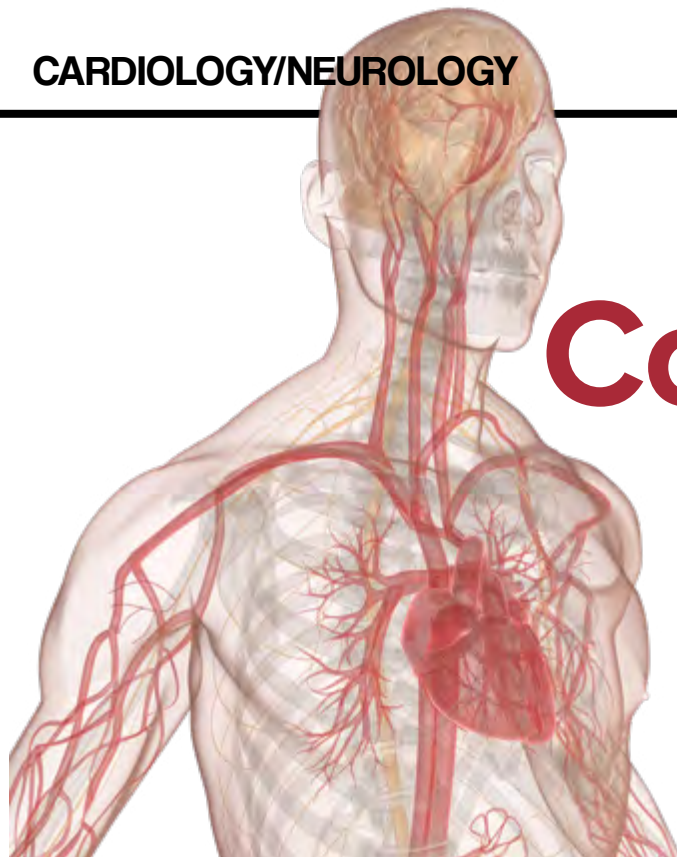
### Conclusion

As EM providers we must remember to consider N<sub>2</sub>O toxicity as a potential etiology of vague neurological complaints, especially paresthesias. Cessation of use and administration of B<sub>12</sub> are the mainstays of treatment; however, symptoms do not always resolve completely, and some effects can be permanent. All but the mildest of symptomatic patients should be admitted to receive B<sub>12</sub> and neurology consultation. ★

**Since its discovery N<sub>2</sub>O has been used in various ways, including general anesthesia, pain control, food preservation, air compressors, additive to fuel to enhance combustion, and for the treatment of withdrawal of nicotine, opioids, and cocaine.**

# Cardiac Complications

## FROM Subarachnoid Hemorrhage



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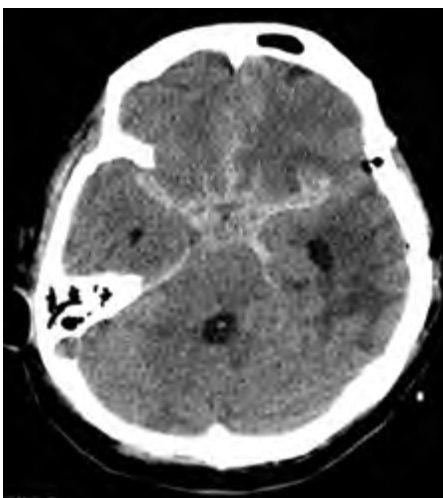
A 50-year-old male is brought in by EMS after pulseless electrical activity (PEA) cardiac arrest. Reportedly, he was well earlier in the day before family witnessed him collapse with seizure-like activity. Bystanders began CPR before EMS initiated care for PEA with subsequent ROSC. Post-arrest, the patient was hypotensive with a 12-lead electrocardiogram (EKG) notable for third degree heart block. On arrival to ED, he was being transcutaneously paced. Norepinephrine was started and a transvenous pacemaker was placed with stabilization of his hemodynamics. The patient was taken to CT and found to have a high-grade aneurysmal subarachnoid hemorrhage from a ruptured aneurysm of the anterior communicating artery.

*How does subarachnoid hemorrhage lead to cardiac arrest, complete heart block, or cardiomyopathy? How frequently does this occur? What is neurogenic stress cardiomyopathy? How does it threaten the neurologically injured patient, and what implications does it have on their care?*

### Introduction

During aneurysmal subarachnoid hemorrhage (aSAH), extravasation of high-pressure arterial blood into the subarachnoid space leads to a sudden intracranial pressure (ICP) increase that can compromise cerebral perfusion. Blood is most commonly found around the circle of Willis (~65%) due to the proximity of the majority of aneurysms arising within this region, or in the Sylvian fissure (~30%).<sup>1</sup> Within the Sylvian fissure underlying the cortex lie the insular lobe and deeper hypothalamus which have important clinical implications, to be discussed later.

(Figure 1)



**FIGURE 1. Noncontrast Enhanced CT Scan**  
IMAGE BY MUSTAFA MOHAMED ALIKHAN VIA MEDPIX

A subset of subarachnoid hemorrhage patients present after cardiac arrest, often in PEA. Among all PEA arrest cases, approximately 7% are due to intracranial hemorrhage.<sup>2</sup> In distinction from other stroke patients,<sup>3</sup> aSAH occur in younger patients without cardiovascular risk factors. An exploration into neurocardiology—the unique interface between the central nervous and cardiovascular systems—will help explain how a neurologic insult such as non-traumatic aneurysmal subarachnoid hemorrhage (aSAH) can lead to acute cardiac dysfunction.

### Cardiac Manifestations

Myocardial injury commonly occurs following aSAH, with 28% of patients with troponin elevations in the first 24 hours,<sup>4</sup> 35% with arrhythmias,<sup>5</sup> and 28% with wall motion abnormalities.<sup>6</sup> Classically, deep septal T-wave inversions (cerebral T waves) and QTC prolongation (associated with insular injury) can be found on EKG. (Figure 2)

While almost all patients will have some abnormality on EKG, and the degree of abnormality is proportional to the cerebral insult,<sup>7</sup> a wide range of findings can be observed. Life-threatening arrhythmias are seen in just 5% of cases.<sup>5</sup>

The degree of troponin elevation correlates with both intracranial injury,<sup>8</sup>

left ventricular (LV) dysfunction, and mortality.<sup>9,10</sup> Regional wall motion abnormalities (RWMA) WMAs extending beyond single epicardial distribution hint at the underlying pathophysiology (to be discussed below), and occur more frequently among postmenopausal women with aSAH.

Transient LV dysfunction in the setting of neurologic injury is commonly described as “neurogenic heart” or “neurologic stunned myocardium” and will be referred to as *neurogenic stress cardiomyopathy* (NSCM) in this article. Many other disease entities have been described to cause a characteristic, self-resolving LV dysfunction, and the medical literature describes over 70 different terms.<sup>11</sup> Definitions of neurogenic stress cardiomyopathy vary but often require the both reversible or transient LV dysfunction to be reversible and a negative coronary angiogram. Estimates from retrospective studies suggest that the incidence of neurogenic stress cardiomyopathy in patients with aSAH range from 1-5%,<sup>12,13</sup> although these studies likely underestimate the true prevalence. Importantly, LV dysfunction is associated with high-grade SAH and is associated with a mortality of 26%.<sup>12</sup> (Figure 3)

### Pathophysiology

NSCM was thought to be caused by myocardial ischemia or coronary vasospasm in response to a surge in adrenal circulating catecholamines, but more likely is directly related to myocardial sympathetic stimulation by a damaged insular cortex.<sup>14-16</sup> This results in myocardial contraction, adenosine triphosphate depletion, mitochondrial dysfunction, and myocardial cell dysfunction or death.<sup>17</sup> The typical apical and mid-ventricular ballooning pattern of neurogenic stress cardiomyopathy reflects the differential distribution of myocardial sympathetic nerve terminals.<sup>18</sup> The subendocardial myocardium receiving autonomic innervation also houses the cardiac conduction system, predisposing injured subendocardial myocytes to arrhythmogenicity.



**FIGURE 2. Cerebral T-wave**  
IMAGE BY MATTHEW HOLT VIA EMRA EKG GUIDE

### Management

Emergent care for the aSAH patient has two concurrent priorities:

1. Obliteration of the aneurysm.
2. Optimizing the hemodynamics and cerebral perfusion to avoid secondary injury.

Prior to aneurysmal repair, the latter entails avoiding hypertension (SBP<160) to decrease risk of rebleeding, the most feared early complication. Rebleeding has a 22% incidence, often occurs in the first 6 hours,<sup>19</sup> and is associated with 67% mortality.<sup>20</sup> Cerebral autoregulation—response of cerebral vasculature to changes in systemic blood pressure by altering vascular tone in order to ensure steady perfusion—is impaired in the first 24 hours (or beyond) in the neurologically injured patient, so the brain is exquisitely sensitive to fluctuations in blood pressure.

The cornerstone of treatment for patients with concurrent NSM and SAH is optimizing cardiac output to ensure adequate cerebral perfusion. Cardiac dysfunction is often early and transient (1-3 days);<sup>6</sup> however, neurogenic stress cardiomyopathy can be prolonged for weeks. If vasopressor support is required, norepinephrine is a reasonable first line agent, and dobutamine may be added for additional inotropy. Rarely, mechanical circulatory support devices such as Impella<sup>21</sup> or intraaortic balloon pump<sup>22</sup> are used.

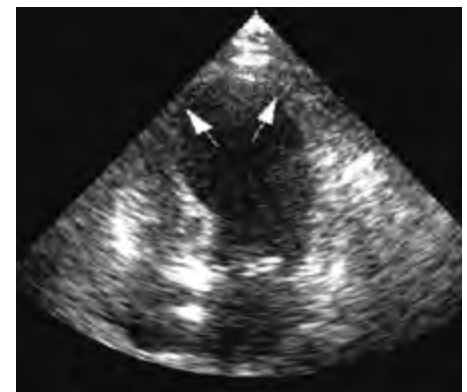
In addition to potentially impaired cerebral perfusion due to depressed cardiac output, NSM presents several other management challenges. First,

induction of anesthesia for aneurysm coiling or clipping procedures often results in further myocardial suppression, which can result in hemodynamic collapse in patients with severe NSM. Second, a major late complication of aSAH is vasospasm leading to delayed cerebral ischemia. It patients are unable to maintain adequate cerebral perfusion pressures in the face of high cerebrovascular resistance due to decreased cardiac output, cerebral ischemia will develop. These are patients who often require inotropic or mechanical support.

### Case Conclusion and Takeaways

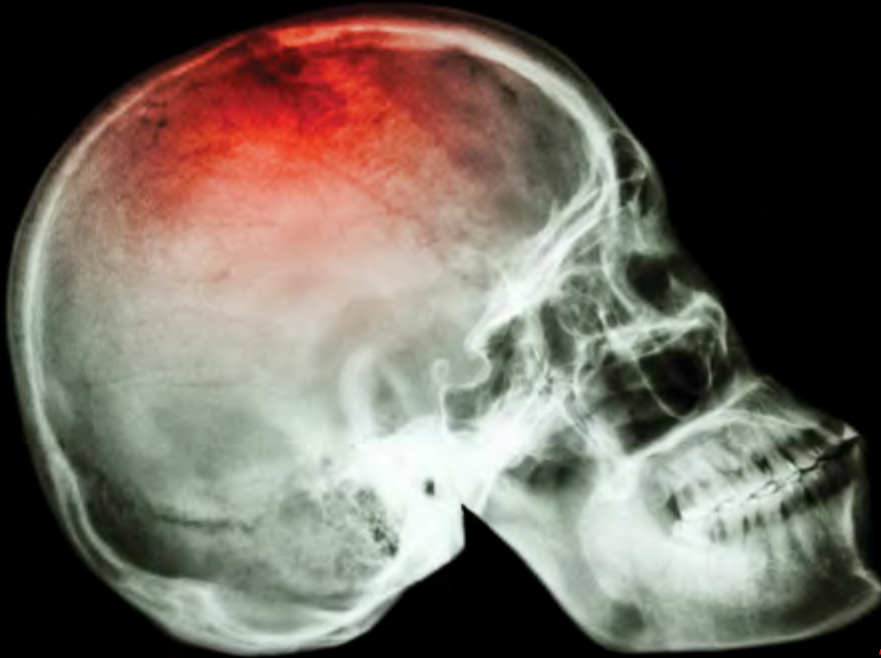
The patient had an external ventricular drain placed in the ED and was admitted to the neurological intensive care unit. He was taken for cerebral angiography and his aneurysm was coiled. He remained on a norepinephrine infusion to maintain adequate cerebral perfusion pressure. A permanent pacemaker was placed, and he was discharged with good neurologic function.

In summary, cardiovascular complications from aSAH are common and neurogenic stress cardiomyopathy can be severe. Emergency neurological life support (ENLS) recommends early screening for cardiac dysfunction, including obtaining an EKG and troponin within the first hour of ED presentation.<sup>23</sup> Maintaining normal blood pressure is essential, as aSAH patients may lose the ability to autoregulate cerebral blood flow and are particularly vulnerable to secondary ischemic insults. Treatment is supportive and may require vasopressors, inotropes, or rarely mechanical support to ensure adequate cerebral perfusion. ★



**FIGURE 3. Takotusbo Cardiomyopathy**  
IMAGE BY TARA C. GANGADHAR, ELISABETH VON DER LOHE, STEPHEN G. SAWADA, AND PAUL R. HELFT VIA CREATIVE COMMONS





# Revisiting Cerebral Perfusion Pressure

## A Framework for Approaching Traumatic Brain Injury

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In the United States, more than 2.5 million patients seek care in the ED for traumatic brain injury (TBI) every year, accounting for approximately 2% of all ED visits.<sup>1</sup> Caring for TBI in the ED can be a daunting task, as there are numerous variables to consider, and care recommendations are constantly evolving.

In this article, we revisit the equation for cerebral perfusion pressure (CPP) as a simplified framework to approach TBI care in the ED. Optimization of CPP is necessary to meet the metabolic needs of the injured brain, prevent secondary injury, and minimize future disability.<sup>2</sup> The equation is as follows:

**Cerebral perfusion pressure = Mean arterial pressure — Intracranial pressure (CPP = MAP - ICP).**<sup>3</sup> The current goal CPP is thought to be between **60 mmHg and 70 mmHg.**<sup>2,4,5</sup>

In this equation, we can see that CPP is contingent on the pressure gradient between MAP and ICP. By evaluating our strategies for controlling MAP and ICP, we can form a conceptual framework for management of TBIs in the ED.

### Mean Arterial Pressure

In TBI, it is important to avoid both hypotension and hypertension, which result in cerebral ischemia and vasogenic edema, respectively<sup>4</sup>. For patients with severe TBI, **early ICP monitoring** should be obtained in consultation with a neurosurgeon to allow for titration of the patient's blood pressure (BP) to target CPP goals. Before ICP monitoring is in place, it is recommended to maintain MAP between 80 mmHg and 110 mmHg (space between the number and the measurement abbreviation).<sup>4</sup>

**Manage hypertension** with titratable, short-acting beta-blockers and calcium channel blockers such as nicardipine, clevidipine, and esmolol.<sup>6</sup>

**Treat hypotension** with fluid boluses or vasopressors such as norepinephrine, epinephrine, and phenylephrine. Identify and address underlying causes of hypotension, such as hypovolemic or neurogenic shock.

Although **airway management** may be necessary for severe TBI, intubation can be accompanied by undesirable hemodynamic changes. Laryngoscopy and tracheal manipulation lead to sympathetic stimulation, which provokes elevation in heart rate and BP. Consider **pretreatment** with fentanyl to blunt the catecholamine surge resulting from direct laryngoscopy. Lidocaine for pre-treatment has not been shown to have significant effect on ICP, CPP,

or MAP in response to laryngoscopy.<sup>6,7,8,9</sup>

Conversely, airway management can also lead to hypotension due to hemodynamic effects of induction agents, sedation agents, and positive pressure ventilation. Close monitoring, judicious medication selection, and optimization of MAP prior to intubation are necessary to avoid negative outcomes.

**Induction medications** to use in TBI include ketamine, propofol, or etomidate. Ketamine, ostracized in the past due to concern of increased ICP, has re-emerged as a reasonable choice of induction agent. In addition to its favorable hemodynamic profile, a growing body of evidence suggests that ketamine does not adversely affect ICP, CPP, neurologic outcomes, or mortality in TBI patients.<sup>6,9,10</sup>

**Paralysis** can be employed with a variety of neuromuscular blocking agents, such as succinylcholine or rocuronium. Succinylcholine has a short duration of onset and can be beneficial for patients requiring frequent neurologic reassessments. However, providers should recall standard contraindications to succinylcholine that may present risk to patients suffering TBI, such as crush injury and prolonged immobilization.<sup>6,9</sup>

**Post-intubation sedation and analgesia** is important for a variety of reasons: it minimizes patient anxiety and

agitation, reduces the cerebral metabolic rate of oxygen consumption, and facilitates mechanical ventilation.<sup>4</sup> Propofol has come into favor as a sedation agent in TBI as it decreases the cerebral metabolic rate and lowers the seizure threshold, as well as being rapidly metabolized to facilitate frequent neurological checks.<sup>4,9</sup> Analgesics such as fentanyl, morphine, and hydromorphone may also be used in conjunction with sedative agents.<sup>4,9</sup>

### Intracranial Pressure

The adult skull is an enclosed space and can hold a fixed volume. Therefore, a change in the volume of one of the contents must result in a change in volume of one of the other contents. The normal brain can autoregulate cerebral blood flow within a certain range of MAP and CPP, but in the setting of TBI, autoregulation can become compromised and require intervention.<sup>3,6</sup> The goal ICP is <22 mmHg. We can look at the interventions available to providers by going through each of the contents of the skull.

#### Vessels/blood

**Carbon dioxide** causes vasodilation (via H<sup>+</sup>) of cerebral blood vessels such that PaCO<sub>2</sub> has a linear correlation with cerebral blood flow between 20 and 80mmHg.<sup>3,10</sup> In intubated patients, it is important to avoid both hyperventilation, which results in ischemia, and hypoventilation, which results in hyperemia.<sup>3</sup> **Goal PaCO<sub>2</sub> is between 35-38 mmHg.**<sup>9</sup> Hyperventilation to

PaCO<sub>2</sub><30 mmHg is no longer globally recommended but can be considered in cases where herniation appears imminent.<sup>11</sup>

**Oxygen** is necessary for the high metabolic demands of the brain. **Maintain SpO<sub>2</sub> >95%**, as hypoxia has been linked to poor clinical outcomes in TBI patients, particularly in conjunction with hypotension.<sup>3</sup> In patients requiring airway management, adequate preoxygenation, apneic oxygenation, and avoidance of prolonged hypoventilation can prevent desaturation during intubation.

**Cervical collars** inhibit cerebral venous drainage and thereby increase ICP. Aim to clear the C-collar as soon as possible.

**Raise the head of the bed** to facilitate venous drainage.

#### CSF

**External ventricular drains (EVDs)** are inserted to provide continuous or intermittent drainage of CSF, and have shown benefit in TBI patients with a GCS<6.<sup>10</sup> These can be done by a neurosurgeon at the bedside in the ED.

