

EM Resident



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MDMA Toxicity

**Steroids in
Septic Shock**

**Congenital
Heart Disease
in Children**

**Pacemaker
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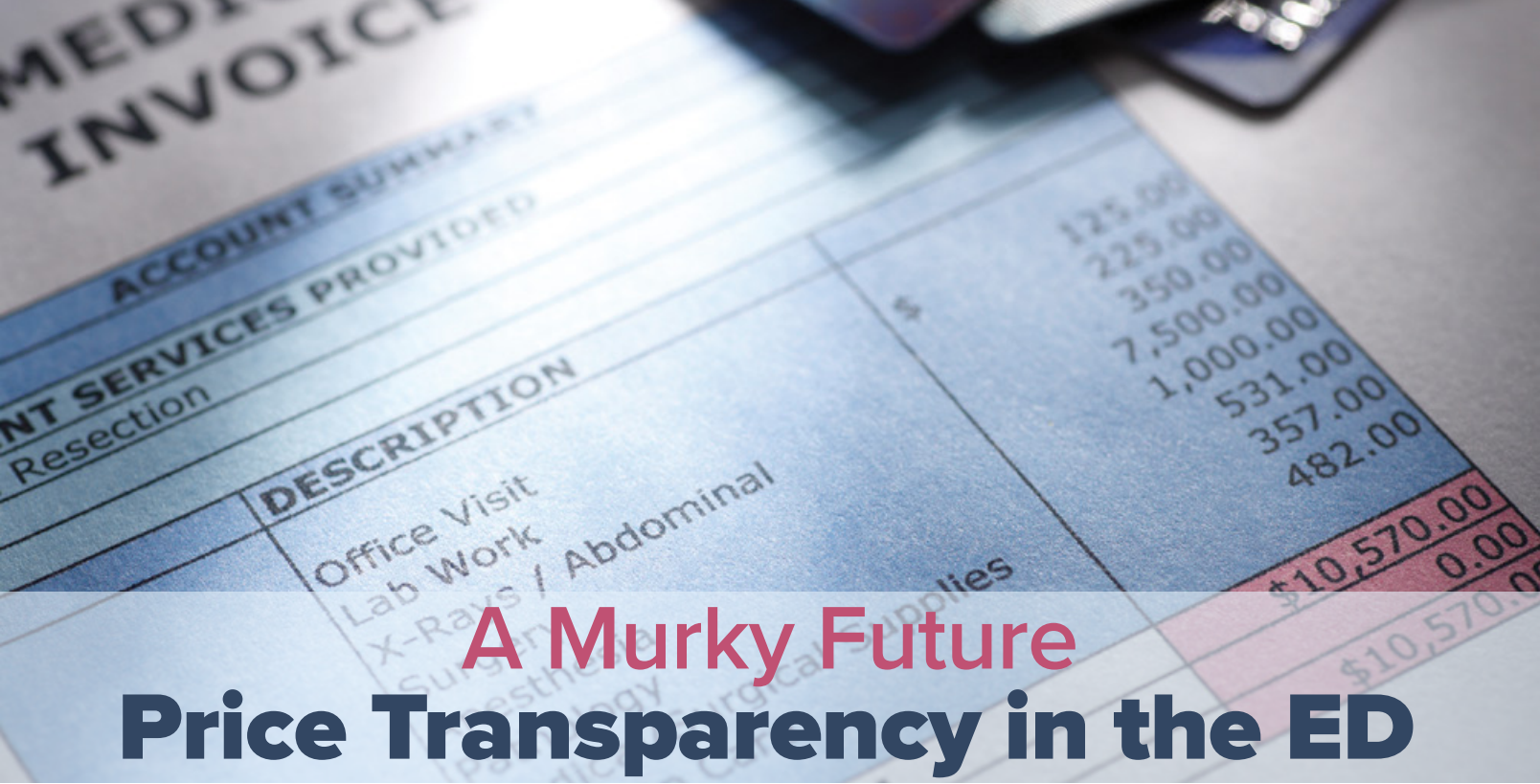
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A Murky Future Price Transparency in the ED

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It's no secret that emergency department visits are expensive. The time-sensitive, high-stakes nature of our work is such that services must be provided with the utmost speed and efficiency — often requiring costly, expedited tests to determine whether a patient has a life-threatening illness.

While higher cost might be perceived as a reasonable trade-off for immediate access to high quality emergency care, there remains a monumental problem in how patients are billed for emergency visits.

The actual cost is impossible to determine until *after* the visit. And no matter what happens, patients are stuck with the bill.

Appropriately, there has been significant public outrage against surprise billing after ED visits. These unexpected charges are multi-factorial and range from physician-dependent items such as rejected out-of-network claims (a.k.a. “balance billing”) to hospital-dependent items such as variable drug costs or facility fees, like radiology services.

With multiple providers and services being involved in even the most basic of cases, it's easy to see how bills can become incredibly complex and highly variable quite fast.

Amid increasing scrutiny of unclear billing practices, physician and patient advocacy groups alike have advocated for better price transparency.

On January 1, the Centers for Medicare and Medicaid Services (CMS) responded to this by enacting a policy requiring that hospitals “establish and make public a list of their standard charges.”

While well-intended, the policy is purposely vague regarding how hospitals are supposed to accomplish this task.

In the emergency department setting, there is one major conflict of interest that has provided hospitals with an incentive to remain as elusive as possible in the reporting of standard charges in line with this policy.

This, of course, is EMTALA.

The Emergency Medical Treatment and Labor Act is a federal law created in 1986 that mandates for all patients to be evaluated, stabilized, and treated upon presenting to an ED, regardless of their ability to pay. EMTALA opened the door to a social revolution in emergency medicine, transforming all EDs into health care safety nets overnight.

It was a major win for the poor and disenfranchised members of American society, who had previously depended on relatively few charity hospitals for their medical care.

The goal and spirit of this act were and remain a core value of emergency physicians, and we continue to provide the most EMTALA-related charity care of any specialty in the house of medicine — by a hefty margin.

So, how does transparent billing interfere with EMTALA?

Unsurprisingly, the answer is money.

Under certain situational interpretations of EMTALA, the act of advertising a price for an emergency service could be perceived as coercive and constitute a costly violation. A single fine can cost up to \$50,000 for a hospital and up to \$50,000 for a physician. Because of this looming threat, some hospitals have gone to great lengths to place multiple barriers in front of the list of standard emergency department charges.

While price transparency is likely to remain a major issue in the future of emergency medicine, patients will continue to receive emergency care regardless of their ability to pay. Without major changes to the structure of the health care system, it is unlikely that billing practices in the ED will become any simpler in the foreseeable future.

As emergency physicians, we must continue to advocate for our patients and strive not only to provide high quality care, but cost-effective care, too. ★

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Advocating to Make Emergency Medicine the Best Specialty It Can Be

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EMRA helps make you *the best doctor you can be*, through our amazing on-shift educational resources like the EMRA Antibiotic Guide, *the best leader you can be* by funding more than 100 medical student and resident leadership positions to shape the future of our specialty, and helps *make EM the best specialty it can be*, through our unrivaled advocacy. With so many exciting initiatives going on, I wanted to share some of them with you.

Supporting Emergency Physicians' Autonomy

The EM community was shocked when the anesthesiologists released guidelines stating that ketamine and propofol, two of our most commonly administered sedation agents, should only be provided by qualified anesthesia providers.¹ EMRA banded together with ACEP and 8 other medical specialty societies — including the gastroenterologists, oral surgeons, and cardiologists — to create an Unscheduled Procedural Sedation Consensus Practice Guideline to help prove to hospital administrators and regulators alike that these agents can and should be administered by emergency physicians and other specialists.