#### Parenchyma

**Mannitol or hypertonic saline** can be used to decrease cerebral edema. The debate continues as to which is more effective, as the current evidence does not support the use of one over the other in terms of morbidity and mortality outcomes. Arguments toward the use of HTS include greater decreases in ICP, longer duration of action, and

lower incidence of acute renal failure and dehydration when compared to mannitol.<sup>12,13</sup>

**Fluid resuscitation** should be employed with iso-osmolar fluids such as 0.9% NaCl and PlasmaLyte to avoid cerebral edema.<sup>14</sup> Avoid lactated Ringer's and other hypo-osmolar fluids, such as D5 0.45%NaCl.<sup>14</sup>

#### Skull

**A decompressive craniectomy** may be a reasonable surgical option for the management of severe TBI. To date, there have been two multi-center randomized controlled trials examining morbidity and mortality outcomes in TBI patients with elevated ICP refractory to medical management: DECRA<sup>15</sup> and RESCUEicp.<sup>16</sup> These trials have demonstrated some conflicting findings, and the effect of decompressive craniectomy in TBI remains unclear. Craniectomies have likely been linked to decreased ICP and reduced mortality in patients with TBI, but they have also been linked to increased survival with vegetative state and severe disability, theorized to be due to axonal stretch as the brain is allowed to stretch outside the bounds of the skull.<sup>11,15,16</sup> The decision to perform a craniectomy should involve family members to determine individual values and goals of care.

**Burr holes** may be employed emergently for suspected or known epidural hematoma with signs of herniation, especially in hospitals without ready access to neurosurgical care.<sup>17,18</sup>

#### Conclusion

In this article, we have suggested a framework for conceptualizing TBI treatment based on the equation for calculating CPP: CPP = MAP – ICP. We have reviewed management options and treatment goals for MAP and ICP, which are summarized in the table below.

Patients requiring airway management are particularly vulnerable to hemodynamic disturbances and hypoxia, and these potential sources of secondary injury should be anticipated and mitigated.

Early neurosurgery consultation for patients with severe TBI is essential, as these patients may benefit from invasive procedures, such as ICP monitoring, external ventricular drains, or decompressive craniectomy. ★

**TABLE 1. CPP = MAP – ICP**

#### MAP (goal 80 mmHg-110 mmHg)

- Anti-hypertensives: nifedipine, clevidipine, esmolol
- Vasopressors: norepinephrine, epinephrine, phenylephrine
- Airway management
  - Maximize SpO<sub>2</sub>, MAP, PaCO<sub>2</sub>
  - Pretreatment (fentanyl, esmolol)
  - Induction (ketamine, etomidate, propofol)
  - Paralytics (succinylcholine, rocuronium)
- Post-intubation sedation/analgesia (propofol, opioids)

#### ICP (goal <20 mmHg)

- Ventilation: PaCO<sub>2</sub> 35-38 mmHg
- Oxygenation: SpO<sub>2</sub> >95%
- HOB elevated
- C-collar clearance
- HTS or mannitol
- Fluid resuscitation with iso-osmolar fluids
- Neurosurgery interventions: ICP monitor, EVD, craniectomy, craniotomy

# THROMBOSIS

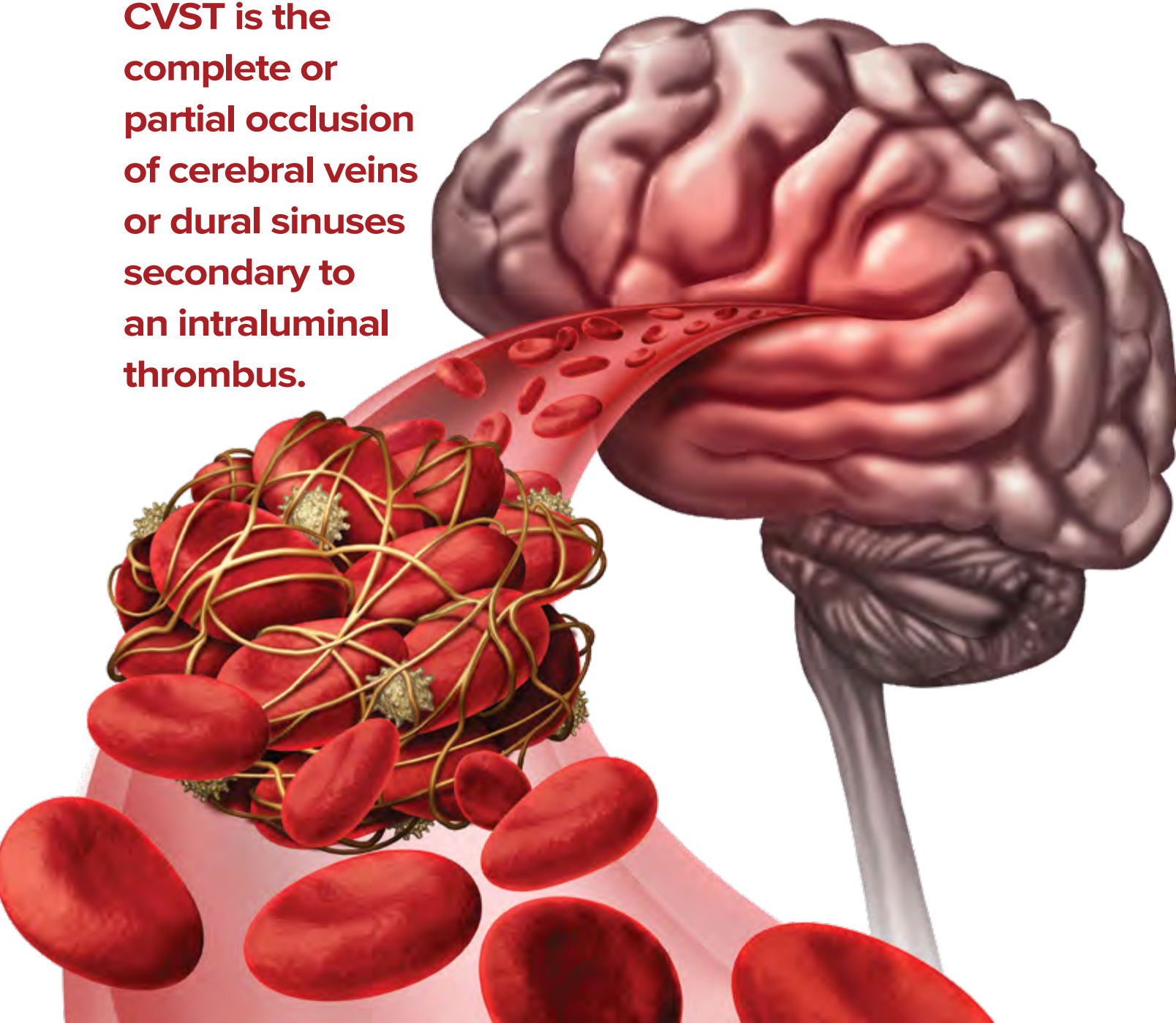
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## in the Brain

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**CVST is the complete or partial occlusion of cerebral veins or dural sinuses secondary to an intraluminal thrombus.**





**A** 45-year-old female with a history of hypertension and ulcerative colitis presents to the ED with a headache that started 2 weeks ago and has not gone away. She believes it is because of her high blood pressure (in triage, it is noted to be 200/108). This morning she woke up with numbness on the left side of her face, nausea, vomiting, and difficulty walking. On physical examination, you note a left facial droop, a left pronator drift, and unsteady gait. She is outside your institution's stroke alert activation window. You obtain a stat head CT, which demonstrates a right thalamic infarction. Given her symptoms, you suspect a possible uncommon cause of her stroke, and perform CT venography, which reveals the presence of cerebral venous sinus thrombosis.

### **What is cerebral venous sinus thrombosis (CVST)?**

CVST is the complete or partial occlusion of cerebral veins or dural sinuses secondary to an intraluminal thrombus. It is a relatively rare condition, with an annual incidence of 0.2 to 0.5 per 100,000<sup>1</sup>. It is 3 times more common in women, and is often associated with sinusitis, ear infections, trauma, surgery, hypercoagulable states, medications such as oral contraceptive pills (OCPs), steroids, and hematologic and inflammatory conditions such as ulcerative colitis (UC) and Systemic Lupus Erythematosus (SLE).<sup>2</sup>

### **How does cerebral venous sinus thrombosis present?**

Headache is the most common symptom associated with CVST. Although the characters of the pain may be variable between individuals, the pain often starts gradually, and progress in severity over a period of days. In some cases, CVST may cause seizures, focal neurological deficits, and may even progress to encephalopathy in severe cases.<sup>2-5</sup>

### **How do you work up a patient for suspected cerebral venous sinus thrombosis?**

Patients with suspected cerebral venous thrombosis should undergo venous imaging. The American Heart Association/American Stroke Association (AHA/ASA) 2011 guidelines indicate that both CT head with venography and MRI head with venography are considered adequate tools for evaluating CVST.<sup>6</sup> Although digital subtraction angiography remains the gold standard for the diagnosis of CVST, this is increasingly less utilized given the accuracy of non-invasive imaging.

Several studies have examined the role of d-dimer levels in evaluating patients for CVST.<sup>7-9</sup> Although no formal guidelines exist on implementing this into clinical practice, the utility may be strictly limited to low-risk patients,<sup>8</sup> as data indicates that d-dimer may inaccurately exclude patients with subacute or chronic disease.<sup>9</sup>

### **How do you treat a cerebral venous sinus thrombosis?**

Systemic anticoagulation is the mainstay of therapy for CVST. Counterintuitively, this is often the case even in the presence of associated intracranial hemorrhage, although some controversy remains in the literature.

The body of literature is largely limited to 2 RCTS.<sup>10</sup> The first one was done in 1991 had to be stopped early due to mortality in the placebo group.<sup>11</sup> The second was done in 1999 found no statistically significant difference between anticoagulation and non anticoagulation groups.<sup>3</sup> A more recent observational study has shown a lower mortality than

has been previously reported, and this is believed to be partly secondary to wider adoption of anticoagulation.<sup>2</sup> There is also success reported with thrombectomy or local thrombolytic therapy.<sup>12</sup> However, this is generally reserved for severe cases.

Antibiotics should be considered in cases where a concomitant infection is suspected. CVST patients should be admitted, either to the ICU or step-down unit depending on the severity of their illness.

### **What is the prognosis of cerebral venous sinus thrombosis?**

If left untreated, CVST may progress from headache to seizures, unilateral or bilateral infarction with focal neurologic deficits, to elevated intracranial pressure, herniation and death. In the past, 'death or dependency' was reported as high as 39%, however more recently as low as 13%.<sup>2</sup> This may be secondary to improved recognition, general improvement of critical care or possibly to wider adoption of systemic anticoagulation in treating CVST.

### **Summary**

CVST is a can't-miss diagnosis in the ED given its high mortality if untreated. Suspect CVST in patients with gradually worsening headache and focal neurologic deficits, especially if they are high risk (SLE, UC, OCPs, procedures, trauma, or infection). Treatment involves anticoagulation and admission to neurologic care. ★

**The American Heart Association/American Stroke Association (AHA/ASA) 2011 guidelines indicate that both CT head with venography and MRI head with venography are considered adequate tools for evaluating CVST.**

# TACTICAL MEDICINE

## Providing Care in the Urban Battlefield

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### History of Tactical Emergency Medicine Services

**T**actical Emergency Medical Services (TEMS) was first recognized during the Civil War and in World Wars I and II. Rapid care of injured soldiers and transport to a hospital provided a better chance of surviving. On the civilian side, the emergency medical services

(EMS) system originated in 1966 with the publication of “Accidental Death and Disability: The Neglected Disease of Modern Society.”<sup>1</sup> The Los Angeles Watts riots of 1965, which left more than 1,000 wounded and 34 dead, and the University of Texas clock tower shooting in 1966, which killed +14 people and wounded 34 civilians, both influenced the first Special Weapons and Tactics unit by the Los Angeles Police Department in 1967.<sup>2</sup> SWAT units were composed of law enforcement officers trained to handle high-risk incidents. The benefit of TEMS to SWAT units was first presented in national conferences in 1989 and 1990 with representatives from law enforcement, emergency medicine, and EMS.<sup>3,4</sup> By 1994, the National Tactical Officers Association (NTOA) released its position statement recommending that “special operations teams (SWAT, Special Emergency Response Team, etc.) [should] include properly trained tactical emergency providers.”<sup>5</sup>

### The Golden Hour

The concept of the “Golden Hour” was first described by R. Adams Cowley from his experiences in combat and providing helicopter transport for injured patients in Baltimore.<sup>6</sup> He recognized that the sooner the patient reached definitive care, the more likely their survival. Throughout the years, it has been debated whether 60 minutes is truly the Golden Hour; currently, the standard of care is rapid transit to the hospital.<sup>7</sup> However, with the integration of physicians as tactical medical providers (TMPs) supporting SWAT units, potentially lifesaving care can be offered on-scene in the urban battlefield.

### Primum non nocere

Hippocrates said physicians must be able “to do good or to do no harm.”<sup>8</sup> But this can be seen as contradictory for the physician carrying a weapon with the

potential to take someone’s life. In truth, not every tactical physician member of a SWAT unit carries a firearm. Those who are sworn officers and carry firearms are bound by the oath of “protect and serve.” **With a comprehensive approach, the physician generally provides more to the team than just immediate medical care during callouts. They offer preventative care as the team physician and perform occupational health duties during long-term operations.** Kevin Gerold, DO, JD, MSED, FCCM, FCCP, described the tactical physician best, as a “hybrid of occupational health, emergency medicine, sports medicine, and [acting as] a health and safety officer.”<sup>9</sup>

### Not Just ATLS in the Field

One might assume the extensive training during residency and daily role of managing resuscitations and performing life-saving procedures would prepare the average emergency physician for a medical emergency in the tactical environment.<sup>10</sup> Rather, a different mindset and training is required to perform Tactical Combat Casualty Care (TCCC). TCCC was first introduced by the U.S. military’s special operations community in the 1990s as a set of guidelines for the treatment of injured operators in the battlefield. With revisions, the guidelines have become the new standard of care in pre-hospital battlefield medicine.<sup>11</sup> TCCC divides care into 3 phases based on the location relative to the threat.

#### 1. Care Under Fire

Taking place in the “hot zone,” this is essentially casualty care under fire. It is limited to tourniquet placement for hemorrhage control and extrication from burning vehicles or buildings.

#### 2. Tactical Field Care

Taking place in the “warm zone,” this represents care rendered while



not under direct fire. The mnemonic MARCH describes warm zone considerations: **M**assive hemorrhage, **A**irway, **R**espirations, **C**irculation, **H**ead injury). Interventions include wound management, application of hemostatic agents, intubation, needle decompression, fluid resuscitation, analgesia, and packaging the patient for evacuation.

### 3. Tactical Evacuation Care

Taking place in the “cold zone,” this involves all of the interventions necessary until definitive care is reached. This includes advanced monitoring, transfusion of blood products, placement of chest tubes, and ventilator management.

### Tactical Medical Assessment

The difference between ATLS and TCCC begins with the medical assessment of the patient. The mnemonic X-ABC (e**X**sanguinate, **A**irway, **B**reathing, **C**irculation) replaces the mnemonic of ABC used in ATLS. X, which can have many meanings, typically takes place during care under fire.

### The X Factor

The first step can often mean eliminating the target, designated “X.” This would occur if the tactical medic is operating on the front line in the hot zone. Before any medical assessment can be performed, all threats must be eliminated. Alternatively, another TMP or SWAT member can protect the position and neutralize any incoming threats while the provider delivers tactical emergency medical care to the patient. In some situations, eliminating the target could mean to simply hold the position and continue the firefight, as the goal is to minimize the number of casualties.

The second step continues with abolishing any potential indirect threats before providing tactical medical care. This means to decrease any possible threats from other operators, including their distraction devices, pepper spray, smoke and gas grenades, and weapons. The confused operator may think the medical provider is a hostile entity and act against him or her. This also means to decrease any risk of secondary explosion, including risk of injury from hazardous materials after an explosion. Finally, any risks from

seemingly innocent individuals may be mitigated by performing a thorough search involving a metal detector.

The third step is reaching the patient, and the fourth step is finally evaluating for any life-threatening exsanguination.

Remember, the priorities for a tactical provider are:<sup>12</sup>

1. Return fire as directed or required
2. Avoid becoming a patient
3. Keep the patient from sustaining additional wounds
4. Stop any life-threatening external hemorrhage with a tourniquet
5. Complete the mission

Once threats are stopped and the patient is behind cover, the provider can continue assessing X-ABC. Re-assess for any external extremity bleeding by applying a tourniquet or compression bandage and perform a rapid scan of major external bleeding. If the patient is unconscious, sweep from head to toe, stopping periodically to look for blood.

### ABC

Evaluate for patency of the airway and respiratory compromise. If necessary, facilitate efforts with airway adjuncts or provide an advanced airway. If signs of shock are present, obtain rapid IV or IO access for fluids or blood products. If extraction to a point of safety is possible, life-saving care can efficiently be provided there.

### Operating in a Hostile Environment

Imagine adding a level of intensity, now performing the initial assessment in a low-lit environment. Most callouts occur at night or in complete darkness, as light is avoided to prevent enemies from knowing the operators' location. Now add another level of complexity including smoke, flashing lights, and gunfire.

There are 2 important patient assessment skills that most training programs include. These are sensory-deprived physical assessment (SDPA) and sensory-overload physical assessment (SOPA). In SDPA, operators are blindfolded and required to perform a complete primary and secondary exam using senses other than vision. Typically, these assessments can be performed on full-scale human patient simulators in military settings or portable SimMan in

civilian settings.<sup>13</sup> In SOPA, distractions are simulated to overload the visual and auditory senses using smoke from dry ice, strobe lights, loud music, gunfire, and explosions via an audio system. The reality is that operators will need to perform an assessment in the dark, in a prone position, with weapons firing, while communicating with incident commander. All the while, the operator must be vigilant with a 360-degree sphere of awareness of incoming threats. This emphasizes the importance of proper training for TEMS.

The tactical provider may also render care from a distance by either direct visualization of the patient or by phone communication. This is known as remote assessment medicine. With the use of binoculars or a rifle scope, the provider may assess situations and communicate with the incident commander to determine if immediate rescue is necessary based on signs of life or injury.<sup>2</sup>

The role of the tactical physician can vary, but it almost always involves preventive medicine, medical intelligence, and operational assistance. These skills make the physician an indispensable part of the team.<sup>14</sup>

### A Look into the Future

The Committee for Tactical Emergency Casualty Care (C-TECC), comprising a broad range of operational and academic leaders in multidisciplinary prehospital medicine, was formed in May 2011 to create TECC guidelines. Modeled after TCCC, this guide translates military lessons into the civilian high-threat, prehospital community with an emphasis on terminology, trauma care recommendations, and operations. C-TECC accounts for differences in the civilian environment, resource allocation, patient population, and scope of practice.<sup>15</sup>

### Learn More

- Join the ACEP Tactical Emergency Medicine Section at [acep.org/tacticalsection](http://acep.org/tacticalsection).
- Read *Tactical Medicine Essentials* by Campbell JE, Wipfler EJ, Heiskell LE.
- Take FEMA courses online.
- Consider taking the Tactical Combat Casualty Care course.
- Visit [c-tecc.org](http://c-tecc.org).
- Contact a local fire/EMS or police medical director to get involved. ★



# COMING IN HOT

## Helicopter EMS Safety



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**C**hances are you have seen a story in the media about a medical helicopter crash, often with fatalities to the crew and patient. But helicopter EMS (HEMS) is less dangerous than often portrayed, and safety is continuously being improved at all levels from the individual, aeromedical agencies, and national regulatory bodies.

The first use of HEMS dates back to World War II for aeromedical evacuation in Burma. HEMS use became routine during the Korean War and is credited with drastically reducing combat-related mortality.<sup>1</sup> These findings prompted the adoption of HEMS into the civilian world, and its use has since evolved to hold an important role in both scene response and interfacility transport, with an estimated 400,000 medical patients flown each year in the United States.<sup>2</sup>

Even with the frequent use of HEMS, there is still no universal standard practice regarding their utilization.

Providers often request HEMS out of the assumption that it will decrease out-of-hospital time and lead to better outcomes. Some of this misconception may come from the historical 60-minute threshold known as the “golden hour,” which was previously accepted in EMS and EM practice but has since faded as a standard of care. Some literature indicates that aeromedical transport improves morbidity and mortality in critically ill medical and trauma patients; however, conflicting studies show equivalent or poorer outcomes when compared to ground-based EMS (GEMS).<sup>3-7</sup>

When requesting HEMS, transport time should always be considered. A major benefit of HEMS is decreased travel time, since helicopters can fly a direct path and exceed the speed of GEMS. However, some aspects of HEMS take more time, which increases the total prehospital interval.<sup>6</sup> Requesting a helicopter requires several steps, including a weather evaluation, flight planning, the crew being alerted, a pre-flight check, lift off, flight to the scene, landing, shutting down the aircraft, handoff, take off, return flight, landing, unloading, and possibly ground transport from the helipad to receiving facility. In many situations, GEMS units are more readily available and can complete the transport in a shorter period of time. Some of these delays can be mitigated by preplanning landing zones and flight

paths, or avoided by standby activations for prolonged extrications or austere environment rescues. In these instances, HEMS response may be beneficial as time delays incurred by air transport occur before the patient is ready for transport. Critical patients can also be brought to the local ED for initial resuscitation with HEMS being dispatched to that ED to transfer to a tertiary center after the patient has been stabilized. This limits scene landings, which are higher risk than landing at an established hospital landing pad.

HEMS often provides a higher level of care, especially in rural areas that may have only basic life support GEMS or rely on volunteers with variable levels of training. HEMS typically operates as a nurse/paramedic team, may carry blood products, and have a scope of practice beyond GEMS advanced life support. This may include surgical airways, chest tubes, a wider range of medication, and the ability to care for patients requiring balloon pumps or ECMO.<sup>8-10</sup> This expanded scope of practice may provide benefit for critically ill patients requiring



## Advances in technology are being incorporated to improve HEMS safety.

interfacility transport; however, these same benefits may be obtained

with lower risk using ground-based critical care transport teams.

Launching a helicopter involves a complex evaluation of risk and benefit. HEMS is both expensive, with each air medical transport costing between \$12,000 and \$24,000, and dangerous for patients and providers.<sup>11</sup> It is impossible to remove all hazards from HEMS, but significant progress has been made over the past 5 years. The Federal Aviation Administration reported that HEMS accidents have been steadily decreasing since 2013, with 106 accidents and seventeen fatalities in 2016.<sup>12</sup> This reduction is attributed to improved safety procedures, technological advances, and increased regulations. After increasing rates of aeromedical crashes in 2008 and 2009 with a total of 125 deaths in 20 years, the FAA revised its policies, releasing Final Rule 2010-0982 in February 2014. It mandates HEMS agencies to adopt enhanced procedures for flying in difficult weather, at night, and in remote terrain and required minimum standards for equipment including radio altimeters and terrain avoidance warning systems. The Final

Rule increased training requirements for both pilots and crew members.<sup>13</sup>

Many HEMS agencies have individual policies in place to improve the safety of their crews and patients. A procedure used by many is “blinding,” where calls are dispatched to the crew without the nature of the call being given. This eliminates emotion as a motivator to accept a mission in conditions the crew would otherwise deem unsafe. When these details are relayed, many agencies continue to keep the pilot “blinded” in order to maintain objective operation of the aircraft. “All-or-none” policies, in which every member of the flight crew must individually agree to fly before the aircraft takes off, are also popular.<sup>14</sup> This allows for the entire crew to evaluate the risk without fear of peer pressure or repercussion. Other policies attempt to combat “helicopter shopping,” where providers may contact a competing HEMS agency if declined by the in first agency due to weather or safety concerns.<sup>15</sup> This is especially a concern among for-profit HEMS agencies, which have an additional pressure to increase helicopter utilization.<sup>16,17</sup>

Advances in technology are being incorporated to improve HEMS safety. Many HEMS agencies are now capable, meaning that the pilots are certified to operate the aircraft in low visibility situations with just the use of GPS and

other instruments for navigation.<sup>16</sup> Night Vision Imaging System, which consists of night vision goggles and lighting systems, improves the safety in operations, both in limited lighting and nighttime take-offs and landings.<sup>18</sup> Wire Strike Protection Systems, known as “wire cutters,” have been implemented into civilian HEMS because 16% of fatal helicopter collisions involve a striking a telephone or power line, primarily during the descent to a non-established landing zone. Wire cutters, the specialized razor blades attached to the top and bottom of the aircraft, sever any wires the aircraft may contact. Studies by the U.S. Army have shown that this simple and inexpensive solution decreases crashes and mortality.<sup>19</sup>

Policy changes and technological improvement have decreased the risk of helicopter crashes – yet the risk can never be eliminated. Crew members wear a helmet and 5-point harness and sit in energy absorbing seats. Unfortunately, even with advancements in safety equipment, helicopter crashes still carry a high rate of mortality, especially for patients, who are more exposed.

Providers can help improve HEMS safety. Ensuring a safe landing zone (LZ) is paramount to an efficient and safe transfer of care. Most helicopters require a 100x100 ft flat area to land, with special attention to overhanging trees or electrical lines. Clear the area of any debris that may become airborne with the wind forces created during the landing. If the area is overly dry, it can be quickly wet down by fire services. In a fire or hazardous materials incident, the LZ should be upwind to prevent possible exposure. Ground crews should remain in contact with helicopter during approach and landing.

HEMS will never be risk-free, but great strides have been made to improve HEMS safety. Each day, more than 1,000 HEMS missions are flown, usually without incident. With continued advances, further research, and evidence-based guidelines for utilization, aeromedical transport will continue to improve medical care and transport. ★





# Tide Pod Challenge

## A Closer Look at the Toxicology

**Aaron S. Frey, DO**

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It seems like every few months a new challenge becomes a viral sensation on social media. Recently, teenagers and young adults have been ingesting laundry detergent pods in the aptly named Tide Pod challenge. Subsequently, the upsurge of intentional exposures has drawn national attention to the toxicological properties of laundry pods—3 and to their potentially lethal effects.

### What are Laundry Detergent Pods (LDPs)?

Although available in Europe since 2001, manufacturers began marketing LDPs in the United States in 2011.<sup>1-4</sup> They are single-use, concentrated liquid detergent packs housed in a water soluble wrapping.<sup>2,3,5,6</sup> Examples include Tide Pods, Purex Ultra Packs, and All Might Packs.<sup>6-7</sup> The exact composition of the LDPs varies by manufacturer, but most contain ethoxylated alcohols and propylene glycol in an alkaline, highly viscous liquid.<sup>2,3,5,7,8,9,10</sup>

### How common are exposures?

Despite the recent media attention and the ongoing social media challenge,

the problem of ingesting LDPs is hardly new, and unfortunately, becoming more common.

The majority of exposures occur in children younger than 6 years old.<sup>11-12</sup> In October 2012, the Centers for Disease Control & Prevention's *Morbidity and Mortality Weekly Report* tracked 485 LDP exposures from May 17 – June 17, 2012. Of these, 454 were in children younger than 5 years old.<sup>12</sup> From 2012-2013, the American Association of Poison Control Centers' (AAPCC) National Poison Data System (NPDS) recorded 17,230 exposures to LDPs in children younger than 6 years old.<sup>13</sup> One year later, there were 22,064 exposures in the same age bracket.<sup>14</sup> In the month of January 2018, the AAPCC recorded 606 exposures in children less than 5 years old.<sup>15</sup>

Of course, exposures in teenagers has been increasing as well. In January 2018, there were 143 teenage exposure reported by the AAPCC. For comparison, in 2017 there were 53 teenage exposures, and only 39 in 2016.<sup>16</sup>

### What are the Routes of Exposures?

The most common route of exposure is ingestion.<sup>4,6,11-14,17</sup> Other routes include ocular, dermal, and aspiration, while some exposures occur via multiple routes.<sup>13,14,17</sup>

### What are the Clinical Effects?

Exposures to LDPs cause more severe effects than those from traditionally packaged laundry detergent.<sup>2,3,6,12,18</sup> At least one death has been caused by LDP exposure.<sup>19</sup> Since the majority of exposures occur via ingestion, the most common effects are nausea, vomiting, and diarrhea.<sup>3,4,6,7,11,13,14</sup> Dysphagia secondary to oropharyngeal and esophageal mucosal burns and ulcers has also been reported.<sup>1,3,7,13,14,18</sup> Many cases include sudden and unexpected changes in mental status, which required intubation for airway protection.<sup>1-3,5,11,13-14,18,20-21</sup> Some patients may experience seizures,<sup>11,14</sup> and the respiratory system may experience effects such as coughing, wheezing and bronchospasm, aspiration pneumonitis, and edema of the epiglottic and aryepiglottic folds.<sup>1-3,5,11,13-14,18,22</sup>

Additionally, hypoventilation and apnea can occur.<sup>3,13-14</sup> Renal and electrolyte abnormalities in the forms of acute kidney injury, metabolic acidosis, hyperlactatemia, and hyperglycemia may arise.<sup>1-3,5,13-14,18,20-22</sup> Ocular exposures often cause chemical conjunctivitis and corneal abrasions,<sup>3-4,7,13-14,23-26</sup> while dermal contact causes irritation, rash, and chemical burns.<sup>4,6,13,14,22</sup>



## What is the Mechanism of Poisoning?

The mechanism of poisoning is currently unknown, but several theories exist. Some propose that the altered mental status, metabolic acidosis, and hyperlactatemia are caused by the propylene glycol and its metabolism into lactate, acetate, and pyruvate.<sup>2,3,5,18,22</sup> Others, however, argue that the concentration of propylene glycol in the detergent packs is not high enough to cause these effects.<sup>2,7,18</sup> Instead, they attribute these effects, particularly the severe mental status changes and seizures, to the ethoxylated alcohols in the LDPs.<sup>2,3,5,18,22</sup> As for the ocular, dermal, gastric, and airway burns and irritation, many postulate that the viscous, alkalotic properties of the liquid are to blame, but some suspect the concentration of the chemicals, not the pH, is responsible.<sup>2,3,5,15,23</sup>

## What is the Treatment?

There is no antidote, and the mainstay of care is symptomatic management.<sup>22,27</sup> Fortunately, the majority of cases are mild and can be safely discharged after a period of observation.<sup>7</sup> The manufacturers often recommend drinking a glass of water to dilute their product if ingested, but several poison control centers suggest using small sips of water due to fear of aspiration.<sup>2,22</sup>

The potential for rapid and unexpected change in mental status and its potential to cause airway compromise should prompt heightened vigilance and frequent reassessments.<sup>1,2,11,27</sup> All patients should undergo continuous pulse oximetry, and end-tidal CO<sub>2</sub> monitoring also may be beneficial.<sup>27</sup> Physicians should be prepared to rapidly intubate. Airway edema may respond to steroids and nebulized epinephrine.<sup>1,7,27</sup> Nebulized beta-2 agonists may improve bronchospasm.<sup>3,27</sup> Patients should be positioned to minimize the risk of aspiration, and anti-emetics should be considered. All ocular exposures should be copiously irrigated and undergo slit lamp examination to assess for corneal ulcers and abrasions.<sup>3,17</sup> Dermal exposures should likewise be irrigated and assessed for chemical burns once contaminated clothes are removed.<sup>15,22</sup> Additionally, toxicology or poison control should be consulted.<sup>22</sup> ★

References available online.

# Toxic YouTube Challenges

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Since its inception in 2005, YouTube has served as a platform for individuals to create and share video content online. Amid innocent videos of cats playing piano, however, a startling trend has emerged. In recent years, posters — often adolescents — have performed a stunt and then dared their friends to do the same. Some, like the ALS Ice Bucket Challenge, have been used to raise money and awareness for important causes. But more often they have devolved into viral dares ranging from silly to outright dangerous. The following are a few particularly toxic dares with consequences that emergency physicians need to know.

## Tide Pod Challenge

Concentrated laundry pods provide convenience to consumers, but the flashy, candy-like appearance of many of these products has arguably led to accidental ingestions and fatalities in pediatric and demented elderly populations.

For reasons unbeknownst to most high-functioning adults, the Tide Pod challenge has emerged in the past year. Posters intentionally place laundry pods in their mouth and attempt to hold it in place as drooling, foaming, and vomiting eventually ensue. (For more on the toxicological implications, please see the accompanying article.)

Tide recently released a PSA in which New England Patriots tight end Rob Gronkowski warns against ingesting Tide pods. As with many things in life, if Rob Gronkowski thinks something is a bad idea, it probably is.

## Cinnamon Challenge

The Cinnamon Challenge involves participants trying (often unsuccessfully) to ingest a tablespoon of ground cinnamon. The irritating, fine particulate substance commonly leads to coughing, choking, and vomiting, making this dare a high aspiration risk. While human data is limited, in rat models aspiration of cinnamon leads to inflammatory changes and ultimately fibrotic changes in the lungs.<sup>1</sup> Most exposures are mild and only require oral hydration and observation, but underlying asthma or cinnamon allergy are risk factors that could lead to dire consequences for this easily avoidable exposure.<sup>2</sup>

## Ghost/Hot Pepper Challenge

The Ghost/Hot Pepper Challenge involves individuals eating one or multiple peppers with a high capsaicinoid content, such as the Ghost Pepper, Carolina Reaper, or Trinidad Scorpion. These peppers have Scoville Heat Units (SHU) in the millions, making them more than a thousand times more potent than your standard jalapeno. While the predominant result is schadenfreude, a few cases have resulted in serious mortality, including a report of one individual developing Boerhaave syndrome and subsequent pneumothorax.<sup>3</sup>

## Soy Sauce Challenge

While not as viral as many of these challenges, high volume soy sauce ingestion have led to at least one interesting case presentation. On YouTube, this challenge involves posters taking shots of soy sauce and then attempting to hold back the sometimes-inevitable emesis. In 2013, a Virginia student was brought to the ED following ingestion of a quart-sized bottle of soy sauce and subsequent seizure.<sup>4</sup> This patient had the highest recorded corrected sodium of 196 mmol/L with survival to discharge. While the outcome was ultimately positive, he required high-level ICU care and heroic measures because of this ingestion.

YouTube and social media sites like Facebook are now taking steps to stem the tide of increasingly dangerous challenges.<sup>5</sup> By watching and sharing such videos, individuals are culpable in the injury of the vulnerable and susceptible adolescent population.

In the case any accidental or purposeful ingestion or exposure, **please call the poison center hotline at 1-800-222-1222** to report any cases encountered in the emergency department. ★



IMAGES COURTESY OF JEFFREY LAZAR, MD

# 3-D

## Printed Emergency Medicine Training

**Yash Chavda, DO**

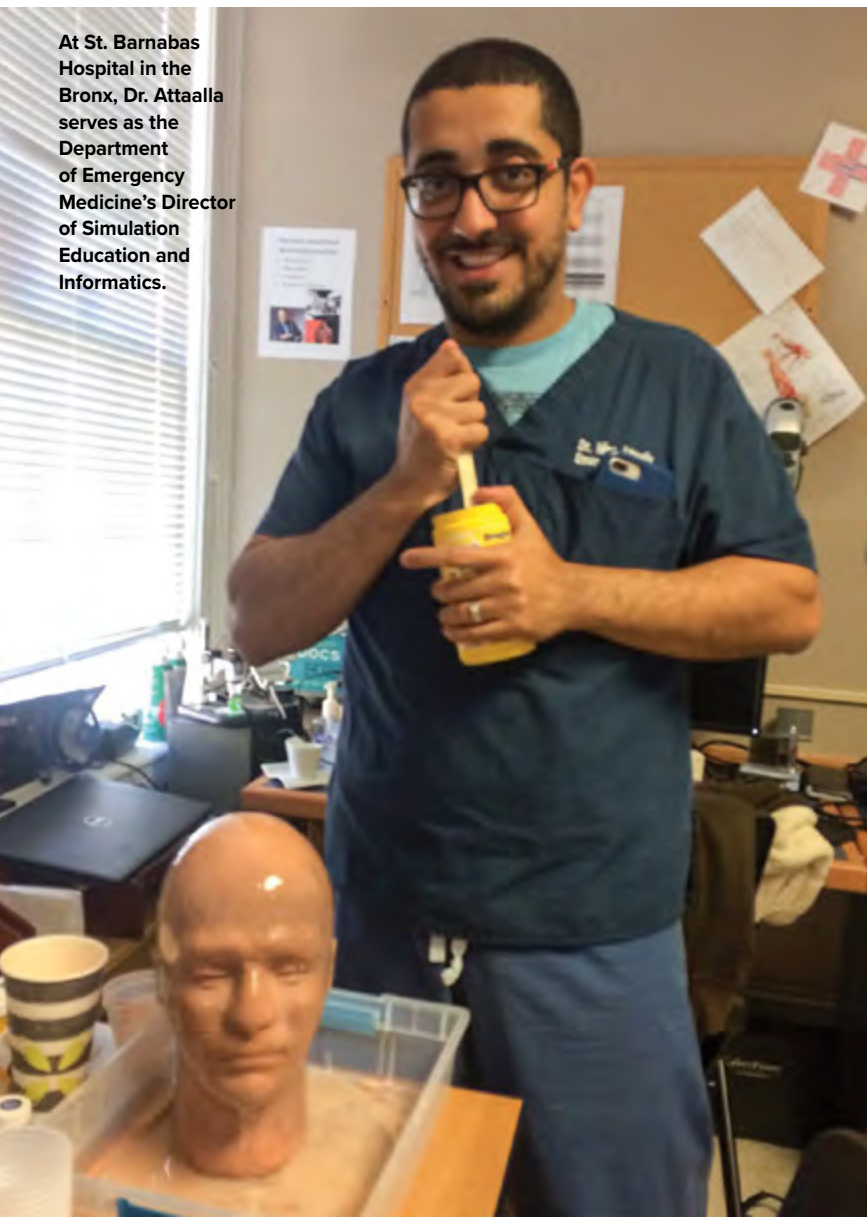
*St. Barnabas Hospital*

In the academic offices of Mina Attaalla, DO, and Jeffrey Lazar, MD, machines whir, gears turn, and belts track along their X, Y, and Z axes, often running for hours or days before finishing their product... which today is a 3-D printed cricothyrotomy trainer. Over time, more of these devices appear. Soon, the tracheas are implanted into foam heads, layered with realistically tinted artificial skin – and voila: an incredibly realistic human neck.