Standing up for Residents' Scholarly Activity

The ACGME requires all residents to perform “scholarly activity.” Because emergency medicine has always been innovative, it has adopted a broad definition of what counts toward this requirement. In addition to peer-reviewed research articles, both EMRA and the ACGME’s Review Committee for Emergency Medicine have supported non-peer reviewed work such as podcasts and case reports, conference presentations, and textbook chapters.² In addition, EMRA also believes curriculum development and national committee leadership roles satisfy the requirement by enriching our training.³ However, an upcoming paper by the SAEM Research Directors Interest Group argues for a narrower definition of what constitutes scholarly activity, so EMRA joined forces with CORD and ACOEP-RSO to stand up for residents and will publish a rebuttal.

Protecting Core Faculty

Historically, the ACGME required EDs to grant EM core faculty protected time to teach and mentor residents.⁴ However, the newest proposed Common Program Requirements remove that protection.⁵ EMRA believes this could have disastrous consequences for resident scholarly activity and mentorship, as well as faculty and resident wellness. That’s why we teamed up with ACEP, CORD, SAEM, AAEM, AAEM/RSA, and AACEM to oppose this change. The ACGME decided to reconsider this issue, and while we’re still waiting for the final verdict, we remain hopeful.⁶

Supporting Gun Violence Research

After the tragic death of Dr. Tamara O’Neal, an emergency physician who was gunned down at her hospital, EMRA spoke out about the need for more gun violence research. We also joined 14 national medical societies across multiple specialties to donate thousands of dollars to supporting firearm injury prevention research through the American Foundation for Firearm Injury Reduction in Medicine (AFFIRM).⁷

Promoting Women in Emergency Medicine

In October, a group of motivated residents identified a structural disadvantage for female EM residents: the family leave policy. Unlike OB/GYN, Internal Medicine, Pediatrics, Radiology, Anesthesiology, and Dermatology, EM residents are not allowed to carry over unused family leave weeks from year to year.⁸ This imposes undue burdens upon residents, particularly those having children. After the issue was raised, EMRA created a joint task force with the American Board of Emergency Medicine (ABEM), and plans to offer a solution for consideration at the next EMRA Representative Council meeting in April. EMRA also sponsored the FemInEM Idea Exchange and is committed to promoting diversity and equity in our field.

Looking to the Future of EM

Historically the need for board-certified emergency physicians far outstripped the number of them. But with the dramatic growth in the number of EM residencies during the past decade and the proliferation of Advanced Practice Providers (APPs) in EDs, that might start to change. EMRA cares deeply that today’s residents will have fulfilling opportunities in the future, so we partnered with ACEP and world-renowned Health Workforce researchers to create an Advanced Practice Providers Task Force to help define the scope of practice for APPs in EM, as well as an EM Workforce Task Force to project the supply, demand, and opportunities for emergency physicians in 10-20 years. While we don’t yet know what the future holds, we’re making sure residents have a seat at the table and won’t hesitate to stand up for our members.

If you want to learn more about these or any other issues, or if you’d like to be more involved, feel free to email me at president@emra.org. ★



Facing the Music

How to Manage MDMA Toxicity

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You are working a regular weekend shift when suddenly you get an influx of young patients presenting to your emergency department with altered mental status (AMS), agitation, tachycardia, hypertension and elevated core body temperatures. The patients all have similar physical findings. They all have temperatures greater than 106° F, are tachycardic into the 120s, and have elevated blood pressures 180/90s. You find out they're all coming from the same music festival.

It is claimed they all consumed methylenedioxymethamphetamine (MDMA) — better known as Molly and Ecstasy.

Is MDMA Dangerous?

If you ask people who frequent the music scenes (festivals, concerts, clubs), they'll tell you MDMA is a safe drug. It gives you a "safe" high. It is known for its empathogenic, euphoric, and stimulant effects. However, since MDMA is a substituted amphetamine it can lead to sympathomimetic

toxicity, serotonin syndrome, and possible multi-organ failure from severe hyperthermia. There also has been adverse events stemming from adulteration. Often, other substituted amphetamines such as cathinones (bath salts) are found in Molly or Ecstasy (X), which may have greater sympathomimetic effects compared to MDMA. Websites such as www.dancesafe.org or www.pillreports.org publish adulterants found in MDMA in various cities as a warning for potential users. Death because of MDMA or presumed MDMA is most commonly tied to multiorgan failure from hyperthermia with core body temperatures > 106° F.

What Are the Clinical Effects?

Think of the stereotypical feelings and behavior of rave-goers: feel really good, hyper-awake, super-sexual, and free. These can all be associated with the effects of MDMA — feelings of euphoria, wakefulness, intimacy, sexual arousal, and disinhibition. These symptoms are primarily caused by an increased release of serotonin, in addition to increased availability of dopamine and epinephrine and inhibition of their reuptake.

MDMA has about one-tenth the CNS stimulant effect of amphetamine and sympathomimetic effects are mild in low doses. However, during large exposures, the clinical presentation is similar to that of other amphetamines, and deaths can result secondary to hyperthermia. Also, importantly, significant hyponatremia can occur with its use, leading to seizures. MDMA and its metabolites increase the release of antidiuretic hormone (ADH). Furthermore, substantial free water intake combined with sodium loss from physical exertion from dancing in a hot environment may exacerbate the development of hyponatremia. Other complications include rhabdomyolysis, acute kidney injury (acute tubular necrosis), cardiac dysrhythmias, intracranial hemorrhage, and hepatic failure.

How Can I Diagnose This?

The choice and extent of diagnostic tests should be guided by the history and physical examination. Qualitative urine immunoassays (ie, urine drug screens) are available for amphetamines but have limited utility in managing acute toxicity. The rate of false-positive and false-negative results are high. For example, many cold preparations such as Sudafed Triple Action, Advil Cold and Sinus, Theraflu Max-D Severe Cold and Flu, and Aleve-D Sinus and Cold contain pseudoephedrine, which is structurally similar and may cross-react with the immunoassay. Even a true-positive result only means the patient has used certain amphetamines within the past several days and does not distinguish remote from acute use. False-negative results may occur with certain amphetamines such as MDMA and cathinones, which may not adequately cross-react with the immunoassay.

The gold standard for drug testing, gas chromatography–mass spectrometry analysis, can be used to identify amphetamine analogs present in urine or blood, but it is costly and the results can take several days. As with a lot of diagnoses that are tox-related, a good history and physical is key to diagnosis.

What Is the Treatment?

The general approach to any poisoned patient is stabilization of airway, breathing, and circulation - and the approach to MDMA is no different. If a patient is in respiratory failure or requires airway protection from MDMA, standard medications for rapid sequence intubation can be used. Circulation abnormalities from MDMA can include tachycardia and hypertension, which are treated with parenteral benzodiazepines.

Hyperthermia treatment is of the utmost importance. There are multiple ways to treat such elevated temperatures. If possible, immersion in an ice bath should be performed for rapid cooling. Benzodiazepines should be used to control psychomotor agitation and shivering. Cyproheptadine may be used for signs of serotonin syndrome. Ultimately the goal is to decrease the temperature as quickly as possible

because the human body simply cannot survive such high temperature for long.

Is There a Role for Gastrointestinal Decontamination?