At St. Barnabas Hospital in the Bronx, Dr. Attaalla serves as the Department of Emergency Medicine's Director of Simulation Education and Informatics. He is spearheading the use of 3-D printing as means to create training devices that can improve the procedural techniques of St. Barnabas' Emergency Medicine Residents. He first became interested in 3-D printing as a result of his obsession with all things technological. After watching a YouTube video of someone 3-D printing a skull, he asked himself if the same technology could be used to teach residents.

Dr. Lazar, Vice Chair and Medical Director of Emergency Medicine, who was already a bit of a maker and an artist with experience in wood and stone carving, metal sculpture, laser and electronic cutting, was introduced to the world of 3-D printing by Dr. Attaalla. Together, they have already purchased several 3-D printers and are making exciting use of them in the field of medical education.

At St. Barnabas Hospital in the Bronx, Dr. Attaalla serves as the Department of Emergency Medicine's Director of Simulation Education and Informatics.





Cricothyrotomy trainers are just the beginning. Dr. Attaalla has produced a design for cutting out a piece of a simulation mannequin's leg and placing a 3-D printed tibia inside, overlaid with artificial skin to develop an IO trainer. "We are working on logistics of printing ED thoracotomy and thoracostomy models, as well as joint reduction, IO placement, and possibly for our surgical resident colleagues a laparoscopic playground simulation," Dr. Attaalla said. Dr. Lazar added, "We are looking into bone and joint creation to study and simulate the management of fractures and dislocations, and we are fine-tuning our simulated spine for lumbar puncture training." Considering the cost of pre-built training devices, often hundreds of dollars each, 3-D printing offers a significant cost-saving approach to training.

For those who are interested, entry-level printers can be purchased for as little as \$250, with filament (the actual printing material) costing as

little as \$15 dollars per spool. But there is a learning curve and an ongoing investment of time, Dr. Lazar warns: "3-D printers are still at a stage where they seem to require some technological aptitude, and the interest/dedication of a hobbyist." Troubleshooting 3-D printers definitely requires research and commitment.

Despite the current challenges, early adopters in the medical sim world will not find themselves alone, Dr. Attaalla said. "One of the other reasons I love 3-D printing so much is the wealth of information available for anyone who wants to start doing this now! This

**Despite the current challenges, early adopters in the medical sim world will not find themselves alone.**

includes groups on Facebook, Github, and forum-based sites that help you get started troubleshooting and perfecting the art of 3-D printing."

Outside of medicine, these devices can be used to make a variety of fun objects. As Dr. Lazar states, "I've used 3-D printing for some sculptural projects, including creating molecular models of toxicologic agents, a model of a neuron, and I also recently printed a chimpanzee skull."

Ultimately, the cricothyrotomy trainers were used during the residency program's Airway Day to teach a variety of cricothyrotomy techniques. They were well-received by the residents, who said they felt more comfortable with the mechanics of the procedure afterward.

While the airway training is valuable, it is by no means the only application for 3-D printing in medical simulation and education. To quote Dr. Attaalla, "The sky's the limit! If you can dream it, with time we can probably model and build it." ★



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# ECG Explained

In our February/March edition, we issued a challenge to interpret this unusual EKG tracing.

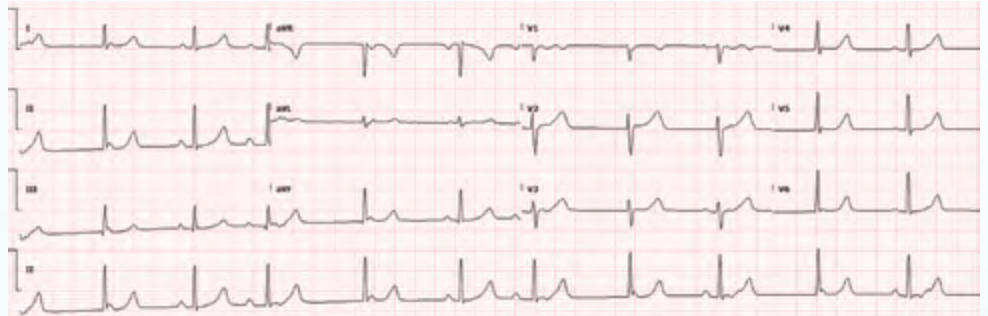
In this edition, learn how the experts interpreted it, and pick up pointers for future cases.

**Jeremy Berberian, MD**  
EMRA EKG Guide Editor-in-Chief  
Christiana Care Health System

This ECG demonstrates a 2<sup>nd</sup> degree type I AV block (Mobitz I) with intermittent junctional beats, non-conducting P-waves, and retrograde P-waves.

Using the Lead II rhythm strip as a guide (see below), here's the pattern:

1. Junctional beat (narrow QRS with no preceding P-wave) followed immediately by a non-conducting sinus P-wave (↓)
2. Sinus beat
3. Sinus beat with longer PR (= Mobitz I) followed by a retrograde P-wave (↓) which resets the atrial conduction system (interrupting Mobitz I)
4. Junctional beat (narrow QRS with no preceding P-wave) followed immediately by a non-conducting sinus P-wave (↓)
5. Atrial conduction system resets and resumes pacing = sinus beat
6. Sinus beat with longer PR (= Mobitz I) followed by a retrograde P-wave (↓) which resets the atrial conduction system (interrupting Mobitz I)
7. Junctional beat (narrow QRS with no preceding P-wave) followed immediately by a non-conducting sinus P-wave (↓)
8. Sinus beat followed by a retrograde P-wave (↓) which resets the atrial conduction system
9. Junctional beat (narrow QRS with



no preceding P-wave) followed immediately by a non-conducting sinus P-wave (↓)

10. Sinus beat with retrograde P-wave (↓)

### More on the retrograde P-waves (↓)

These P-waves are inverted in the inferior leads, which is expected for retrograde conduction. They reset the atrial conduction system, interrupting Mobitz I, so there isn't the expected dropped QRS after a P-wave. Most likely, they conduct via the "retrograde only" accessory pathway since the AV node will be refractory to retrograde conduction that quickly following the QRS. Alternately, they conduct retrograde over a dual AV nodal pathway within the AV node. This is referred to as an echo beat or reciprocal beat, and typically follows a QRS preceded by a prolonged PR interval.

### More on the non-conducting P-waves (↓)

After any beat with a retrograde P-wave, the atrial conduction system needs time to reset before it resumes pacing. During this time a junctional focus competes with the SA node. The junctional focus occurs just before the SA node impulse, so these P-waves are non-conducting because the rest of the conduction system has already depolarized from the junctional focus. These P-waves are sometimes entirely seen (after beat 4) and sometimes

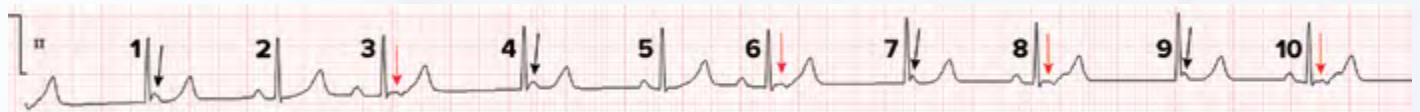
only partially seen (after beat 7) due to the close timing of the SA node in comparison to the junctional focus.

These P-waves look like the other sinus P-waves, which is expected if they are all initiated in the SA node.

### Ladder Diagrams

A ladder diagram uses lines with arrows and bars to illustrate the electrical activity within the cardiac conduction system that may not be evident on the surface EKG (ie, concealed conduction). It is constructed of 3 tiers that represent the atria, AV junction, and ventricle. The top horizontal line represents SA node, the 2nd horizontal line represents the beginning of AV conduction, and the 3rd horizontal line represents the end of AV conduction and the start of ventricular conduction. Solid circles represent the start of any impulse. Circles on the top line represent impulses originating from the SA node. Circles in the middle of the AV tier represent impulses originating in or around the AV node. Arrows indicate the direction of conduction and horizontal bars indicate a conduction block. Solid lines represent conduction via the fast pathway and dashed lines represent conduction via the slow pathway (or the accessory pathway in our case).

Read the description of the pattern (#1-10) while you work through this laddergram.





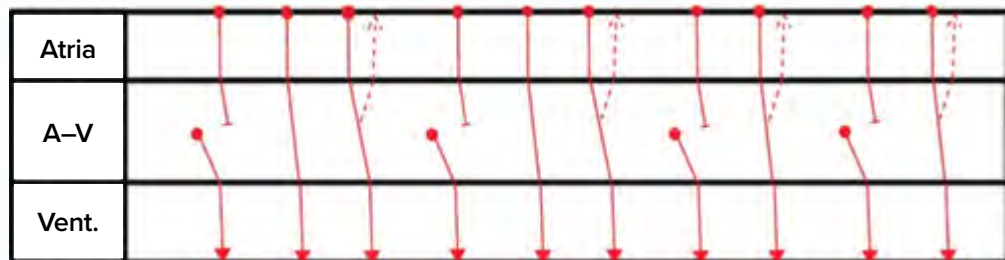
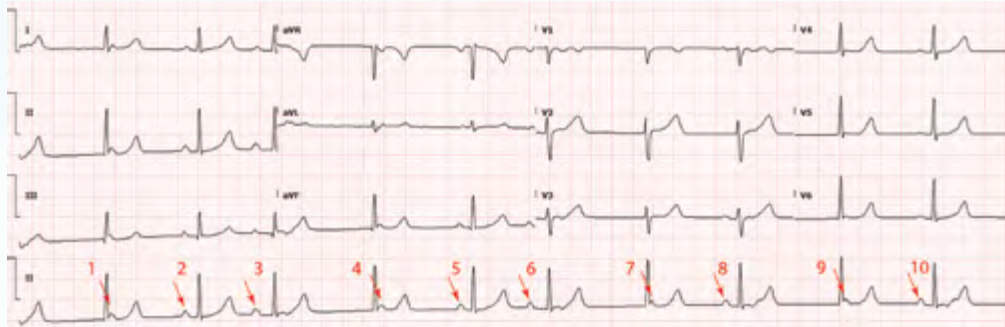
## CASE COMMENTARY

Ken Grauer, MD

This challenging case highlights the point that more than a single interpretation may be possible for a complex arrhythmia — and that sometimes, a definitive answer will only be forthcoming after EP study. We emphasize these principles:

- Awareness of the history may shed light on the probable mechanism of an arrhythmia. The patient in this case, a 19-year-old woman with a history of recurrent SVT episodes, was found on EP study to have an accessory pathway that was felt to participate in sustaining her arrhythmias.
- A laddergram clarifies our theory for the mechanism of this arrhythmia.
- Use calipers! This is the fastest way to enhance your ability to interpret complex arrhythmias — because it instantly allows you to assess intervals and regularity of the atrial and ventricular rate.
- When there are a number of elements in a given arrhythmia, it is helpful to assess the “easier-to-interpret” elements first.
- Try to determine if there is an underlying rhythm, such as sinus rhythm or atrial fibrillation. It is often much easier to figure out more complex elements after you have recognized the underlying rhythm.
- “Step back” to view the entire tracing before diving in to the details. Doing so may prove invaluable for recognizing certain phenomena such as “group beating.”
- Label sinus P-waves with arrows. This simple step will help you recognize which P-waves are probably conducting and which are not.
- Number the beats in order to intelligently discuss a complex rhythm with others.
- When an AV block is present in which some P-waves are conducted but others are

Read the description of the pattern (#1-10) while you work through this laddergram.



not, a wonderful clue that a P-wave is probably conducting is if a beat occurs significantly earlier than expected.

Applying these principles to this tracing, begin by stepping back! You should notice the pattern of group beating: The appearance and interval duration for beats #1,2,3 and #4,5,6 are virtually identical. The same is true for beats #7,8 and #9,10. This is not by chance.

- The upright P-waves in the long lead II rhythm strip that are highlighted by red arrows are all sinus P-waves. The P-P interval shows no more than minimal variation except for the 3 relatively longer pauses (ie, between beats #3-4; 6-7; and 8-9). This indicates the underlying rhythm is sinus arrhythmia and that *something else* is probably accounting for these 3 much longer pauses. Looking for a cause of these pauses, we see the negative notch in the early part of the ST segment of beats #3, 6, 8, and 10 reveals the negative notch that indicates hidden atrial activity.
- There is no P-wave preceding beats #1, 4, 7, and 9. Since these QRS complexes look the same as all other QRS complexes, these 4 beats must be junctional escape beats. In support of this theory is the finding that the R-R intervals preceding beats #4, 7, and 9 are both equal and *longer* than the R-R interval preceding all other beats.
- Using calipers, it should now be easy

to see that beats #2,3; 5,6; 8 and 10 are all preceded by an R-R interval that is *shorter* than the R-R interval preceding the junctional escape beats. This suggests all of these beats are probably conducted. The normal and equal PR intervals preceding beats #2, 5, 8, and 10 supports this theory. The fact that the PR interval preceding beats #3 and 6 is both equal and longer tells us there is progressive lengthening of the PR interval for conducted P waves within the first 2 groups — and that is the definition of Wenckebach!

**Note:** We cannot rule out the possibility that the negative deflections in the early part of the ST segment for beats #3, 6, 8, and 10 are PACs. Even if we knew these negative deflections represent retrograde atrial activity, we cannot say for certain from this single ECG that conduction is over the AP instead of a dual AV nodal pathway. That said, awareness of this patient’s history (ie, knowing she has recurrent SVT episodes with an AP) and knowing that retrograde conduction is facilitated by progressive preceding PR interval lengthening (which allows more time for retrograde conduction to occur) makes the mechanism illustrated in this laddergram the most plausible explanation. Retrograde conduction over an AP is a common substrate for facilitating orthodromic AVRT.

Dr. Grauer’s ECG Blog can be found at <http://ecg-interpretation.blogspot.com>. For a primer on laddergrams, see the entry at <http://tinyurl.com/KG-Blog-69>. ★

**FIGURE 1.**  
Dissection involving the ascending aorta and arch



**FIGURE 2.**  
Dissection involving the descending aorta



# The Near-Fatal Panic Attack

**Christina Long, MD**

Loyola University Medical Center

**I**t's 2 am in a busy Level I trauma center. The triage nurse brings back a 30-year-old female and states, "I think she is having a panic attack." The patient tells you she was awakened by her infant twins and noticed a Charley horse in the right side of her neck. She then developed substernal chest pain that was severe, constant, and radiated to her back. En route to the ED, she developed left upper extremity and right lower extremity numbness, as well as nausea with non-bloody emesis. While you are speaking to her, the pain migrates to her abdomen. On further history, she says she's healthy and had a hysterectomy earlier this year. She has been told she has a heart murmur. She does not smoke or use drugs. Her father has a history of an aortic aneurysm that has not required surgical repair. Her vitals are: Temp, 97.9 F; Pulse, 101; BP, 94/51; Respiratory, 24; SpO<sub>2</sub>, 100%. On exam, she appears very distressed, pale, diaphoretic, and is vomiting clear emesis into a basin. Cardiovascular exam reveals a grade III diastolic decrescendo murmur and intact distal pulses. She is neurologically intact. Her EKG shows sinus tachycardia without acute ischemic changes. Pertinent labs include a normal chemistry panel, normal hemoglobin and hematocrit, negative troponin, and d-dimer >6000 ng/mL. You obtain a STAT CT angiogram of her aorta.

## Pathophysiology

While aortic dissection is relatively rare, it remains the most common and lethal form of the acute aortic syndromes.<sup>1</sup> The incidence is reported to range from 2.6 to 3.5 per 100,000 person-years.<sup>2</sup> The

inciting event for a classic aortic dissection involves a tear in the intimal layer, followed by blood entering the medial layer, forming a "false lumen." From there, blood can propagate anterograde or retrograde, with varying catastrophic sequelae. Variants of aortic dissection include aortic intramural hematoma, intimal tear without hematoma, and penetrating atherosclerotic ulcer.<sup>2</sup> Mortality rates remain as high as 30%, with immediate mortality rate up to 1% per hour after symptom onset.<sup>1,2</sup>

Aortic dissection has been classified according to the Stanford and DeBakey systems. Stanford is more widely used; it classifies dissections into those that involve the ascending aorta as type A, and all others distal to the left subclavian artery as type B.<sup>1,3</sup> The DeBakey system classifies dissection based on the site of origin and is divided into types I, II, IIIa, and IIIb.<sup>1,3</sup>

## DeBakey classification:

- Type I involves the ascending aorta, arch, and descending thoracic aorta and may progress to involve the abdominal aorta.
- Type II involves the ascending aorta.
- Type IIIa involves the descending thoracic aorta distal to the left subclavian artery and proximal to the celiac artery.
- Type IIIb dissection involves the thoracic and abdominal aorta distal to the left subclavian artery.

## Stanford classification:

- Type A involves the ascending aorta and may progress to involve the arch and thoracoabdominal aorta.
- Type B involves the descending thoracic or thoracoabdominal aorta distal to the left subclavian artery without involvement of ascending aorta.

## Presentation

The most common symptom is acute pain, reported by approximately 85% of patients.<sup>3</sup> Type A dissections present more often with anterior chest pain, while more Type B dissections present with back or abdominal pain, though overlap exists.<sup>3</sup> The International Registry of Aortic Dissection says patients describe pain as sharp rather than ripping or tearing.<sup>3</sup>

Hypertension is present in Type B dissections (71%) more than Type A (36%).<sup>1,4</sup> Conversely, hypotension, syncope, and shock are more common in Type A dissections. Hypotension and shock accompanying acute aortic dissection can result from cardiac tamponade, aortic hemorrhage, acute MI, true lumen compression by false lumen, aortic rupture, severe aortic regurgitation resulting in heart failure, or intra-abdominal catastrophe.<sup>1,4</sup>

Peripheral pulse deficits are present in 15-31% of patients, more commonly in Type A dissections.<sup>3,4</sup> The classic "decrecendo diastolic murmur" of aortic regurgitation is found in approximately 40-50% of patients with Type A aortic dissection.<sup>3,5</sup>

It is estimated up to 40% of patients with aortic dissection present with acute neurologic findings, whether transient or permanent.<sup>6</sup> This can mimic a stroke, especially in patients who present without pain (up to 15%).<sup>6</sup> Neurologic manifestations can include altered mental status, TIA, stroke, encephalopathy, Horner's syndrome, seizure, spinal cord ischemia, ischemic neuropathy or plexopathy, and nerve compression syndrome.<sup>6</sup>

## Risk Factors

Risk factors for aortic dissection include conditions or mechanisms that result in weakening or degeneration of the aorta's media layer, or place extreme stress on



the aortic wall.<sup>4,5</sup> Hypertension is the most important predisposing factor.<sup>2</sup> Conditions that may transiently increase blood pressure, such as cocaine use, energy drinks, pheochromocytoma, and high-intensity weightlifting have also been associated with aortic dissection.<sup>1,2</sup> Advancing age and male sex are also important risk factors. According to the IRAD, the mean age of patients studied was 63 years, and approximately two-thirds of patients were male.<sup>3,4</sup>

Major inherited connective tissue disorders known to be associated with aortic dissection include Marfan's syndrome, Ehlers-Danlos syndrome, and Loeys-Dietz syndrome.<sup>4,5</sup> Turner's syndrome, familial thoracic aortic aneurysm and dissection syndrome, and annuloaortic ectasia are also important genetic conditions associated with increased risk.<sup>4,5</sup>

Structural cardiovascular abnormalities associated with dissection include bicuspid aortic valve and coarctation of the aorta. Bicuspid aortic valve is one of the most common types of congenital heart disease.<sup>7</sup> This variant predisposes the ascending aorta to aberrant, turbulent blood flow, and is often associated with underlying cystic medial degeneration.<sup>7</sup> Dissection occurs 5-10 times more commonly in patients with bicuspid valve compared to tricuspid valve, and affects a younger population.<sup>7</sup>

Other risk factors include inflammatory conditions that cause vasculitis, pregnancy and delivery, trauma, and iatrogenic factors.<sup>2,5</sup>

## Diagnosis

In addition to checking for pulse deficits, BP variations in the upper extremities (>20 mmHg), and a wide pulse pressure, an EKG and portable chest x-ray are quick bedside tests to evaluate for aortic dissection. According to the IRAD, EKG shows non-specific ST-segment or T-wave changes in 41%, no abnormalities in 31%, and ischemic changes in 15%.<sup>3</sup> The classic widened mediastinum (measuring >8 cm at the level of the aortic knob), was present in 63% of Type A dissections and 56% of Type B dissections.<sup>2,3</sup>

D-dimer levels have been suggested as a "rule-out" marker. However, given that it is elevated in other acute chest conditions

in the absence of dissection, it should be used to risk stratify patients when suspicion for the disease is low and reliance on the strong negative predictive value of the test is desired. In the IRAD study, d-dimer had a sensitivity of 97% and specificity of 47% in aortic dissection when using the cutoff of 500 ng/mL.<sup>8</sup> The negative predictive value was 95% when used in the first 24 hours of symptom onset.<sup>8</sup>

CT angiography is the gold standard and diagnostic modality of choice.<sup>2</sup> Consider transesophageal echocardiography in the hemodynamically unstable patient, with the disadvantage of requiring esophageal intubation and procedural sedation.<sup>2</sup> MR angiography can be used for stable patients but is not ideal in a crisis.<sup>2</sup>

## Treatment

Initial management aims to stabilize the patient. When surgical management is indicated, notify the surgeon immediately. Minimize or avoid IV fluids in the hypotensive patient. Perform bedside cardiac ultrasound to evaluate for pericardial tamponade, valvular, or left-ventricular dysfunction.<sup>9</sup> Hemodynamic instability, altered mental status, or airway compromise are indications for rapid-sequence intubation. An analgesic such as fentanyl (3 mcg/kg) should be given prior to induction to prevent the catecholamine release associated with intubation. Etomidate (0.3mg/kg) should be used as an induction agent for the patient who has known cardiovascular disease or is hemodynamically unstable.

Alternatively, in the hypertensive patient, use propofol (1.5 to 3 mg/kg), which suppresses catecholamine release (thereby reducing MAP). Use an arterial line to facilitate blood pressure management. Medical therapy is aimed at pain control and anti-impulse therapy. Morphine, hydromorphone, or fentanyl are acceptable analgesic agents. Anti-impulse therapy reduces left-ventricular contraction, thereby minimizing shear stress and propagation of the tear.<sup>9</sup> The preferred antihypertensive agents are beta-blockers. Esmolol has a short half-life and is easily titrated, making it a good choice. The bolus dose is 250-500 mcg, followed by 25-50 mcg/kg/minute. Labetalol is another option, given in sequential boluses or as a drip. An initial bolus can be given of 20 mg, followed by

40-80 mg every 10 minutes up to 300 mg total as needed. The dose for labetalol drip is 0.5-2 mg/minute. The target heart rate is <60 bpm, and systolic BP of 100-120 mmHg. If the target BP cannot be achieved, or there is a contraindication to beta-blockers, nitroprusside can be used — but heart rate must be controlled first, as it can activate the reflex sympathetic pathway.<sup>9</sup>

Acute aortic dissection involving the ascending aorta is a surgical emergency. Repair involves excising the intimal tear, obliterating the entry into the false lumen, using a synthetic interposition graft, and repair or replacement of the aortic valve.<sup>10</sup> Surgery aims to halt progression of disease, improve flow, prevent rupture, and improve valve function.<sup>11</sup>

Uncomplicated Type B dissections are managed medically. Given that most patients with Type B dissections are hypertensive, medical management is aimed at BP reduction, typically with beta-blockers.<sup>1</sup> Ongoing management for all aortic dissections focuses on anti-impulse therapy and serial imaging.<sup>10</sup> Screening for associated genetic conditions is also important.<sup>10</sup>

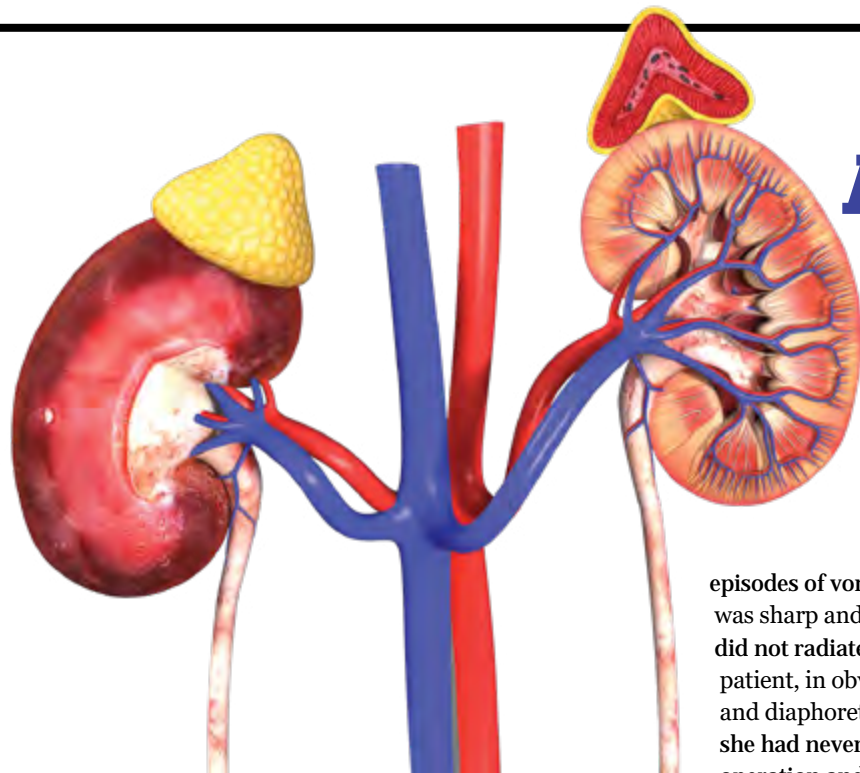
## Case Resolution

CT angiogram of the aorta revealed an extensive dissection of the non-dilated aorta, extending from the aortic root to the distal abdominal aortic bifurcation, into the bilateral common carotid arteries, as well as the bilateral proximal iliac arteries. This is consistent with a Type A dissection, a surgical emergency. Permissive hypotension was recommended to minimize expansion; she was given a 500cc NS fluid bolus, hydromorphone for pain, and antiemetics. During emergency surgery, she was found to have a bicuspid aortic valve. Surgery involved repair of the dissection with an ascending aortic supracoronary graft, suspension of the bicuspid aortic valve, and repair of the aortic root under deep hypothermic circulatory arrest using retrograde cerebral venous perfusion. Aortic valve replacement was not performed. She was discharged home 8 days later.

This case is notable because aortic dissection is rare in young, seemingly healthy, non-pregnant women, but underlying predisposing conditions (in this case, a bicuspid aortic valve) can be lurking. ★

# CASE STUDY

## Abdominal Pain and Unusual Catastrophic Diagnosis



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Spontaneous bilateral adrenal hemorrhage (SBAH) is a rare condition that results in adrenal crisis, shock, coma, and death. Unfortunately, symptoms are not easy to identify because they are non-specific and generalized (Dahan, *et al*, 263). In fact, the diagnosis often results from scans for symptoms correlated to other diseases. It is most commonly related to sepsis, physical trauma, anticoagulant therapy, coagulopathies, post-operative states, and severe stress. It can be associated with people of any age but is more common to people 40-80 years old. However, difficulty arises when there are no known specific symptoms to be associated with the underlying pathology for early diagnosis. High awareness and suspicion must be perpetuated in patients who are clinically ill under high risk factors.

### The Case

A 50-year-old Hispanic female presented to the ED at 10 am complaining of severe left upper quadrant and left flank pain since 2 am the night before. The pain was associated with nausea and 4

episodes of vomiting. Pain was sharp and stabbing and did not radiate anywhere. The patient, in obvious distress and diaphoretic, confirmed she had never had a surgical operation and was not taking any medications. Vital signs

upon presentation were temperature: Temperature, 97.4 F (36.3 C) oral; BP, 105/92; Pulse, 106; Respiratory, 28; MAP, 96; SpO<sub>2</sub>, 97%; Pain 1-10 scale, 10; Weight 64.3 kg.

While awaiting lab results, the patient complained of worsening pain in the same location, but this time a bit more generalized. Patient became hypoxic, stopped breathing, and suddenly became pulseless. The patient was intubated and CPR was initiated, with several rounds of epinephrine and bicarbonates. At the end of the third round of CPR, there was no pulse and there was a flat line on the monitor, with no spontaneous respirations and no heart tones.

When labs came back the only abnormality found was a platelet count of 13,000, indicating thrombocytopenia. CT scans were negative for any bleeds but did show perinephric fluid on the left side slightly greater than normal, suggesting possible inflammation (see Figure 1).

### Etiology

SBAH can occur because of anticoagulant therapy or an underlying coagulopathy. For example, heparin-induced thrombocytopenia (HIT) is a known cause and results from an antibody reaction by the patient's body against the platelets (Dahiya *et al*. 27). Bleeding

is rare — platelet counts do not fall below 20,000 normally — but can cause adrenal hemorrhaging because of presumed adrenal vein thrombosis. HIT can cause severe arterial and/or venous thrombosis of both adrenal glands and usually presents within the first 2 weeks of commencing anticoagulant therapy. However, in this case the patient had thrombocytopenia with a platelet count of less than 20,000 and there was no use of heparin.

The adrenal gland has a rich supply of blood but has poor drainage because it depends on only one vein. Additionally, stress can exacerbate the condition. A hormone called adrenocorticotropic hormone (ACTH) increases when an individual is stressed, causing vessels to dilate and leading to an increase in blood flow through the arteries, which in excess amounts cannot be compensated by the single draining vein. Coupling this with coagulopathies, the likelihood of SBAH is much higher.

SBAH is also attributed to a disease called Waterhouse–Friderichsen syndrome (WFS), which leads to the failure of the adrenal glands, also known as adrenal crisis, because of excessive bleeding into the gland itself (Hammond *et al*. 2,476). It was commonly associated with the pathogen *P. aeruginosa* in children with sepsis; however, the most common pathogens responsible for this disease now are *N. meningococcus* and *S. aureus*, especially in adults. This leads to meningococemia, which leads to the severe hemorrhaging of the glands.

SBAH can be a complication of other critical illnesses, such as shock or sepsis. Other known causes are physical trauma



and postoperative states. These stressful illnesses can mask SBAH, as the non-specific symptoms associated with it cannot be distinguished from the current illness.

### Signs and Symptoms

Adrenal hemorrhage has a number of generalized yet very dramatic symptoms that are seen with many different conditions — even when a single gland is affected. When both adrenal glands are affected, this disease tends to become life-threatening, especially if adrenal crisis occurs. Concern for SBAH should increase when considering the extent and duration of blood loss and the impact on adrenal gland function. The key is to provide proper therapy before symptoms of shock arise, because this can lead to coma or even death.

SBAH can be suspected through identifying symptoms like hypotension and shock greater than 90% of the time. Abdominal, back, and flank pain occurs in more than 86% of cases. Fever, nausea, and vomiting occur more than 50% of the time. In the cases including the abdomen, acute abdominal rigidity and rebound tenderness were reported in 15-20% of the cases.

Many times patients present with symptoms after more than 80% of the adrenal cortex has already suffered damage. Patients with high risk factors should raise suspicion that adrenal hemorrhage may already be occurring even

though they present with non-specific yet very dramatic symptoms. Consider these cases to be emergent, with prompt examination, proper labs, and CT scans.

### Management and Treatment

SBAH can be managed and treated when it's identified in the early stages. But when the diagnosis is delayed, the condition becomes very difficult to manage and treat, which may lead to patient death. If there is suspicion of adrenal hemorrhage, then empiric treatment with glucocorticoids should be started, as patients tend to deteriorate quickly.

The most common way of finding SBAH is by doing a CT scan as a quick form of imaging. The CT scan can differentiate a hematoma from other pathologies such as tumors related to the soft tissue glands. However, an early sign of SBAH associated with bleeding cannot always be seen clearly on CT scans. MRI can be used to detect acute, sub-acute, or chronic bleeding and would be able to detect early signs of bleeding, yet would not be of much help finding other pathologies such as neoplasms. Unfortunately, it takes longer to get results with an MRI, especially when the patient is unstable. Additionally, the suspicion for SBAH would be needed for the MRI to be ordered.

Treatment may include replacement of the body fluids as well as the electrolytes, along with restoration of deficient glucocorticoids and mineralocorticoids, which are normally secreted by the adrenal glands (Marti *et al*, 80). The glucocorticoid replacements are typically hydrocortisone, prednisone, and dexamethasone, while the mineralocorticoid replacement is fludrocortisone.

In some cases — such as severe bleeding secondary to trauma — management and treatment for SBAH may be surgical. In all cases, however, prompt attention is crucial, because a patient who goes into shock has a higher rate of mortality.

### Complications and Mortality

Complications of SBAH may result in a crisis like adrenal insufficiency. In cases where this occurs, sepsis or shock may follow, which can eventually lead to death. The key, as mentioned earlier, is diagnosing the illness in its early stages

by maintaining a strong suspicion for this condition.

The mortality rate in patients suffering from SBAH varies depending on the severity of the predisposing illness, but it has been estimated at roughly 15%. Because many patients present with symptoms after more than 80% of the adrenal cortex is already damaged, time for diagnosis becomes crucial. Autopsy studies show that SBAH incidence used to be about 0.14%-1.8% (Xarpy VP, 214). Prior to CT and MRI scans, this diagnosis was only made during autopsy. Thanks to improved imaging modality, the rate of autopsy findings for SBAH has declined tremendously. Now the rate of autopsy finding for SBAH is usually linked to misdiagnosis of this rare complication.

### Conclusion

SBAH rarely occurs, even in those with high risk factors, but when it happens, empirically starting treatment should be the first step in management. SBAH can be a fatal disease, and mortality increases when patients have prolonged symptoms. The non-specific yet increasingly exaggerated nature of the symptoms, however, make the condition hard to detect.

In this case, lab results showed thrombocytopenia with a platelet count of 13,000. Patient had a negative past medical history of other illnesses and was not on any medications. CT scan showed left perinephric fluid more than the typical amount, but did not indicate an acute bleed. An MRI is better at picking up acute bleeds and could have picked up the acute hemorrhage, yet a very high suspicion would have been needed. In this case study, the patient also had the only physical symptom of severe LUQ and left flank pain, making the assumption of SBAH an even higher possibility because of the presenting high risk factor of thrombocytopenia. Once the patient became hypotensive and symptoms of shock manifested, deterioration occurred rapidly. Those with high risk factors should have the necessary imaging and labs done in order to diagnose the illness promptly — yet in this case, even with the proper labs and scans done promptly, the diagnosis wasn't made in time to treat the patient. ★

**FIGURE 1. Left Perinephric Fluid** (slightly greater than typical could suggest inflammation)



SOURCE: LOYOLA UNIVERSITY HEALTH SYSTEM RADIOLOGY DEPARTMENT 04/2017; USED WITH PERMISSION

# Factors Affecting Standardized Video Interview Performance



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## Preparation Elements and the Testing Environment

### AAMC Writing Group

The AAMC launched the **Standardized Video Interview** (SVI) pilot project for medical students applying to emergency medicine residency programs in summer 2017. The SVI is an online, asynchronous interview designed to assess applicants' proficiency in two Accreditation Council for Graduate Medical Education (ACGME) competencies: *Interpersonal and Communication Skills* and *Knowledge of Professional Behavior*.<sup>1</sup> Questions are presented in text and applicants provide an audio/video response. The purpose of the SVI is to provide easy-to-use and reliable information about applicants' proficiency on the target competencies, balance emphasis on Step scores, and provide a new set of standardized evaluation criteria that may affect the

student population invited to in-person interviews.<sup>2</sup>

The addition of the SVI to the residency selection process has generated questions about added value to the selection process, how applicants should prepare, and whether the setting in which they complete the SVI influences their score. Answers to these questions are particularly important because they could be used to better inform all students about how to prepare, and address their concerns about the amount of time needed to prepare for the SVI.

### Methods

All EM applicants who submitted a completed SVI were invited to participate in two online surveys about their experience preparing for and taking the SVI. The first survey contained 11 questions and was administered



immediately following the SVI. The response rate was 83% (n = 2,906). The second survey contained 29 questions and was administered between October and November 2017. The response rate was 58% (n = 2,074).

Applicants' responses were linked to SVI scores for applicants who provided email addresses as part of the survey; only responses that could be linked to SVI scores were retained for analyses.<sup>3</sup>

Descriptive statistics, including counts, mean, and standard deviations were computed. Table 1 summarizes the sample by demographic group.

### Preparation Strategy

Applicants used a variety of strategies to prepare for the SVI. As shown in Figure 1, most participants reported reading AAMC's *Tips for Applicants* (79%), completing at least one practice question in advance (66%), and/or studying interview questions related to the target competencies (54%). Only 7% did not prepare in advance and their scores were significantly lower than applicants using any preparation strategy ( $t(1503) = -3.405, p = .001$ ). There were no practical differences in SVI scores by preparation strategy.

### Who Helped You Prepare

As shown in Figure 2, 76% of applicants prepared for the SVI on their own, 16% prepared with a medical school advisor, 15% prepared with classmates, and 8% did not prepare in advance. SVI scores did not differ based on who helped the applicant prepare for the SVI, but applicants who did not prepare for the SVI had significantly lower SVI scores ( $t(1503) = -3.582, p < .001$ ).

### Hours Spent Preparing for the SVI

As shown in Figure 3, most applicants spent some time preparing for the SVI. Forty-six percent of applicants spent 1-2 hours and 26% spent 3-4 hours preparing for the SVI. About eight percent did not prepare for the SVI. Applicants who did not prepare at all for the SVI had slightly lower scores than applicants who spent between 1-6 hours preparing ( $t(1419) = -4.138, p < .001$ ). Among those who prepared for the SVI, the amount of time spent preparing did not affect SVI scores.

### Taking the Interview

Because of the flexible nature of the SVI, applicants could record the interview at various locations. As shown in Figure 4, the majority of applicants completed the SVI at home (75%), whereas others completed it at a recording space arranged by their school (8%) or other locations like hotels or offices (17%). Location did not influence final SVI score, suggesting that applicants should complete the SVI in the location that is most convenient for them (and has a good internet connection).