Typically, MDMA ingestions are not in large amounts that require removal or absorption from the GI tract. In addition, many of these patients are at risk of not protecting their airway, so GI decontamination is typically contraindicated. If patient is seizing it is important to figure out if it's because of hyponatremia; if it is, then a hypertonic 3% saline bolus (100 mL) is appropriate; if not, then treat with benzodiazepines.

There is no reversal agent for MDMA, and treatment is primarily supportive. Severely ill patients with seizures, hyponatremia, or hyperthermia with multi-organ dysfunction should be admitted to an intensive care setting. If symptoms are mild and patient is improving, then several hours of observation and discharge is appropriate as well. ★

Take-Home Points

- MDMA is an exceedingly popular drug at music festivals and events, so it is very likely that if a festival is coming to town, you will be seeing toxicity from this recreational drug.
- MDMA intoxication can cause severe life-threatening pathologies like hyperthermia, profound hyponatremia, and sympathomimetic toxicity.
- Human body cannot tolerate temperatures greater than >106° F for prolonged periods of time. Rapid temperature reduction with either ice bath immersion or cool water mist with fanning might be a possible solution.
- MDMA can cause profound hyponatremia leading to lethargy and seizures. A bolus of 100 mL 3% hypertonic saline may be appropriate; consult your local Poison Control Center. ★

Deep Dive into the Evidence: Steroids in Septic Shock

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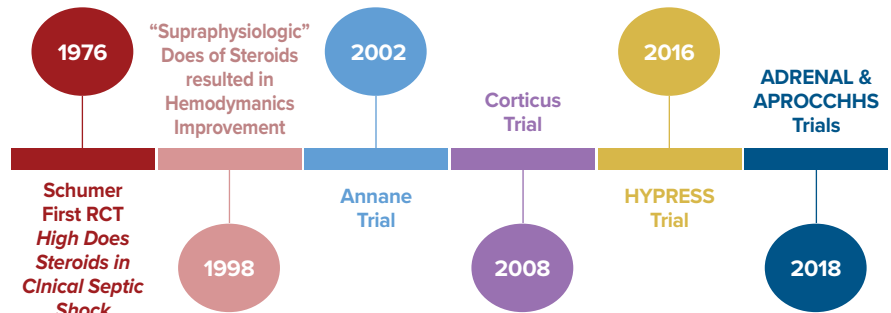
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Sepsis is a major global health issue with a high mortality rate ranging from 30-45%. Septic shock is a state of dysregulated immune response to an infection which results in metabolic and cellular abnormalities, organ dysfunction, and cardiovascular instability.¹ The use of exogenous steroids in septic shock is a controversial issue with inconsistent data on mortality to date. Steroids are thought to modulate the immune response in septic shock patients. The body relies on the hypothalamic-pituitary-adrenal (HPA) axis, the autonomic nervous system and the immune system to respond to stress and infection. There is evidence that the HPA axis becomes dysregulated during sepsis. Exogenous steroids have been shown to improve cardiovascular performance and reduce organ failure.² However, it remains unclear whether exogenous steroids improve patient-centered outcomes such as hospital length of stay and mortality.



The first reported prospective, randomized trial of steroids in septic shock was in 1976. In this trial, septic shock patients were randomized to receive 3 mg/kg dexamethasone, or 30 mg/kg methylprednisolone, or placebo.³ Their outcome was favorable towards improved mortality. However, the subsequent trials using high dose steroids failed to show improvement in mortality and suggested increase in adverse outcomes.

In 1998, Bollaert et al. reported that "supraphysiologic" dose of

methylprednisolone (100 mg IV three times daily for 5 days) resulted in significant improvement in hemodynamics of septic shock patients and beneficial effect on survival.⁴ The concept of using supraphysiologic low dose steroid reignited the interest in investigation of the use of steroids in septic shock.

Annane et al. in 2002 conducted a double-blinded, randomized, placebo-controlled trial in France.⁵ The patients were subdivided into ACTH stimulation responders and non-responders.

Annane Trial

Effect of Treatment with Low Doses of Hydrocortisone and Fludrocortisone on Mortality in Patients with Septic Shock

Annane D, S ebille V, Charpentier C, et al. Effect of Treatment With Low Doses of Hydrocortisone and Fludrocortisone on Mortality in Patients With Septic Shock. *JAMA*. 2002;288(7):862-871.

Methods

- Double blinded
- Randomized
- Parallel-groups (corticotropin test responders vs non-responders)
- 19 French ICUs

Intervention

50 mg IV boluses of hydrocortisone q6hrs **AND** one 50 ug fludrocortisone tablet QD **OR** placebo for 7 days

RESULTS

28 day mortality in septic shock patients with relative adrenal insufficiency (non-responders to corticotropin test)

Hydrocortisone+Fludrocortisone: 60/114 deaths (53%)

Placebo: 73/115 deaths (63%)

Secondary Outcomes	Steroid	Placebo
Mortality at end of ICU stay	58%	70%
Mortality at 1 year follow up	68%	77%
Vasopressor Therapy discontinuation at 28 days	57%	40%
Median time to Vasopressor Therapy Discontinuation	7 days	10 days

THE BOTTOM LINE

There was no significant effect of steroids on mortality in responders, but among nonresponders (patients with septic shock and adrenal insufficiency) low dose steroids do reduce 28 day mortality and vasopressor dependence time. The number needed to treat to save one life at 28 days was 7 people.



N=300



N=229



N=70

Nonresponders
Placebo 115
Steroid 114

Responders
Placebo 34
Steroid 36

The authors showed a 28 day mortality and shock resolution benefit. (See Annane Trial figure, p. 8) The Corticus trial in 2008 was a multi-centered, randomized study that also looked into 28 day mortality. **This study showed no mortality benefit but there was a faster resolution of shock.**⁶ The authors also concluded there was an increased risk of superinfection; however, it wasn't statistically significant. (See Corticus Trial figure)

Given the seemingly contradictory findings of the Annane trial compared to the Corticus trial, the practice of using steroid in treating septic shock patients remains controversial. The HYPRESS trial was a multi-centered randomized study in Germany that was designed to examine if steroids would help prevent progression of sepsis into septic shock.⁷ The primary endpoint was the occurrence of septic shock within 14 days or discharge from the ICU. **The authors concluded that the steroids did not prevent the deterioration of sepsis into septic shock. However, the study was underpowered to reach this conclusion.** (See HYPRESS Trial figure)

The two most recent trials on this topic, ADRENAL and APROCCHSS, came to seemingly conflicting conclusions as well. ADRENAL trial was an international, double-blinded RCT which enrolled 3,800 patients.⁸ The authors investigated whether continuous IV infusion of hydrocortisone for 7 days versus placebo would reduce mortality in septic shock. The primary outcome was 90 day mortality. **Despite no difference in mortality among the two groups, the authors found important**

CORTICUS Trial

Hydrocortisone Therapy for Patients with Septic Shock

Sprung CL, Annane D, Keh D, et al. Hydrocortisone therapy for patients with septic shock. *N Engl J Med.* 2008;358(2):111-124

Methods

- 52 medical and surgical ICUs in 9 countries
- Randomized, Double-blind, Placebo controlled
- Intention to treat, interim, and post hoc subgroup analyses

Intervention

50 mg of IV hydrocortisone q6hrs **THEN** tapered **OR** placebo

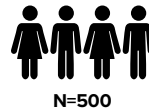
RESULTS

No significant difference in 28-day mortality in patients who did not have a response to corticotropin was identified.