### Key Take-Aways for

#### Those Taking the SVI in 2018

- Use the preparation methods you need to feel comfortable before taking the SVI, but more than a couple of hours of practicing does not seem to be necessary. SVI scores did not differ based on number of hours spent preparing for the SVI, nor did they differ by preparation strategy. However, students are still advised to read the AAMC's *Tips for Applicants* as a way to prepare for and familiarize themselves with the SVI.
- Take the SVI at whatever location is convenient for you, as long as there is a good internet connection.

**FIGURE 1. Preparation Strategy by Usage and SVI Score**

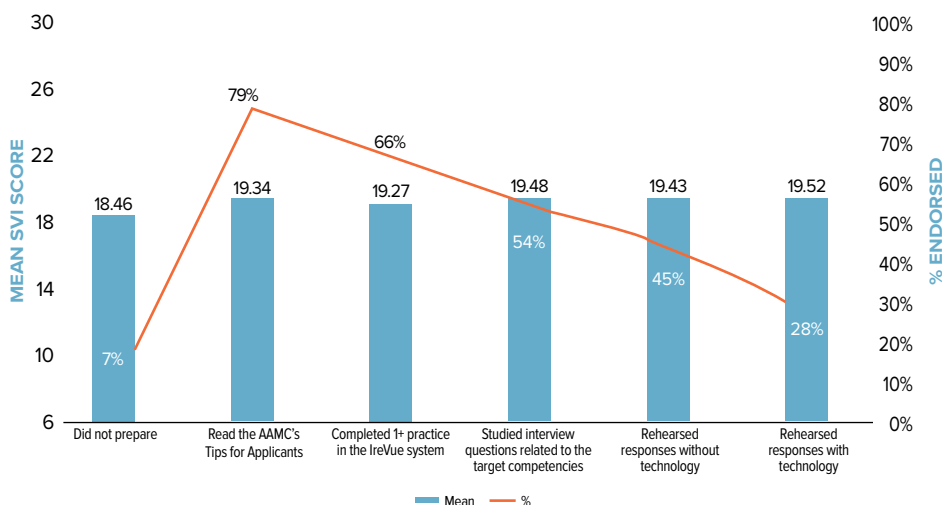


Figure notes: N = 1,505; Applicants could select multiple preparation strategies; SVI total scores range from 6 to 30.

**FIGURE 2. Who Helped You Prepare by Use and SVI Score**

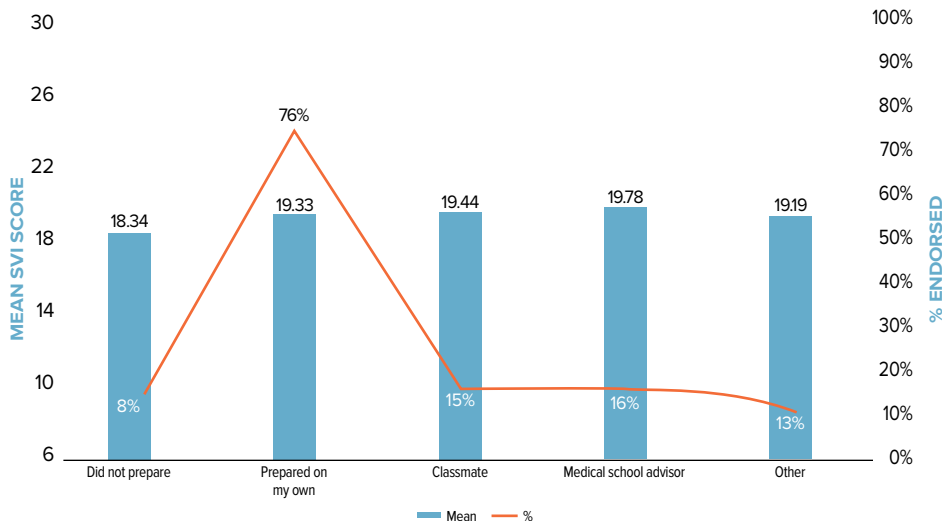


Figure notes: N = 1,505; Applicants could select multiple preparation strategies; SVI total scores range from 6 to 30.

# MEDICAL STUDENTS

Differences in mean SVI scores across recording locations were also insignificant, suggesting that applicants should complete the SVI in any location that makes the applicant comfortable.

## FOOTNOTES

1. The ACGME core competency Professionalism was renamed Knowledge of Professional Behavior to acknowledge that the video interview is not a direct observation of behavior but rather an inference of an applicant's proficiency based on their description of past experiences or what they would do in a hypothetical situation.
2. Bird S, Blomkalns A, Deiorio NM, Gallahue FE. Stepping up to the plate: Emergency medicine takes a swing at enhancing the residency selection process. *AEM Education and Training*. 2018;2(1):61-65.
3. AAMC researchers linked survey respondents to SVI scores using email addresses provided during the survey and matching with a student's email provided in ERAS. Responses from 1,401 applicants were excluded from analyses in survey #1 due to having inconsistent email addresses in the survey and in ERAS. There was negligible difference between the average SVI scores for those retained in survey 1 (M=19.22, SD=2.99), survey 2 (M=19.21, SD=3.06), and overall scores to the SVI (M=19.12, SD =3.08). \*

**FIGURE 3. Time Spent Preparing for the SVI by and SVI Score**

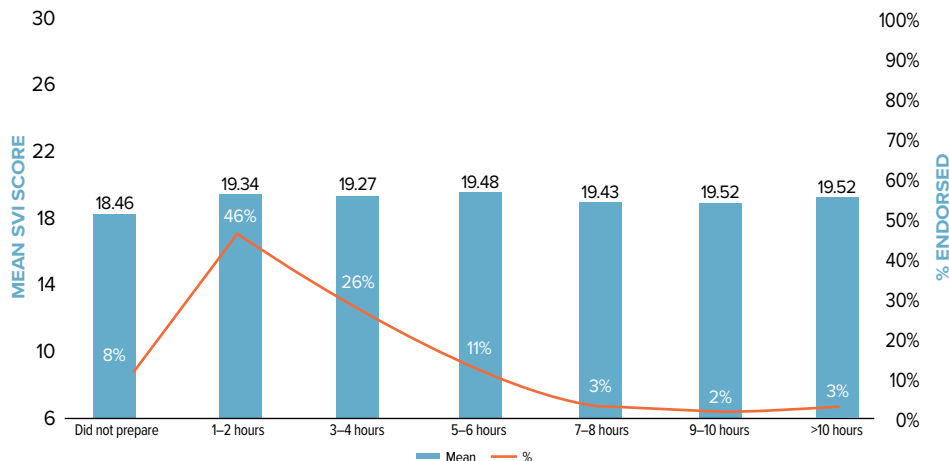


Figure notes: N = 1,491; SVI total scores range from 6 to 30.

**FIGURE 4. Location of taking the SVI by Use and SVI Score**

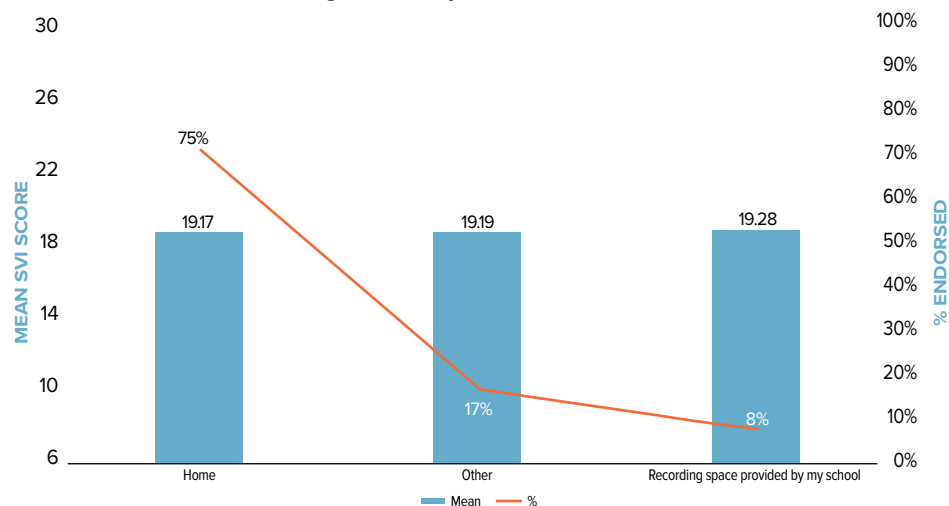


Figure notes: N = 1,892; SVI total scores range from 6 to 30.

**TABLE 1. Final Survey Samples by Demographic Group**

Race/Ethnicity	Survey 1		Survey 2	
	N	% of Matched Sample	N	% of Matched Sample
Asian	251	17	347	17
Black	100	7	121	6
Hispanic	112	7	140	7
Native American	2	<1	2	<1
Native Hawaiian or Pacific Islander	8	<1	17	<1
White	889	59	1,264	61
Other	52	3	58	3
<b>Gender</b>				
Women	532	35	750	36
Men	964	64	1,317	64
<b>Applicant Type</b>				
US-MD	842	56	1,327	64
US-DO	400	27	499	24
US-IMG	152	10	144	7
FMG	97	6	96	5

1. Individuals who self-identified as white alone were classified as white, individuals who self-identified as black alone or in combination with other races (including white) were classified as black, and individuals who self-identified as Hispanic alone or in combination with other races (including white) were classified as Hispanic. Similar rules were used to classify Asian, American Indian/Alaska Native, Native Hawaiian/Other Pacific Islander, and other individuals.

2. US-MD = graduate of an MD-granting medical school; US-IMG = U.S. citizen graduate of an international medical school; DO = graduate of DO-granting medical school; FMG = non-U.S. citizen graduate of an international medical school.





# TOBACCO CESSATION

## IN THE EMERGENCY DEPARTMENT



**Zachary Smith, MSIV**

*Albany Medical College*

**M**edical students represent a potentially under-utilized resource for discussing tobacco cessation counseling with ED patients. Students in the emergency department often have more time to spend with patients than any other member of the clinical team. In addition, nicotine addiction is a frequently encountered co-morbidity in the ED.<sup>2-3</sup> This is especially true in the context of clinical learning environments where attending and resident physicians rarely have time to provide nicotine addiction counseling or are hesitant to do so in the ED setting.<sup>4-6</sup> Additionally, physicians can bill for their time spent counseling patients on tobacco cessation.<sup>7</sup>

By routinely incorporating a clinical tool for smoking cessation into patient encounters, medical students can play a pivotal role in helping busy clinical teams achieve an often-unmet key clinical objective defined by the EM community.<sup>8-10</sup>

All it takes is starting the conversation, one patient at a time. While you may stumble initially, practice precedes confidence, and you may be surprised how receptive your patients are.<sup>12</sup>

The following “5 As” represent a validated approach to tobacco cessation in a busy clinical environment.<sup>11</sup> Furthermore, this smoking cessation tool can easily be employed during a clinical encounter to prompt the student and help initiate a brief discussion about nicotine addiction.

**Assess.** Attempt to quantify both the number of total pack-years and current nicotine consumption. Don’t forget to ask about all tobacco products (chewing tobacco, etc.)

**Advise.** Every patient should be counseled to reduce and eventually quit all tobacco consumption. Studies have shown that telling patients to stop makes a difference. If relevant, advise the patient of any relationship between tobacco consumption and their presenting chief complaint.

**Agree.** Collaborate with the patient to understand personal barriers to kicking his/her nicotine habit, understand his/her motivation to quit, and formulate a mutually agreeable goal (simple reduction, tapering, cold-turkey, etc.).

**Assist.** Many patients may not be aware of available resources. Whether encouraging the patient to follow-up with a PCP for outpatient medication, printing state quit-line literature, or educating the patient on over-the-counter nicotine replacement products, try to formulate a plan educate and equip your patient for success.

**Arrange.** Provide appropriate contact information for PCP follow-up and/or offer to assist with scheduling an appointment specifically dedicated to the patient’s nicotine addiction. ★



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# CONFERENCE IN REVIEW

# SIMS Conference 2018

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**T**raditionally, medical education has taken the form of textbook readings, lectures, and direct clinical practice with patients. Over the past few years, the med-ed movement has witnessed incredible shifts in presentation style, delivery methods, and performance improvement techniques. Medical schools and residencies more frequently use novel ways of teaching medicine, interpersonal communication, and other germane aspects of care. Among these, simulation has made an incredible impact on the world of education.

In 2016, Evan Strobelt, a fourth-year medical student at Baylor College of Medicine, created an organization dedicated to helping medical students organize simulation programs. Mostly student-driven, the Student Initiative in Medical Simulation (SIMS) connects students with faculty mentors, provides them with a large database of cases, and gives them the basic knowledge of how to use simulation to enhance their learning. Strobelt and others have published multiple interactive casebooks (free online) with a library of more than 80 faculty-reviewed cases to use in simulation exercises. Regional liaisons work with diverse student leaders across the country to set up their own sim programs. Finally, the organization plans to host multiple Sim Wars around the country to foster a fun and competitive atmosphere that facilitates learning. The goal is to make medical education effective, engaging, and accessible.

On Jan. 20, 2018, SIMS and UTHealth McGovern Medical School



in Houston, Texas, hosted the first annual SIMS Conference. More than 100 students from 13 medical schools participated in a high-fidelity simulation tournament consisting of 9 clinical scenarios, 6 individual medical skills sessions, and a poster presentation session. Throughout the tournament, high-acuity cases — such as patients suffering from a heart block, a ruptured abdominal aortic aneurysm, and an acute allergic transfusion reaction — challenged teams to demonstrate their medical knowledge through the proper diagnosis, evaluation, and treatment of the patient. Other cases challenged teams to utilize procedural skills, such as cricothyrotomy to manage airway compromise in a burn patient. A panel of physician judges evaluated each team until a winner was determined. Demonstrating efficient teamwork and sound clinical decision-making, the University of Texas Medical Branch (UTMB) at Galveston emerged as the 2018 SIMS champion.

All conference attendees, including those not competing in the tournament, participated in skills labs covering procedures such as lumbar puncture, bedside ultrasound, basic suturing, central line placement, medical moulage, cricothyrotomy, and airway management. A separate high-fidelity simulation suite provided the opportunity to practice guided simulated medical cases in a non-competitive setting. Post-conference feedback was largely positive and



More than 100 students from 13 medical schools participated in the SIMS Conference 2018.

PHOTOS BY EMMA STROBELT

highlighted a request for simulation-oriented lectures and presence of more residents and faculty mentors, for both learning and networking.

Going forward, SIMS leaders seek to continue outreach efforts through regional leadership. In keeping with the mission of SIMS and the spirit of FOAMed, we will continue to provide high-quality resources and mentorship to medical students around the country who are developing their own simulation programs. As evidenced by the SIMS Conference 2018, regional and national simulation competitions are a great way to bring students together and provide an opportunity to test their skills in a fun, competitive, and educational environment.

If you are a medical student looking to get involved or to start your own simulation program at your school, reach out to us via our website, or talk to the EMRA Simulation Division leaders! ★



# *In the* **SPOTLIGHT**

## **EMRA Honors 2018 Spring Awards Recipients**

Please join EMRA in congratulating our 2018 Spring Awards recipients.

*Special thanks to our EMRA Awards Committee for doing the work to make this recognition possible.*

### **2018 EMRA SPRING AWARD WINNERS**

#### **MERIT AWARDS**

##### **Academic Excellence Award**

Ari Friedman, MD, Harvard Affiliated Emergency Medicine Residency

##### **Alexandra Greene Medical Student Award**

Evan Strobel, Baylor College of Medicine

##### **Chief Resident(s) of the Year**

Hospital of University of Pennsylvania

Jennifer Love, MD

Brian Wexler, MD

Amy Zeidan, MD

##### **Fellow of the Year**

Jessica Nelson, MD, University of Colorado

##### **Resident of the Year**

Alex Koo, MD, Madigan Army Medical Center

##### **Jean Hollister EMS Award**

Tiffany Abramson, MD, LAC+USC Medical Center

##### **Rosh Review "One Step Further" Award**

Kathryn Lupez, MD, Carolinas Medical Center

##### **Robert Doherty, MD Teaching Fellowship Award**

Jessica Nelson, MD, University of Colorado

#### **FACULTY AWARDS**

##### **Residency Director of the Year**

Kaushal Shah, MD, FACEP, The Mount Sinai Hospital

##### **Associate Residency Director of the Year**

Anne Messman, MD, FACEP, Sinai-Grace Hospital

##### **Residency Coordinator of the Year**

Gloria Daniel, Wayne State University/Detroit Receiving Hospital

##### **Mentorship Award**

Keith Boniface, MD, George Washington University Hospital

#### **GRANTS**

##### **Local Action Grant**

Team project from Wayne State University/  
Detroit Receiving Hospital

Lauren Kava, MD

Jennifer Noble, MD

Yagnaram Ravichandran, MBBS, MD, FAAP

Kelsey Vargas, MD

##### **Research Grants**

Emily Cleveland, MD, MPH,

Harvard Affiliated Emergency Medicine Residency

Hani Kuttub, MD, University of Chicago

#### **TRAVEL SCHOLARSHIPS**

##### **ACEP Leadership & Advocacy Conference Scholarship**

Thomas Fowler, MD, Albert Einstein Medical Center

##### **AEM Consensus Conference Scholarship**

Morgan R. Bobb, University of Iowa Carver College of Medicine

##### **Airway Lab Scholarship**

Allen Chang, MD, Stanford University/  
Kaiser Permanente Medical Center

Lotiffa Colibao, MD,

John H. Stroger Jr. Hospital of Cook County

Sean Nardi, DO, Naval Medical Center San Diego

Kristina Shigyo, MD, Harbor-UCLA Medical Center

William D. Weber, MD, University of Chicago

Bryan Wilson, MD, University of Arizona

##### **Critical Care Medicine Conference Scholarship**

Stephen Hallisey, MD, Hospital of University of Pennsylvania

##### **EDDA Scholarship**

Maureen Canellas, MD, University of Chicago

##### **EMBRs Scholarship**

Cameron Gettel, MD, Brown University

##### **SAEM Scholarship**

Michael Wade, Howard University

# *Congratulations*

# EMRA is Building More EM Leaders of Tomorrow

One of the things EMRA loves most — and does best — is help EM residents and medical students grow into confident, inspiring leaders. Now there are more opportunities than ever: With the addition of 30+ new vice-chair positions to EMRA's most active committees, and the debut of the new Administration & Operations Committee, **there are more than 100 funded national leadership opportunities at EMRA!**

**But we're not stopping there.**

EMRA is building bridges with CORD and ACEP committees. With more opportunities to interface with these groups, you can get involved, too. Join us at CORD for our in-person meetings or virtually join an EMRA committee today (it only takes a few clicks at [emra.org](http://emra.org)), and you'll be the first to know when new opportunities arise.



## EMRA is starting a Leadership Academy!

The yearlong curriculum focuses on personal and professional development. Participants will learn the skills needed to be engaged, effective, inspirational, visionary, collaborative leaders.

The Leadership Academy will include in-person sessions at CORD and ACEP, along with once-per-month virtual sessions throughout the year. Instruction, content, and resources will be developed and presented by established leaders in emergency medicine and other industries.

The first class, which gets started in April, comprises the EMRA Committee leaders. Watch for more details in the future as we expand this important program. ★

## RECOGNIZING OUR OUTGOING LEADERS

Please help us recognize the lasting contributions and positive impact of the 2017-2018 EMRA committee leaders, whose tenure officially ends in April. If you know or see these stars of EM, thank them for their time, work, and attention!