Hydrocortisone: 49 of 125 died (39.2%)

Placebo: 39 of 108 died (36.1%)

Secondary Outcome	Steroid	Placebo
Time to Reversal of Shock (all patients)	3.3 days	5.8 days
Time to Reversal of Shock (response to corticotropin)	2.8 days	5.8 days
Time to Reversal of Shock (no response to corticotropin)	3.9 days	6.0 days



THE BOTTOM LINE

Hydrocortisone did not improve survival or reversal of shock in patients with septic shock, although reversal of shock was faster in those who received hydrocortisone. There was a non-statistically significant increase in the number of episodes of superinfection and hyperglycemia in the hydrocortisone group as compared to placebo.

HYPRESS Trial

Effect of Hydrocortisone on Development of Shock Among Patients With Severe Sepsis

Keh D, Trips E, Marx G, et al. Effect of Hydrocortisone on Development of Shock Among Patients With Severe Sepsis: The HYPRESS Randomized Clinical Trial. *JAMA.* 2016;316(17):1775-1785.

Methods

- Multicenter; 34 ICUs in German Community and University Hospitals
- Placebo-controlled
- Double-blind, randomized controlled trial

Intervention

IV hydrocortisone 200 mg/day for 5 days **THEN** sequentially tapered for 5 days **OR** placebo

RESULTS

No significant difference in development of septic shock within 14 days of treatment initiation was identified.

Hydrocortisone: 36 of 170 developed septic shock (21.2%)

Placebo: 39 of 170 developed septic shock (22.9%)

ADVERSE EVENTS

Hyperglycemia (Insulin administration was not significantly different between two groups)

****Secondary infections, weaning failure, muscle weakness, hypernatremia, or other adverse events were not significantly different between treatment groups**



THE BOTTOM LINE

A continuous infusion followed by tapering of hydrocortisone did not prevent severe sepsis to septic shock progression within 14 days of treatment initiation when compared to placebo.

differences in secondary outcomes. The hydrocortisone group had significant benefits in shock reversal, increased ventilator free days, decreased length of stay in the ICU, and fewer blood transfusions. While the hydrocortisone group had increased adverse events compared to placebo most were clinically insignificant and overall hydrocortisone appears fairly safe. (See *ADRENAL Trial figure*)

APROCCHSS trial, on the other hand, was a multi-centered, double blinded French RCT that enrolled 1,241 patients in septic shock.⁹ The study looked into 90 day mortality benefit in patients who were given hydrocortisone plus fludrocortisone versus placebo. **The study showed a benefit in 90 day mortality along with benefits in secondary outcomes such as vasopressor and ventilator free days, and faster time to shock resolution.** It is important to note that at an interim analysis of the ADRENAL study at roughly similar number of enrolled patients as APROCCHSS trial, there was evidence of mortality benefit. Perhaps if the APROCCHSS trial has been expanded further we would no longer see the mortality benefit. (See *APROCCHSS Trial figure*).

Steroid or No Steroid? Should we give steroids for septic shock patients as emergency physicians? Is it a reasonable therapy? If so, when should we give it? There is no universal consensus when it comes to the role of steroids in septic shock patients. Despite seemingly contradictory outcomes of these studies, there is a consistent trend among the secondary outcomes. Steroids appear to lead to shock reversal, decreased vasopressors need, increased ventilator free days, and less time in the ICU. These outcomes are patient-centered and important in reducing critical care burden within the hospital. According to the data that is available so far, it appears that steroids administration would hasten resolution of septic shock. It remains unclear whether steroids improve mortality in septic shock patients. There is also a consistent trend throughout the data available

that steroids are fairly safe with most of the adverse outcomes being non-patient centered. Given the safety profile of steroid administration to septic shock patients and its potential benefit in hastening shock resolution, perhaps it is reasonable to consider steroids in management of septic shock patients. ★

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ADRENAL Trial

Adjunctive Glucocorticoid Treatment in Critically Ill Patients with Septic Shock

Venkatesh B, Finfer S, Cohen J, et al. Adjunctive Glucocorticoid Therapy in Patients with Septic Shock. *N Engl J Med.* 2018;378(9):797-808.

Methods

- International, pragmatic, double-blind, parallel-group RCT
- Intention to treat, subgroup, and sensitivity analysis

Intervention

Continuous IV hydrocortisone 200 mg/ day **OR** placebo

RESULTS

No significant difference in 90-day mortality was identified in the 6 prespecified subgroup analyses (sex, admission type—medical/surgical, catecholamine dose, site of sepsis, APACHE II score, and time from shock onset to randomization)

Hydrocortisone: 511 of 1832 died (27.9%)

Placebo: 526 of 1826 died (28.8%)

Secondary Outcomes	Steroid	Placebo
Time to Resolution of Shock	3 days	4 days
Time to Discharge from ICU	10 days	12 days
Duration of Mechanical Ventilation	6 days	7 days
Blood Transfusion	37%	41.7%



THE BOTTOM LINE

Continuous infusion of hydrocortisone did NOT result in lower 90-day mortality. Steroid use led to more rapid resolution of shock, shorter inpatient ICU time, and lower incidence of blood transfusion.

APROCCHSS Trial

Hydrocortisone Plus Fludrocortisone for Adults with Septic Shock

Annane D, Renault A, Brun-Buisson C, et al. Hydrocortisone Plus Fludrocortisone for Adults with Septic Shock. *N Engl J Med.* 2018;378:809-818.

Methods

- Multi-centered, pragmatic, double-blind, parallel study in France.
- Intention to treat, subgroup, and sensitivity analysis.
- 1241 patients in 69 medical and surgical ICUs in 69 countries

Intervention

IV hydrocortisone 50 mg q6hrs **AND** PO fludrocortisone 50 µg **OR** placebo

RESULTS

A significant difference in 90-day mortality was identified with an absolute risk reduction of 6.1%, P=.03, NNT of 17.

Steroid: 264 of 614 died (43%)

Placebo: 308 of 627 died (49.1%)

Secondary Outcomes	Steroid	Placebo
Vasopressor Free Days	15 days	17 days
Vasopressor Free Days	10 days	11 days
Organ Failure Free Days	12 days	14 days
Hyperglycemic Days	3.4 days	4.3 days



THE BOTTOM LINE

Hydrocortisone and fludrocortisone combination showed both a mortality and resolution to shock benefit. The steroid regimen was shown to be safe, with hyperglycemia noted as the most common adverse reaction.

that steroids are fairly safe with most of the adverse outcomes being non-patient centered. Given the safety profile of steroid administration to septic shock

patients and its potential benefit in hastening shock resolution, perhaps it is reasonable to consider steroids in management of septic shock patients. ★



A Blue Baby Comes into Your ED

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Cases

1. A 5-day-old boy presents with central cyanosis, mild tachypnea, and lethargy. SpO₂ was 50-60% and did not improve after 10 minutes of oxygen. Physical exam revealed a grade III/VI systolic murmur at the lower sternal border without rales or wheezing. The liver was palpable and electrocardiogram (EKG) showed right axis deviation, a tall p-wave and right ventricular hypertrophy. Chest x-ray (CXR) showed cardiomegaly with black lung fields.¹
2. A 4-month-old girl, ex-37 weeker presents with two days of increased work of breathing and poor feeding. She has no fever, cough, or diarrhea, but has siblings with colds. At her 2-month check-up, a diastolic murmur was noted but has not been followed up. Mom states she recently spends >40 minutes feeding per breast and appears sweaty afterward. Vitals include a respiratory rate of 60/min and a SpO₂ of 90% on room air. On physical exam, you note respiratory distress with scattered rales, rhonchi, and wheezing. You consider bronchiolitis, but decide to obtain a CXR which shows cardiomegaly and patchy perihilar opacities.²
3. A 7-day-old boy, born full-term at home by a midwife with minimal prenatal care, presents by ambulance with fatigue during feeding and “fast breathing” for 1 day. He appears ashen and limp, with vital signs of 36.2°C, pulse of 198 beats/min, breathing 80 breaths/min on room air at 92% SpO₂. Your differential includes sepsis, metabolic disease, and congenital heart disease. You initially order broad-spectrum antibiotics, fluids, chest radiography, and a bedside transthoracic echocardiogram (TTE) but consider whether empiric prostaglandins (PGE₁) are safe to give prior to receiving the results of the workup.²

As emergency medicine providers, how can we best approach the cyanotic and non-cyanotic infant? The most common categories of lesions that present to the ED are:

- Right-sided obstructive ductal-dependent
- Left-sided obstructive ductal dependent
- Shunting or mixing lesions

However, these are most easily thought of in terms of age and degree of cyanosis. Strobel and Lu created a simple system for the presenting pink, blue, and gray baby (Table 1).³

Presentation to the ED

The approach to an infant with suspected congenital heart disease is multi-faceted. These patients can present with shock, cyanosis, tachypnea, and pulmonary edema. To differentiate central vs peripheral cyanosis in an infant, especially in darker skin tones, examine the gum-lines and tongue for purple-blue hues. Isolated circumoral cyanosis may be an indicator of peripheral cyanosis.⁴ A cyanotic infant differential diagnosis must include:

1. CHD
2. Sepsis
3. Respiratory disorders
4. Hematologic disorders such polycythemia and methemoglobinemia

Respiratory disorders are often difficult to distinguish from cardiac causes in the neonate, especially when a CHD presentation may exclude an audible murmur and may not be reflective of the older child presentation, such as dyspnea on exertion, exercise intolerance, syncope, or abdominal pain. CHD infants instead will exhibit irritability, sweating, crying with feeds, failure to thrive, and “quiet tachypnea.” Alternatively, an infant with a respiratory disorder may have similar features but will generally respond to oxygen and have positive lung field findings on chest radiograph. The most sensitive and specific variables ($p < 0.0001$) for congestive heart failure (CHF) include a history of feeding < 3 oz or spending > 40 minutes per breast, an abnormal respiratory pattern with resting rate >60/min, a diastolic murmur, and hepatomegaly.⁵

The hyperoxia test is another highly effective method of distinguishing respiratory from cardiac tachypnea.⁶ The standard approach tests PaO₂ from serial ABGs before and after 10 minutes of 100% O₂, with a rise above 150 mmHg indicating a respiratory cause. A modified approach measures pulse oximetry before and after 10 minutes of 100% O₂. With tachypnea in suspected CHD, assess differentials of pulse oximetry, pulse, and blood pressure in the pre-ductal right arm, and either leg. A ductal dependent lesion is suggested with a differential in >3% on pulse oximetry, in discrepancy of pulse strength palpation, or in mean arterial pressure (MAP) > 20 mmHg.⁴

PEARL. What is the best objective measure of distinguishing between respiratory and cardiac cause of tachypnea?

The modified hyperoxia test measures pulse oximetry before and after 10 minutes of 100% O₂, and is a rapid alternative compared to serial ABGs of the standard hyperoxia test.

Management and Treatment

Initial diagnostic work-up for suspected congenital heart disease in infants should include EKG, CXR and bedside TTE. A pediatric EKG may show sinus tachycardia, left ventricular hypertrophy, ST-T changes, first degree AV block or inferolateral Q-waves, suggesting ALCAPA.⁶ Classic CXR findings include Snowman sign of TAPVR, the Boot-shaped heart in TOF, and the egg-on-a-string sign in TGA. Of note, a right aortic notch with leftward tracheal deviation is associated with CHD 90% of the time.⁷ A black lung field in a hypoxic neonate with metabolic acidosis suggests a right-sided obstructive lesion, whereas white lung fields suggest pleural effusions in left to right shunts. Finally, bedside TTE will provide a quick

determination structural or functional lesions are present.

PEARL. Can any labs help distinguish worsening heart failure in the setting of respiratory illness?

Consider brain natriuretic peptide (BNP or amino-terminal [NT]-proBNP) for distinguishing between cardiac and respiratory etiology.⁸ In a recent study, a BNP of 95 pg/ml (sensitivity: 0.71, specificity: 0.91, positive predictive value: 0.83) was the optimal cutoff for concern of heart failure. However, note that plasma BNP levels are higher in patients with left ventricular volume overload compared to patients with right ventricular pressure or volume overload.⁹

Following initial stabilization and management, the ED treatment is

clinical presentation guided. In the blue neonate, focus on lowering pulmonary vascular resistance (PVR) for improved pulmonary blood flow (PBF) by providing supplemental oxygen and inhaled nitric oxide (iNO). When a right-sided obstructive lesion with a closing ductus arteriosus is suspected in a cyanotic neonate, start PGE1 and intubate. PGE1 can be started with an initial dose of 0.05 mcg/kg/min and titrated up to a maximum of 0.1 mcg/kg/min q15-20 min.¹⁰ If the patient begins to deteriorate after the initial dose, discontinue the infusion as this could signify pulmonary venous or left atrial obstruction as seen in TAPVR, or an infant with TGA without VSD or TOF.

PEARL. What is the preferred induction agent?

TABLE 1. The Presenting Pink, Blue, and Gray Baby³

	BLUE BABY	GRAY BABY	PINK BABY	BLUE BABY
Age of Presentation	< 2 weeks	< 2 weeks	1-6 months	1-6 months
Pulmonary vs. Systemic Congestion	Poor pulmonary circulation	Poor perfusion and pulmonary circulation	Pulmonary congestion	Obstructed pulmonary blood return
Category	Right obstructive lesion	Left obstructive lesion	Left to Right shunt	Mixing Right to Left shunt
Overlying Presentation	Central cyanosis	Cardiogenic shock	Heart failure	Cyanosis + Heart failure
Physical Exam	Failed hyperoxia test SpO2 < 80%	Differential SpO2, BP, and pulse in RUE vs LE Delayed capillary refill	Hepatomegaly Murmur	Failed hyperoxia test SpO2 < 80% Hepatomegaly
Management and Clues	CXR: Black lungs EKG: RVH	CXR: White lungs EKG: LVH in > 7 days of life, RVH in newborn Bedside Echo: Possible single ventricle	CXR: White lungs EKG: Possible extreme right axis deviation or AV block Bedside Echo: Possible interventricular defect BNP > 100 pg/mL	CXR: White lungs, BNP > 40-100 pg/mL
Treatments	1) PGE1 to shunt left to right across ductus, then intubate 2) Use supplemental O2 and iNO to increase pulmonary blood flow and decrease resistance	1) PGE1 to shunt right to left across ductus, then intubate 2) Minimize O2 and add PPV and iNO 3) Initiate antibiotics 4) Milrinone or dobutamine for afterload reduction 5) IV fluids (10 mL/kg boluses) 6) Epinephrine and dopamine for hemodynamic support	1) Minimize O2 and add PPV or intubate to increase pulmonary vascular resistance 2) Milrinone or dobutamine for afterload reduction and inotropy, 3) Judicious IV fluids (5-10 mL/kg boluses) 4) Often need diuretics to prevent volume overload	1) Add milrinone to increase right to left shunt and inotrophy 2) Minimize O2 and add PPV to increase pulmonary vascular resistance 3) Start diuretics 4) Restrict IV fluids
Lesions	Tricuspid atresia Pulmonary atresia Pulmonary stenosis Ebstein's anomaly	HLHS Coarctation of aorta Interrupted aortic arch Aortic stenosis or atresia	PDA VSD AVM AV canal defect	TAPVR Truncus arteriosus Double outlet right ventricle TGA with VSD or PDA
Lesions exceptions	TGA without VSD (no improvement with PGE1), TOF (presents up to 6 months old; no improvement with PGE1)	ALCAPA (presents up to 6 months old, EKG shows characteristic lateral Q waves or ST elevation anterolaterally)		