### Critical Care Committee

Joshua Glick, MD, University of Pennsylvania  
Abdullah Bakhsh, MD, King Abdulaziz Hospital

### Diversity & Inclusion Committee

Vidya Eswaran, MD, Northwestern University

### Education Committee

Michael Yip, MD, Yale University

### Informatics Committee

Michael Hoaglin, MD, Duke University

### International Committee

Eric Lee, MD, Mount Sinai SOM – New York

### Pediatric EM Committee

Alexander Chang, DO, University of Illinois at Peoria

### Prehospital & Disaster Medicine Committee

Michael Spigner, MD, University of Cincinnati

### Research Committee

Olga Kovalerchik, MD, Yale University

### Simulation Committee

Xiao Chi (Tony) Zhang, MD, Thomas Jefferson University

### Sports Medicine Committee

Albert Leung, MD, University of Chicago

### Toxicology Committee

Alexandra Amaducci, DO, Lehigh Valley Health Network

### Ultrasound Committee

Franklin A. Poff III, MD, University of South Florida

### Wellness Committee

Randy Sorge, MD, Icahn SOM at Mount Sinai

### Wilderness Committee

Geoff Comp, DO, FAWM, Ohio Health Doctors Hospital



## WELCOMING OUR INCOMING LEADERS

As the baton is passed to incoming committee chairs, chairs-elect and vice chairs this spring, these groups are positioned to build on some incredible momentum — especially with the help of so many more funded leaders. Please help us welcome the new crew!

### Administration and Operations

*(newly formed committee!)*

Chair Joshua Gauger, MD,

University of Cincinnati College of Medicine

Chair-Elect Arnab Sarker\*, University of Virginia Medical School

Vice Chair Greg Fernandez, MD, Mount Sinai SOM – New York

### Awards

Chair Kathleen Yip, MD, Harbor UCLA Medical Center

### Critical Care

Chair Clark Owyang, MD, Mount Sinai SOM – New York

Chair-Elect Tanya Belle, MD, University of Connecticut

Vice Chair Matt Guess, MD, Harbor UCLA Medical Center

Vice Chair Yanika Wolfe, MD, Albert Einstein Medical Center

### Diversity & Inclusion

Chair Linelle Campbell, MD, NYU School of Medicine

Chair-Elect Christopher Harris, MD, University of Chicago

Vice Chair Amy Zeidan, MD, University of Pennsylvania

### Education

Chair Deena Khamees, MD, Baylor College of Medicine

Chair-Elect Thomas Yang, MD, Aventura Hospital & Medical Center

Vice Chairs Jeff Liu, MD (Baylor College of Medicine); Anthony DeVivo, DO (Mount Sinai St Luke's Roosevelt Hospital Center); Ross Christensen, DO (Maricopa Medical Center); Erin Karl\* (University of Minnesota); Hannah Hughes, MD (University of Cincinnati); Greg Tanquary, DO (Ohio Health Doctors Hospital); Jonathan Brewer (Texas A&M College of Medicine)

### EM Resident Editorial Team

Editor-in-Chief Tommy Eales, DO, Indiana University

Deputy Editor Brian Fromm, MD, Thomas Jefferson University

Editors Jeremy Lacocque, DO (MWU/CCOM @ Provident Hosp Cook County); Jayram Pai, MD (Mount Sinai SOM – New York); Megan Cobb, MD (University of Maryland); Leah McDonald, MD (NYU School of Medicine); Karen Bowers, MD (Emory University); Danny Van Valkinburgh, MD (University of Tennessee @ Memphis)

### Health Policy

Chair Allen Wang, MD, MPH, George Washington University

Chair-Elect RJ Sontag, MD, UT Health Science Center @ San Antonio

Vice Chairs Kyle Ragins, MD (UCLA Olive View); Neil Dubey, MD

(Mount Sinai SOM – New York); Erik Blutinger, MD (University of

Pennsylvania); Sushant Kapoor, MD (Christiana Care Health System);

Hannah Gordon\* (FIU Herbert Wertheim COM); Petrina Craine, MD

(Alameda Health System – Highland Hospital)

### Informatics

Chair Archana Shah, MD, University of Chicago

Chair-Elect DJ Apakama, MD, Mount Sinai SOM – New York

### International

Chair Brian Strickland, MD, University of Chicago

Chair-Elect Jon Dyal, MD, Baylor COM

Vice Chair Ravi Katari, MD, Mount Sinai SOM – New York

### Medical Student Council

Chair Sarah Ring, University of Miami

Vice Chair Mike Messina, Ohio University College of Osteopathic Medicine

### Pediatric EM

Chair Jijoe Joseph, MD, Nassau University Medical School

Chair-Elect Cindy Chang, MD, Harbor UCLA

### Prehospital & Disaster Medicine

Chair Seth Kelly, MD, University of Maryland

Chair-Elect Mike Sachon\*, Edward Cia COM - Virginia

### Research

Chair William Bianchi, DO, Naval Medical Center Portsmouth

Chair-Elect Alex Janke, MD, Yale New Haven Medical Center

Vice Chair Aadil Vora\*, Nova Southeastern University

### Simulation

Chair Aga DeCastro, MD, University of Connecticut

Chair-Elect Shyam Murali\*, Texas A&M College of Medicine

Vice Chair Alex H. Tran, Brown Medical School

### Sports Medicine

Chair Beau Grantier, MD, University of Mississippi

Chair-Elect Dustin Harris, MD, University of Chicago

Vice Chair Stewart Lee, MD, Albany Medical Center

### Toxicology

Chair Aaron Frey, MD, Lehigh Valley Health Network

Chair-Elect Daniel Bral\*, Nova Southeastern University

### Ultrasound

Chair Landon Pratt, MD, University of Arizona

Chair-Elect Ben Karfunkle, MD, Baylor College of Medicine

Vice Chair Kevin Hu, MD, Mount Sinai SOM – New York

### Wellness

Chair Leann Mainis, MD, UCSF-Fresno

Chair-Elect Jayram Pai, MD, Mount Sinai SOM – New York

Vice Chair Taryn Webb, MD, Mount Sinai SOM – New York

### Wilderness

Chair Charlie Duke, MD, Yale New Haven Medical Center

Chair-Elect Lauren Altschuh, MD, Wellspan York Hospital

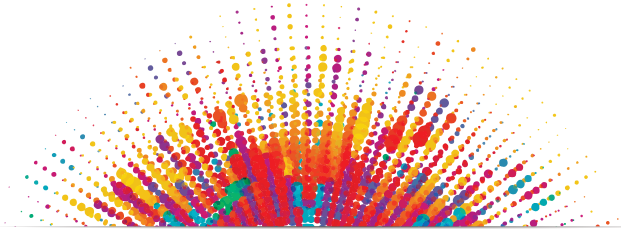
Vice Chairs Marc Cassone, DO (Geisinger Medical Center);

Yevgeniy Maksimenko, MD (Boston Medical Center)

\*Graduating medical student

# Make Art and Get Smart!

## EMRA Events at CORD AA



### EMRA Art Therapy Experience

Explore, engage, create, and connect through physician-made art

**Sunday, April 22**

**8 am – 5 pm**

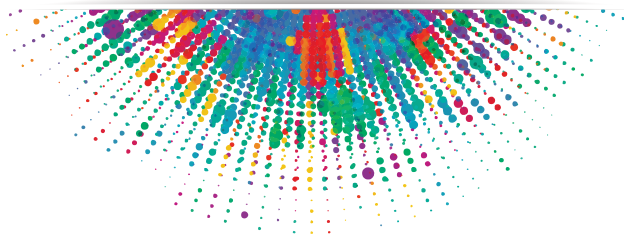
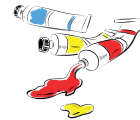


**Play in the proverbial sandbox!**

Art therapists will guide you through the process.  
No need to be an artist! Physicians and families welcome.  
We provide all art supplies!



**FREE  
and  
FUN!**



**Sunday, April 22**

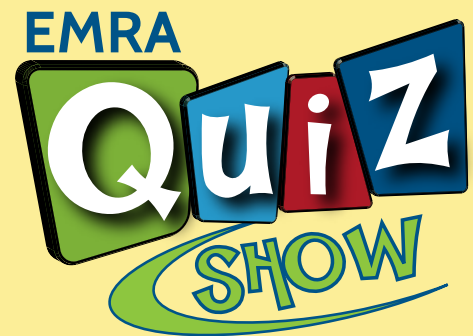
**5:30 – 7:30 pm • Presidio ABC**

**Twelve teams compete at the EMRA Quiz Show!**

Music, madness and tons of fun!

Watch Virginia Commonwealth University defend their title in this trivia-medical-pop culture game of wits!

And we're not kidding here — you might win some big cash this year!



**Tuesday, April 24**

**9 am – Noon • Presidio ABC**

**A new event, only EMRA could dream up!**

It's an all-around skills competition! What makes it chaotic?

We draw the players at the event; there's a Bloody Mary–Mimosa cash bar; the challenges are just plain head-scratchers!

There's more, but you have to show up to find out.

**SO SHOW UP!**





EMRA

# Party

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at Pat O'Brien's?

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and the legendary  
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Monday  
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# empower

Sharing our Stories

## Your Time, Your Legacy

Ije Akunyili, MD, MPA, FACEP

**Medical School:** University of Maryland

**Residency:** University of Texas – Houston

**Current Position:** Chair and Medical Director, Department of Emergency Medicine, Waterbury Hospital in Connecticut

**Ije Akunyili was embarking on a career in economic policy and development at the World Bank when she realized her passion for helping people, particularly the disadvantaged, could be served more directly through medicine. She changed course — and she's been changing patients' lives ever since.**

**A former EMRA Speaker of the Council, Dr. Akunyili offers a unique perspective on how to forge your path in this specialty while fulfilling even bigger goals.**

### Who gave you your best career advice — and what was it?

My very best career advice was from my Mom, who passed away 4 years ago. She said you have to work very hard, be 10 times as good as the next person, and remember the world is not fair, so no whining.

### If you were just starting residency, what would you do differently?

I was a very, very hardworking resident, and I had 2 small kids. In addition to the work I did for EMRA, maybe I'd do more free clinics and more international work. It's just a luxury I didn't have.

### How did your EMRA service impact your residency years?

My work on the EMRA Board was transformational. I was just a resident in Texas. All of a sudden I had a national platform where I was able, as a resident, to influence big decisions in emergency care and be at the forefront of advocacy. Through EMRA I found my political voice. I was able to do much more to take care of patients beyond my own ED. I still keep in touch with the people who were on my board. We meet up at ACEP and we check on each other regularly. It's a nice brotherhood and sisterhood.

### How did your expectations differ from reality when you transitioned out of residency into your full-time career?

I think the biggest surprise, as much as we feel like residents are not as engaged as they should be, is how much less engaged a practicing physician can be.

At my first group a lot of my colleagues were not even ACEP members and had no concept of the issues the specialty was grappling with. Also, especially if you're going to work at a community hospital, you can no longer just call the renal fellow, for example — *you're* the doctor and you need to have the answers. That's why I love the EMRA apps. I have all of them on my phone. I tell residents, "Look at yourself in your third year. Take a good look, because that's your peak." That's when you're absorbing the most amount of knowledge, the best skill set, your most focused attention.

### After working first in community medicine, you moved into a leadership position at an academic hospital and have continued up the ranks in administration. What keeps you coming to work every day?

I love the balance I have. I love every facet of my life. I love shift work, and I also love being an administrator. I think I'm a better physician for being the leader of the team because I have a global perspective, and I'm not just affecting the lives of however many patients I see on shift, but also the 50,000 patients that pass through our ED. To lead a team that changes the story of emergency care, that's amazing.

### Best advice for surviving a shift during full moon?

Take your own pulse during a chaotic shift. I will intentionally dial back. I take longer breaths. I move very deliberately. Then I triage. Who's going to die? Who just thinks they are? You can't be hassled; you're the

captain of the ship, and everybody takes their cue from you. I'm never nervous. I have a plan A, B, C — I have a plan Z.

### In my coat pocket right now?

I used to have everything — all the cards and books and pamphlets and notes. Now I have my iPhone. ★

## GET TO KNOW IJE AKUNYILI

**VIPs in your life?** I have 2 beautiful children and my husband. They keep me going.

**What do you do for fun?** Oh...fun? I take care of my children. I try to read and exercise, and I listen to podcasts on every topic you can imagine.

**What makes you laugh every time?** My son. He's a great kid. He's 10 years old and he laughs all the time.

**What goes on pizza?** Nothing anymore; pizza is for residency.

**How you get your exercise?** I do CrossFit, I run, and I lift weights.

**Most-used app on your phone:** Hmm... somewhere between PEPID and the EMRA Antibiotic Guide and my podcast app.

**Last song that stuck in your head?** My daughter plays flute in her band, and she has practiced *Majestica* a million times. I've listened to it in all variations. She's such a hardworking kid.

**Beach or mountains?** Oh, beach for sure. If I'm off, I just want to lie on the beach and do nothing.

# WELLNESS IN ALL FORMS

## The Daddy Dance

**Eric McDonald, MD**  
ACGME RC-EM Liaison  
University of Mississippi

It was a peaceful day in the pediatric emergency department. For a change, we were overstaffed for our current patient load. In fact, I had just started a water gun fight with saline syringes. That's when it happened — the tracker board lit up with a notification that a 3-year-old girl who had just been run over by a car was en route.

She wasn't doing well. Her blood stained the Frozen shirt she was wearing. She needed immediate intubation. She received blood products, chest tubes, and even neurosurgical intervention. We worked for hours, trying everything possible.

She didn't make it.

In that moment, I couldn't help but think about my own 3-year-old. In fact, I couldn't help but think about all 7 of my

children and my beautiful bride.

I called them on my way home, as the loss of my tiny patient weighed heavily on my heart. Despite my own tears, it calmed me to hear the chaos that my wife and I call normal going on around her. All I could hear was laughing and screaming as our children circled her.

I couldn't wait to dive into that rowdy, raucous, life-affirming melee.

Just like every other day, I walked through the door of my house, and there it was—the daddy dance.

The “daddy dance” is what my wife and I call our kids' reaction when I get home from work. They sing, scream, and dance. Imagine being circled and serenaded with made-up songs filled with shouts and laughter. It's amazing! Within two strides of the door, I become a jungle gym, with children hanging off of every extremity. I realize this may not sound

like everyone's idea of the most relaxing end to a stressful day. But to me, it's a sweet dream.

That day in particular, the tragedy and stress of the job seemed to melt away. The daddy dance refocused my thoughts on what is important to me. It is my escape, my retreat, and my freedom. While nothing can — or even should — change the sorrow and empathy we feel for our patients, the daddy dance is my safe haven.

What is yours?

Residency and emergency medicine are hard. I can't think of a more stressful situation than to voluntarily be part of someone's worst day, every day. But this is what we chose. To many of us, it's a calling. In an environment like that, it's more important than ever to find a way to vent and stay sane. You have to find your “daddy dance.” ★

## Graduating Residency in 2018?

Shilpa Cheela, MD  
Edison, NJ  
2014 Residency Graduate

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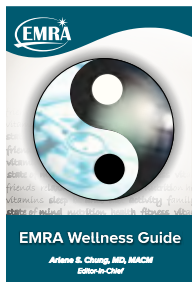


[www.acep.org/renew](http://www.acep.org/renew)



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## New Guide Offers Tips for Boosting Wellness

The brand-new **EMRA Wellness Guide**, launched in conjunction with Emergency Medicine Wellness Week in March, takes a close look at the subject of wellness during residency: what it entails, what threatens it, and how to strive for it.

Editor-in-chief Arlene S. Chung, MD, MACM, led a team of residents and faculty in carefully addressing the many aspects of wellness and resiliency during residency and beyond. The result is a guide that offers concrete steps that individuals and programs can take, both on the job and in life outside the hospital. The team explores team-building, debriefing, on-the-job safety, second victim syndrome, and more.

Dr. Chung, the 2018 chair of the ACEP Wellness Section, previously participated in an EMRA Hangout to address questions from residents and medical students interested in (or struggling with) wellness. Watch the Hangout at <http://bit.ly/2vAqrEK>.

You can access the Wellness Guide in 3 ways:

- Read an **HTML version** online at <https://emra.org/books/emra-wellness-guide/cover>.
- Download or read a **PDF version** at <https://emra.org/globalassets/emra/publications/books/emra-wellnessguideonline-2018.pdf>.
- Listen to an **audio version!** Go to <https://emra.org/about-emra/publications> to find the link.

Please join EMRA in thanking the team that brought you this guide! Along with Dr. Chung, authors include Angela Chen, MD; Taryn Webb, MD; Lara Vanyo, MD, MSc; Daniel Lakoff, MD, FACEP; Kevin Hu, MD; Christie Lech, MD; Jayram Pai, MD; Randy Sorge, MD; Ramin Tabatabai, MD, FACEP; and Alicia Pilarski, DO, FACEP. Eric Wei, MD, served as reviewer. ★



## EMRA Website Gets Smart

If you've visited [emra.org](http://emra.org) in recent weeks, you've noticed some big changes.

Welcome to the new — and smart — EMRA online!

This new site has built-in functionality that will remind you of important dates, feed tailored content we think you will want (or need), and make it easy for you to plug in to the specialty.

**But first — help us help you!**

1. Go to [emra.org](http://emra.org).
2. Log in as an EMRA member.
3. Join the committees and divisions doing work that interests you.
4. Click through to your membership benefits with single sign-on when you log in at [emra.org](http://emra.org). ★

## Annals of Emergency Medicine Calls for Abstracts from Residents, Fellows

Now in its 20th year, the Residents' Perspective section in *Annals of Emergency Medicine* comprises articles by emergency medicine residents and fellows. The purpose of the column is to create a forum for the discussion and analysis of topics affecting trainees in emergency medicine. They are written as informative instructional pieces, educational research, referenced position papers, or unique resident perspectives on current emergency medicine topics. Perspective articles can be found at [annemergmed.com/content/residents](http://annemergmed.com/content/residents).

### Invitation

We would like to invite you to **submit an unstructured abstract** (limited to 300 words) outlining the background and significance of the topic to emergency medicine residents.

Authors of promising abstracts will be invited to submit a full manuscript (maximum length 2,500 words) for peer review.

The following themes are of particular interest for the Residents' Perspective section at this time:

- Ethics of Utilizing Placebos in Clinical Practice
- Economics of Health Insurance
- Technology in Emergency Medicine

### Instructions

Submit your abstract via *Annals'* online submission system, Editorial Manager, at [editorialmanager.com/annemergmed](http://editorialmanager.com/annemergmed) (select the "Residents' Perspective" article type).

Invited manuscripts will undergo the same peer review process as all other submissions to *Annals*.

More information for authors can be found at [annemergmed.com/content/categories#residentsperspective](http://annemergmed.com/content/categories#residentsperspective). For questions, contact Editorial Board Resident Fellow Josh Mirkin, MD, at [annalsfellow@acep.org](mailto:annalsfellow@acep.org). ★



## ABEM Announces Alternative to ConCert™ Exam

The ConCert™ Exam is the current way for physicians to show they continue to possess the knowledge and cognitive skills of an ABEM-certified emergency physician. In 2020, a second way to demonstrate these competencies will begin to be phased in.