Table Legend: SpO2: peripheral capillary oxygen saturation; RVH: right ventricular hypertrophy; LVH: left ventricular hypertrophy; O2: oxygen; iNO: inhaled nitric oxide; BP: Blood pressure; RUE: right upper extremity; LE: lower extremity, BNP: brain natriuretic peptide; PPV: positive pressure ventilation; IV: intravenous; TGA: Transposition of the great arteries; VSD: Ventricular septal defect; TOF: Tetralogy of Fallot; HLHS: Hypoplastic left heart syndrome, ALCAPA: Anomalous Left Coronary Artery from the Pulmonary Artery; PDA: patent ductus arteriosus; AVM: arteriovenous malformation; AV: arteriovenous; TAPVR: Total Anomalous Pulmonary Venous Return; PDA: patent ductus arteriosus.

Etomidate is the agent of choice for patients in decompensated shock. Delayed side effects include seizures and decreased adrenocortical function, however adrenal reactions have been regarded as not clinically significant following a single dose¹¹. Fentanyl is the preferred second line agent. Avoid ketamine in induction if CHD is suspected as it can increase systemic vascular resistance, worsen left to right shunts and lead to cardiovascular collapse.

PEARL. When are prostaglandins indicated?

PGE₁ should be started as soon as possible in neonates with suspected CHD in the first month of life. In infants > 1 mo, they can cause apnea and hypotension. For neonates whose CHD improves with PGE₁ administration, an effect should be seen within about 10 minutes in the blue baby and may take several hours in the gray baby. **NEVER walk away after giving prostaglandin. Have a ventilator present and be ready for intubation.**

For the gray neonate in cardiogenic shock, a left obstructive lesion may also be presenting in shock due to closure of the ductus arteriosus. In the ED, we should attempt to reopen the PDA to improve shunting from right to left and reduce afterload, as well as minimize oxygen consumption, add iNO and PPV. To reduce afterload, we can use milrinone or dobutamine. Use caution in giving milrinone in the hypotensive patient, as it can cause peripheral dilation due to vasodilatory properties.

Treatment goals for the pink infant with left to right shunt and pulmonary congestion involve increasing PVR. Lesions can be caused by a PDA, VSD, and AV canal defect. To decrease PVR, focus on minimizing oxygen levels and add PPV. Afterload should be reduced with milrinone or dobutamine, and they may benefit from diuretics to lessen pulmonary congestion and slow judicious 5-10 mL/kg IV fluid boluses as needed.

Lesions of the blue infant with CHF due to a mixing right to left shunt should focus on increasing PVR by

further increasing the right to left shunt. Where PVR is low, PBF will be excessive and should be restricted with ventilatory manipulations to achieve balanced circulation. Ventilator settings should aim for a SaO₂ of 70-85% with a relatively normal pCO₂ of 35-45 mmHg. PGE should not be given in this instance, as it will increase left to right shunt across the PDA, thereby increasing pulmonary blood flow. Shunt blood away from the lungs by mildly hypoventilating, add PPV and decrease supplemental oxygen. Milrinone will also improve right to left shunting and ionotropy.

CHD can predispose pediatric patients to numerous infections, including infective endocarditis,¹² with the most common being gastroenteritis and bronchiolitis, in parallel with the general pediatric ED population.¹³ Arrhythmias are common as well, with the most common being tachyarrhythmias including atrial flutter, atrial fibrillation, and paroxysmal ventricular tachycardia.^{12,14}

Case 1 Conclusion

This patient presented with central cyanosis at < 2 weeks, indicating a high probability of right obstructive lesion, worsening due to closure of the ductus arteriosus. The lesion is reliant upon the ductus to provide blood flow to the pulmonary artery and as it closes, the infant develops progressive respiratory distress and cyanosis which is not improved with oxygen administration. Management included intubation, increased oxygen to lower pulmonary vascular resistance, and PGE₁. Bedside TTE in this case revealed severe tricuspid regurgitation and functional pulmonary atresia, most commonly seen in neonates with Ebstein's anomaly. This patient will require balloon valvuloplasty or surgical repair.

Case 2 Conclusion

Despite initially suspecting bronchiolitis as the cause of the respiratory symptoms, you are suspicious of the cardiomegaly, history of murmur, and prolonged feeding with failure to thrive. You reexamine the patient and this time note

PEARLS AND PITFALLS

- ✓ Be judicious about oxygen and fluids on CHD patients.
- ✓ Prostaglandins should be given for any patient < 1 month old with suspected CHD, however never walk away once PGE₁ is given without being prepared to intubate.
- ✓ A modified hyperoxia test is preferred for distinguishing between a respiratory and cardiac cause of tachypnea.
- ✓ Etomidate is first line for intubation induction.
- ✓ BNP can be used for distinguishing between a cardiac and respiratory presentation, and a cutoff point of over 95 pg/mL is concerning for heart failure.

hepatomegaly. You order a BNP which returns as elevated, further supporting a diagnosis of CHF. After ordering furosemide, the patient has a significant urine output leading to resolution of her tachypnea and improvement of SpO₂ to 95% on room air. An echo confirms a diagnosis of large VSD with left to right shunt.

Case 3 Conclusion

You further your physical exam with an assessment of a blood pressure differential on the right arm and right leg. The patient has a significant reading of 84/40 in the arm and 60/32 in the leg. As a coarctation of the aorta seems most likely, you start a PGE₁ infusion. Within 10 minutes, you notice improvement in capillary refill while a bedside echo confirms critical coarctation of the aorta. You admit the patient for surgical correction.

Acknowledgements

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Pacemaker Basics

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Can you distinguish between an abnormal EKG and a cardiac device malfunction at a glance, recall common pacer codes, or identify pacemaker problems on an X-ray? As the use of cardiac devices increases,¹⁻² emergency physicians must know how to manage not only patients who may need a pacemaker, but also those who present to the ED with a device already implanted. This article summarizes the indications for pacemaker insertion, evaluating a pacemaker on a chest X-ray, understanding pacer codes, interpreting normal and abnormal EKG findings with paced rhythms, and pacer malfunctions.

Indications for Pacemaker Insertion

Some of the Class I indications for pacemaker insertion per the 2012 ACCF/AHA/HRS guidelines³ include:

- Symptomatic sinus bradycardia
- Symptomatic chronotropic incompetence (failure to achieve 85% of age-predicted maximal heart rate during formal or informal stress test or inability to mount age appropriate heart rate during activities of daily living)
- Third-degree AV block
- Mobitz Type II second-degree AV block
- Recurrent syncope caused by spontaneously occurring carotid sinus stimulation and carotid sinus pressure that induces ventricular asystole of > 3 seconds
- For sustained pause dependent VT, with or without QT prolongation
- CHF with electromechanical ventricular dyssynchrony

Complications from Implantation

Although relatively rare, complications from pacemaker implantation typically occur within the first 6 weeks of placement and include:

- Infection
- Venous thrombosis of the upper extremity
- Superior vena cava syndrome
- Hemothorax
- Pneumothorax
- Pocket hematoma (bleeding associated with facial plane dissection for pocket creation)

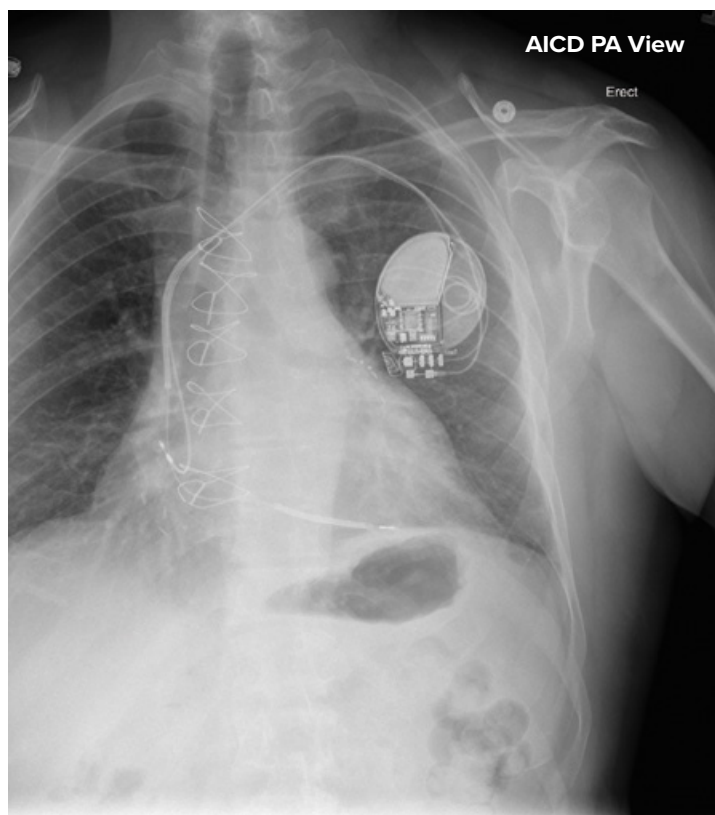
Imaging

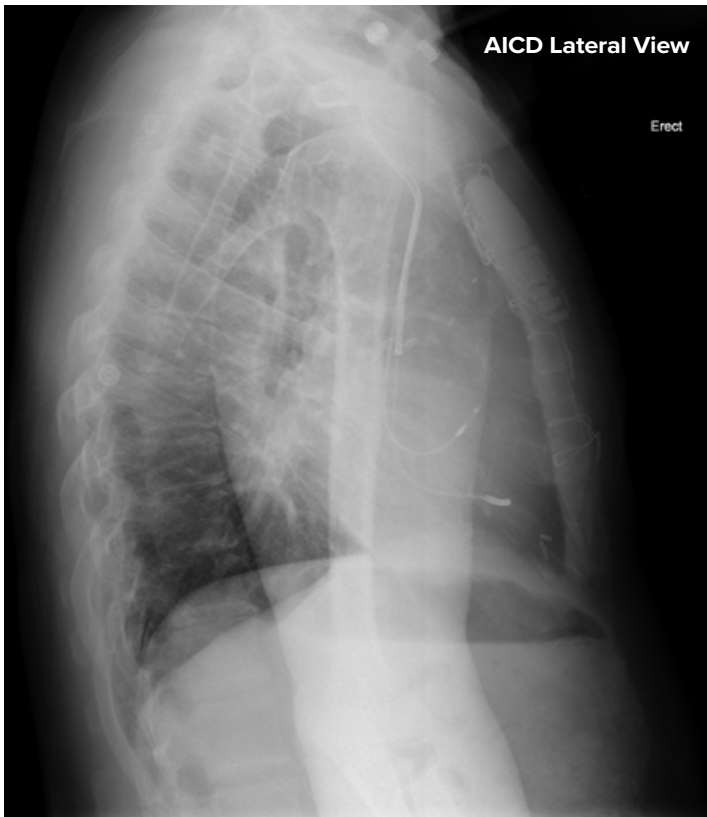
All cardiac pacemakers consist of a pulse generator and at least 1 lead connecting the generator to the myocardium. A chest x-ray should be obtained to evaluate for:

- Manufacturer code, which is found on pulse generator
- Compare pulse generator location with a prior CXR if available
- Leads (number, location, and continuity)
 - A common fracture site is between the first rib and the clavicle
 - AICD will have a thick coil that differentiates it from a pacemaker (as seen in AICD PA and Lateral Views)

Here’s what to look for on each view:

- PA view
 - The J portion of the right atrial lead will be medial
 - Right ventricular leads will point downward with the tip between the left edge of the vertebral column and the cardiac apex
- Lateral view
 - Right atrial lead has a J-shaped appearance as it enters the right atrium and curves upward and anteriorly into the right atrial appendage
 - Right ventricular lead will course anteriorly and caudally
 - Leads placed in the left ventricle or coronary sinus should course posteriorly





Normal ECG Findings with Pacemakers

- Pacing spikes are usually visible on the EKG.
 - Spikes before P waves indicate atrial pacing.
 - Spikes before QRS complexes indicate ventricular pacing.
 - Spikes before both P waves and QRS complexes indicate dual chamber pacing.
- Atrial pacing
 - Usually implanted in the right atrial appendage
 - Typically results in P waves with normal morphology
- Single ventricle pacing
 - Usually implanted in the apex of the right ventricle
 - Typically results in an abnormal but predictable depolarization pattern that mimics a LBBB in the limb leads and anteroseptal precordial leads
- The major difference between an intrinsic LBBB and a paced rhythm is that the QRS will almost always be negative in V5-V6 with a paced rhythm
 - Pacing from the apex also often leads to left axis deviation
- The presence of right axis deviation suggests that the pacing leads are implanted in the right ventricular outflow tract
 - Epicardial pacing leads are sometimes placed over the left ventricle leading to a RBBB
- New RBBB pattern from prior suggests lead displacement
- Biventricular pacing
 - Typically results in narrower QRS than with single ventricular pacing
 - There will often be a dominant R wave in V1 +/- V2

Ischemia in Paced Rhythms

As with an intrinsic LBBB, ventricular pacing results in depolarization and repolarization abnormalities that can confound the EKG's ability to detect an AMI and/or other findings concerning for ACS. In general, evaluate a paced EKG for ischemia as you would a LBBB.

Sgarbossa et al.⁵ found that concordant STE > 1 mm had the highest specificity (94%) for AMI in paced rhythms.

Maloy et al.⁶ showed that discordant STE > 5 mm had a 99% specificity for AMI. It's important to note there were no cases in this study with STE > 1 mm. There are no studies applying the Modified Sgarbossa criteria to paced rhythms.

PEARL. The Sgarbossa criteria are specific but not sensitive for AMI, so the absence of these findings does not rule out an AMI.