The alternative will consist of:

- Shorter, more frequent tests: Each test will assess one or more specific content areas relevant to the clinical practice of Emergency Medicine, such as cardiovascular disorders or trauma. The tests will be about an hour long.
- The ability to take a test again if it's not passed the first time: Additional chances will be available to retake and pass a test, which will give physicians a clearer idea of what topics need to be reviewed.

ABEM talked with emergency physicians across the country and selected an alternative that integrated many of their ideas. ABEM put a high priority on providing physicians flexibility, enhanced relevancy, and greater opportunities to maintain certification. As this program moves forward, ABEM will continue to reach out to emergency physicians for their ideas.

In summary:

- In 2018, no changes to the ConCert Examination.
- In 2019, the ConCert Exam will be administered twice: once in the spring, and again in the fall. This schedule will continue indefinitely.
- In 2020, the new alternative assessment will begin to be phased in.
- All other requirements will remain the same.

For more information and to review the FAQs, visit the ABEM website at [abem.org](http://abem.org). \*

## Two New Members Elected to ABEM Board

Felix K. Ankel, MD, and Diane L. Gorgas, MD, recently joined the ABEM Board of Directors. Dr. Ankel was nominated by ACEP, and Dr. Gorgas was nominated by the EM community-at-large. ACEP is an ABEM sponsor organization.



Dr. Ankel is Vice President for Health Professions Education at HealthPartners Institute in Bloomington, Minnesota, and Professor of Emergency Medicine at the University of Minnesota. Dr. Gorgas is a Professor and Vice Chair of Academic Affairs in the Department of Emergency Medicine, and Director of the Office of Global Health at The Ohio State University in Columbus, Ohio.



They will begin their terms as ABEM directors at the close of the summer board meeting. The ABEM Board of Directors is composed solely of volunteer, board-certified, clinically active emergency physicians who are participating in ABEM Maintenance of Certification, a program of continuous learning and periodic assessment. \*

## Make Plans Now to Celebrate National EMS Week in May

**National EMS Week 2018**, presented by ACEP in partnership with the National Association of EMTs (NAEMT), will be held May 20-26. The EMS Strong theme this year is "Stronger Together," and each day of the week has a special focus:

**Monday:** Education

**Tuesday:** Safety Tuesday

**Wednesday:** EMS for Children Day

**Thursday:** Save-A-Life (Hands-only CPR and Stop the Bleed)

**Friday:** EMS Recognition Day

For resources and ideas to help organize National EMS Week activities, visit [acep.org/emsweek](http://acep.org/emsweek). \*



**ACEP's 50th anniversary** — which will be celebrated all year — reflects on the moments that matter most for our specialty, our members, our patients — moments that matter most to you.

Throughout 2018, ACEP will honor the titans of emergency medicine who blazed the trails so others could follow. Participate in the conversation! Watch for:

- *Social media campaigns to share the highs, lows, and life-changing moments in emergency medicine*
- *50th anniversary-themed podcasts on the past, present, and future of emergency medicine*
- *Anniversary editions of ACEP Now and Frontline*
- *A blow-out celebration at ACEP18 in San Diego featuring an interactive history museum that chronicles the journey of emergency medicine from battlefield to inner city to rural America and every spot in between*

ACEP will release a commemorative anniversary book chock-full of images that show how the specialty has evolved during the past half-century. Pre-order your copy by visiting [acep.org/50thbook](http://acep.org/50thbook).

Follow the conversation on Twitter via [#EMeverymoment](https://twitter.com/EMeverymoment). \*

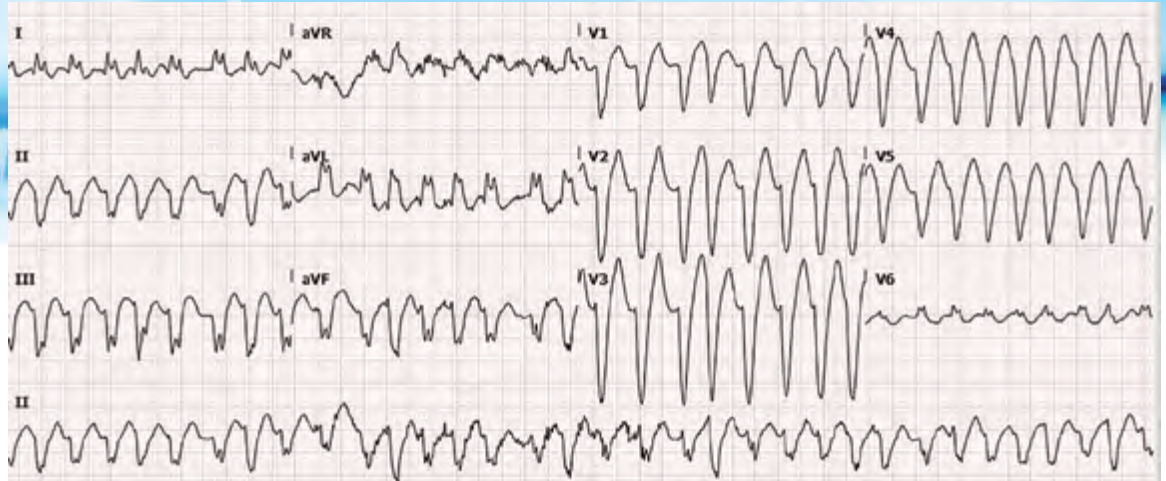
# ECG Challenge

**Sarah Watson, DO**

Emergency Medicine PGY-2  
Christiana Care Health System  
Newark, DE

**Jeremy Berberian, MD**

Associate Director of Resident Education, Dept. of Emergency Medicine  
Christiana Care Health System  
Newark, DE  
@jgberberian



**CASE.**

A 39-year-old female with no prior cardiac history presents with palpitations.

What is your interpretation of the following ECG, and how would you treat this patient?

See the ANSWER on page 46



## EMF Resident VIP Club

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A \$120 tax-deductible gift (you can make quarterly installments of only \$30) will place you in the prestigious *Resident VIP Club*. Resident VIP Club members\* receive:

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\*Emergency medicine resident or medical student who contributes \$120 annually.

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# Assess this Treatment



**Cosby G. Arnold, MD, MPH**  
 University of Tennessee Emergency  
 Medicine Residency at Memphis

**Is this central venous line okay to use?  
 What should be done, if anything?**

**See the ANSWER  
 on page 47**

## PEER

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EMR\_0418\_1013\_0318

# ECG Challenge



For more, see  
p. 24-32 of the  
EMRA EKG Guide

## ANSWER

The ECG shows an irregular wide-complex tachycardia with a ventricular rate of ~192 bpm, left axis deviation, and a left bundle branch block pattern. The absence of P-waves or flutter waves narrows the differential (see Learning Points) to atrial fibrillation with aberrant conduction or polymorphic VT (PMVT). The LBBB pattern suggests a supraventricular origin and makes PMVT less likely. There is some subtle variability in the QRS morphologies with ventricular rates up to 300 bpm, which should raise suspicion for the presence of an accessory pathway (AP) and the possibility of AFib with WPW. To help distinguish between AFib with a rate-related BBB and AFib with WPW, it is critical to review prior ECGs for conduction abnormalities (BBBs) and signs suggesting WPW (eg, delta wave). This is important because treatment with AV nodal blockers should be avoided in AFib with WPW. When in doubt, treatment options for hemodynamically stable patient with an irregular WCT include electrical cardioversion, amiodarone, and procainamide.

## Why to avoid AV nodal blocking agents in AFib with WPW?

To answer this, it is necessary to understand the fundamentals of cardiac conduction. Anterograde conduction is toward the ventricle and retrograde is away from the ventricle. An AP can conduct in either direction. Orthodromic conduction describes anterograde conduction via the AV node and retrograde conduction via the AP. In the absence of a pre-existing conduction abnormality (eg, BBB), the QRS will be narrow. Antidromic conduction describes anterograde conduction via the AP and retrograde conduction via the AV node. Anterograde conduction via the AP leads to abnormal ventricular depolarization and a wide QRS. The participation of the AP in a reentry tachycardia is what differentiates AVRT from AVNRT (in which anterograde and retrograde conduction both occur within or around the AV node).

The importance of conduction via an AP in AFib is due to its short refractory period relative to the AV node. The ventricular rate in AFib is usually limited by the refractory period of the AV node. Nodal blockers will slow conduction through the AV node but not the AP. Since the AP has a shorter refractory period, atrial impulses can conduct at a much faster rate. This can lead to ventricular fibrillation, either because the impulse arrives during the vulnerable phase of the ventricular cycle or because the rapid ventricular rate causes such a low cardiac output that there is myocardial ischemia. In AFib with aberrant conduction, rates  $> \sim 220$  bpm suggest anterograde conduction via an AP as this exceeds the refractory period for the AV node.

## Case Resolution

The patient was treated with amiodarone and spontaneously converted to a normal sinus rhythm with no signs of ischemia or abnormal conduction, supporting the final diagnosis of AFib with a rate-related LBBB. Labs, including electrolytes and troponin, were normal. She was admitted to the Cardiology service and an echocardiogram showed an ejection fraction of 35% with no mural thrombus. Myocardial perfusion studies showed global hypokinesis with no sign of ischemia, consistent with a tachycardia-related cardiomyopathy. She was started on a beta blocker and novel anticoagulant, and discharged with cardiology follow-up.

## LEARNING POINTS

### Irregular Wide Complex Tachycardia

- Rate  $> 100$  bpm, QRS  $> 120$  ms, and variable RR interval
- DDx includes
  - Atrial fibrillation with aberrant conduction (BBB or WPW)
  - Atrial flutter with variable block and aberrant conduction
  - MAT with aberrant conduction
  - Polymorphic ventricular tachycardia
  - Ventricular fibrillation

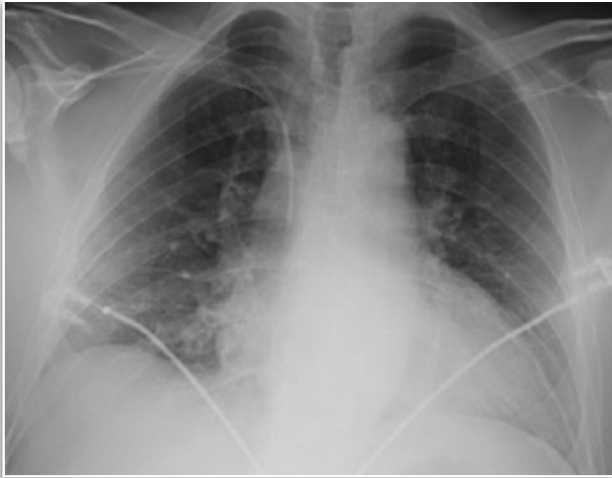
### Tachydysrhythmias in WPW

- Can be narrow (orthodromic conduction via AV node) or wide (antidromic conduction via accessory pathway)
- WPW with orthodromic AVRT
  - Rapid, regular narrow complex tachycardia
  - Treat like AVNRT (PSVT)

- WPW with antidromic AVRT
  - Rapid, regular wide complex tachycardia
  - Can look like monomorphic VT- treat like VT
- WPW with atrial fibrillation
  - Very fast, irregularly irregular tachycardia (ventricular rate typically 180-300 bpm)
  - Variable QRS morphologies in any single lead (i.e., beat-to-beat variation in QRS morphology)
  - Often misdiagnosed as atrial fibrillation with aberrancy from BBB
  - AV nodal blockers should be avoided; can precipitate ventricular fibrillation and cardiovascular collapse
- Procainamide and cardioversion/defibrillation are safe treatment options for any wide complex tachycardia in a patient with WPW. ★

# Assessment

## Malpositioned Central Line



**This AP chest radiograph shows malpositioning of a right subclavian approach central venous catheter.** The catheter does not follow the anticipated course into the distal superior vena cava or proximal right atrium but instead sharply curves into the azygos vein. The catheter was subsequently withdrawn and repositioned to terminate in the superior vena cava.

Placement in the azygos system occurs in approximately 1.2% of cases and is more likely to occur if the vein is dilated, as in heart failure, or if cannulated from the left side.<sup>1,2</sup> Potential complications include azygos vein perforation, thrombosis, or stenosis.<sup>3</sup> To avoid these complications, catheters found to cannulate the azygos vein should be slightly withdrawn or removed and recannulated at an alternative site. ★

# Board Review

## QUESTIONS

**NEW! PEER IX QUESTIONS NOW AVAILABLE!**

*PEER (Physician's Evaluation and Educational Review in Emergency Medicine)* is ACEP's gold standard in self-assessment and educational review. These questions are from *PEER IX*, which made its print debut in June 2017.

**For complete answers and explanations, visit the Board Review Questions page under "Features" at [emresident.org](http://emresident.org).**

To order PEER IX, go to [acep.org/bookstore](http://acep.org/bookstore).



1. A 32-year-old woman who is 3 weeks postpartum presents with lightheadedness, generalized fatigue, and difficulty with her vision. Her blood pressure is 210/107, her pupils are normal and reactive, and she has a bilateral visual field defect. She suffers a generalized tonic-clonic seizure in the ED and is taken for a head CT, which shows ill-defined areas of low attenuation in the bilateral occipital lobes. What is the most likely diagnosis?
  - A. Adrenergic agonist overdose
  - B. Basilar artery aneurysm
  - C. Posterior reversible encephalopathy syndrome
  - D. Sheehan syndrome
2. Considering the clinical presentation of a patient with suspected infective endocarditis, which of the following antibiotics is the most appropriate initial choice for empiric treatment in the ED?
  - A. Cefepime
  - B. Gentamicin
  - C. Moxifloxacin
  - D. Vancomycin
3. Which of the following is the most appropriate course of action to manage a 15% stable nontraumatic pneumothorax?
  - A. Administer oxygen and repeat x-ray in 4 hours
  - B. Admit for 24-hour observation
  - C. Insert a 36 Fr chest tube
  - D. Perform needle aspiration
4. Which of the following, after ingestion, is associated with delayed GI symptoms and hepatic failure?
  - A. Amanita mushroom
  - B. Oleander
  - C. Poison hemlock
  - D. Water hemlock
5. Which of the following physical examination findings is pathognomonic in flail chest?
  - A. Bradypnea
  - B. Hemothorax
  - C. Paradoxical movement
  - D. Seatbelt sign ★

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





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### ALASKA

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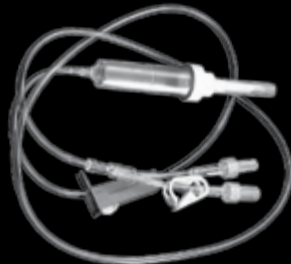
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## Department of Emergency Medicine Fellowship Opportunities

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Program Director: Dr. Christopher Miller
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Qualified candidates should email a letter of interest to the appropriate fellowship director c/o Ms. Dildred Houston, Education and Practice Coordinator, Department of Emergency Medicine, at [Dildred.Houston@UHhospitals.org](mailto:Dildred.Houston@UHhospitals.org)



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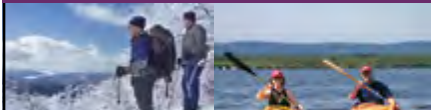
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Contact: Denise Romand, Medical Staff Recruiter,  
Saratoga Hospital:  
[dromand@saratogahospital.org](mailto:dromand@saratogahospital.org)  
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# { Job Opportunities }

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The Emergency Medicine Department at Penn State Health Milton S. Hershey Medical Center seeks energetic, highly motivated and talented physicians to join our Penn State Hershey family. Opportunities exist in both teaching and community hospital sites. This is an excellent opportunity from both an academic and a clinical perspective. As one of Pennsylvania's busiest Emergency Departments treating over 75,000 patients annually, Hershey Medical Center is a Magnet® healthcare organization and the only Level 1 Adult and Level 1 Pediatric Trauma Center in PA with state-of-the-art resuscitation/trauma bays, incorporated Pediatric Emergency Department and Observation Unit, along with our Life Lion Flight Critical Care and Ground EMS Division. We offer salaries commensurate with qualifications, sign-on bonus, relocation assistance, physician incentive program and a CME allowance. Our comprehensive benefit package includes health insurance, education assistance, retirement options, on-campus fitness center, day care, credit union and so much more! For your health, Hershey Medical Center is a smoke-free campus. Applicants must have graduated from an accredited Emergency Medicine Residency Program and be board eligible or board certified by ABEM or AOBEM. We seek candidates with strong interpersonal skills and the ability to work collaboratively within diverse academic and clinical environments. Observation experience is a plus.

*FOR ADDITIONAL INFORMATION, PLEASE CONTACT:*



**PennState Health**



Susan B. Promes, Professor and Chair, Department of Emergency Medicine, c/o Heather Peffley, Physician Recruiter, Penn State Health Milton S. Hershey Medical Center, 500 University Drive, PO Box 855 Mail Code A595, Hershey PA 17033, Email: [hpeffley@pennstatehealth.psu.edu](mailto:hpeffley@pennstatehealth.psu.edu)

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## EMERGENCY DEPARTMENT OPENINGS SAN LEANDRO AND ALAMEDA HOSPITALS

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For More Information:  
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Chair, Emergency Medicine  
[bsimon@alamedahealthsystem.com](mailto:bsimon@alamedahealthsystem.com)

WESTERN PENNSYLVANIA

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## SEEKING EMERGENCY DEPARTMENT PHYSICIANS

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## THE AREA

Cape Fear Valley Health is located in the thriving and diverse community of Fayetteville, NC which consists of more than 319,000 residents. Fayetteville has received the prestigious All-America City Award three times from the National Civic League.

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## CAPE FEAR VALLEY HEALTH

Please contact Ashley Dowless, Interim Director,  
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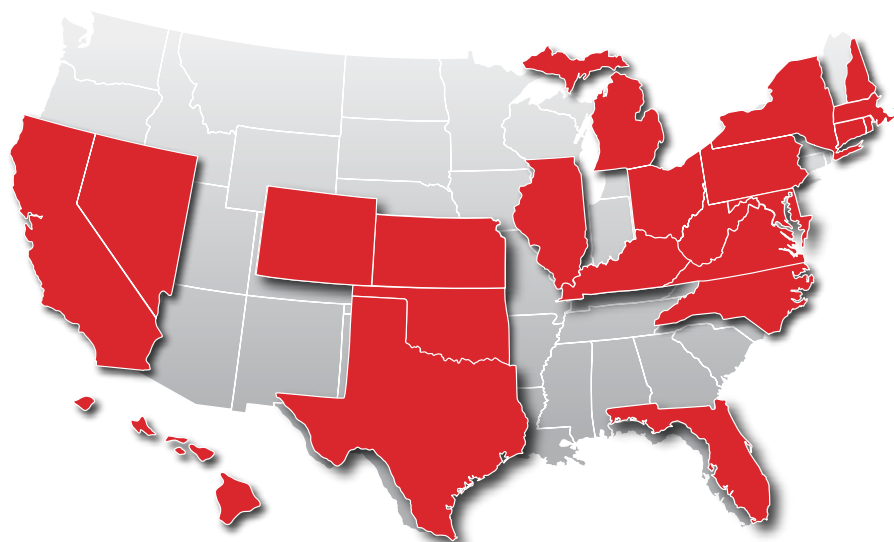
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