Pacer codes (*see table below*) describe the function of the pacemaker. There is a 5-position code, of which the first 3 letters are the most relevant to the EM physician. The following are examples of typical codes:

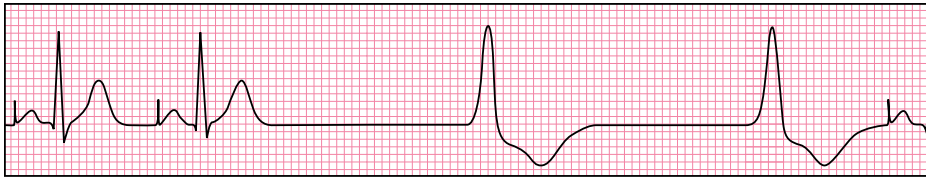
- VOO:** The ventricle is paced asynchronously.
- Ventricle will be paced regardless of native cardiac activity.
- VVI:** The ventricle is paced and sensed.
- Ventricle is paced at a set rate.
 - Pacing is inhibited if pacer senses intrinsic ventricular activity.
- DDD:** Both the atria and ventricle are paced and sensed.
- The pacemaker will either trigger or inhibit pacing based on whether it senses native cardiac activity.
 - Atrial pacing is inhibited if a native atrial beat is sensed.
 - Ventricular pacing is inhibited if a native ventricular beat is sensed.
 - The pacer will trigger a ventricular beat if it senses an atrial beat (either paced or native) and there is no intrinsic ventricular beat within a programmed amount of time.

PACER CODES⁴

Chamber(s) paced	Chamber(s) sensed	Response to sensing	Programmability/Rate modulation	Multisite pacing
A→Atrium V→Ventricle D→Dual chamber O→None	A→Atrium V→Ventricle D→Dual chamber O→None	T→Triggered I→Inhibited D→(Dual triggered and inhibited) O→None	P→Simple programmable M→Multi-programmable C→Communicating R→Rate modulation O→None	A→Atrium V→Ventricle D→Dual chamber O→None

PACEMAKER MALFUNCTIONS

Failure to Pace⁴



General Features

- Paced stimulus is not generated when expected

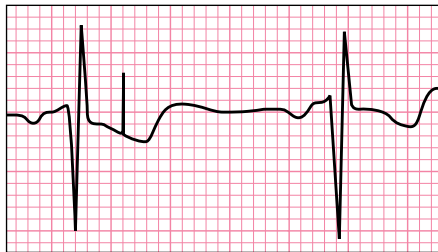
EKG Features

- Decreased or absent pacemaker spikes

Clinical Significance

- Causes: Oversensing (most common), lead fracture, or insulation defect
- Oversensing: Pacing is inhibited by non-cardiac activity (eg, skeletal muscle activity) inappropriately recognized as native cardiac activity

Failure to Sense⁴



General Features

- Sensing refers to the pacer's ability to recognize native cardiac beats
- Pacemaker fails to sense native cardiac activity → asynchronous pacing

EKG Features

- Pacing spikes within QRS complexes

Clinical Significance

- Causes: Lead insulation break, new intrinsic bundle branch block, electrolyte abnormalities, and Class IC antidysrhythmics (eg, flecainide)

Failure to Capture⁴



General Features

- Delivery of pacing stimulus without subsequent myocardial depolarization

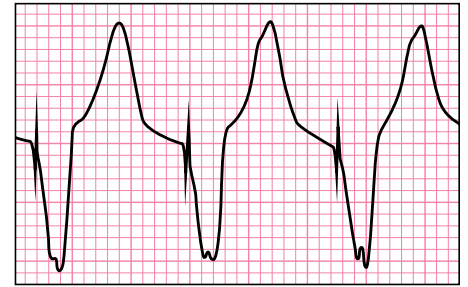
EKG Features

- Absence of depolarization after pacing spikes

Clinical Significance

- Functional etiologies include electrode displacement and wire fracture
- Pathologic etiologies include ischemia/infarct and electrolyte abnormalities (especially hyperkalemia)

Pacemaker-Mediated Tachycardia⁴



General Features

- Re-entry tachycardia with antegrade conduction through the pacemaker and retrograde conduction through AV node
- Only occurs with dual chamber pacemakers

EKG Features

- Paced wide complex tachycardia (may see retrograde P-waves)

Clinical Significance

- Rate limited by pacemaker programming
- Treat with adenosine or magnet

Summary

It's important to gain a solid understanding of pacemaker evaluation and management in the ED. Emergency physicians should have a low threshold to obtain further information from device interrogation and involve Cardiology when indicated. ★

As the use of cardiac devices increases, emergency physicians must know how to manage not only patients who may need a pacemaker, but also those who present to the ED with a device already implanted.

A CASE REPORT

Abnormal Presentation of Pacemaker Interference

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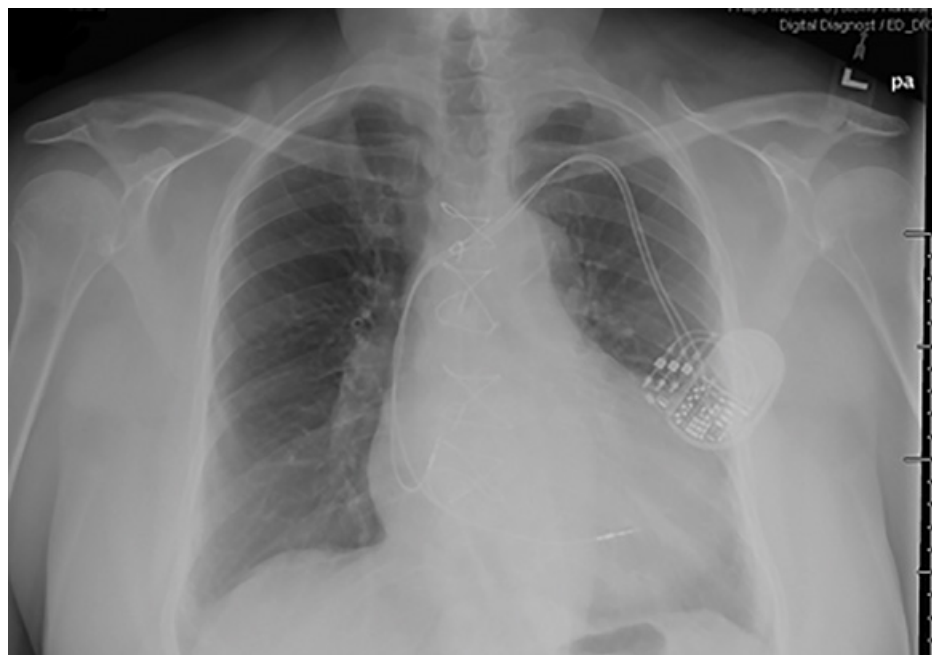
Case Summary

A well appearing 81-year-old female with a past medical history of hypertension and cardiac pacemaker placement 9 months prior for dysrhythmia presents to the ED complaining of hearing a “beeping sound” from her cardiac pacemaker. She states that while in a meeting, she noticed multiple episodes of this beeping sound, without any complaints such as chest pain, shortness of breath, syncope, cough, fever, or chills. She denies any previous complications with her pacemaker, stating all has been well since placement at an outside hospital. Her vital signs are: 97.6 F, HR 66, BP 133/62, SaO₂: 99% on room air, and RR of 16. Her physical exam is unremarkable, with obvious pacemaker to left chest.

Learning Points

Pacemaker malfunctions are not an infrequent reason for an emergency department evaluation. As a result, there is a wide differential into the underlying potential etiologies. Patients can present with problems regarding the pacemaker itself, commonly involving battery failure. Other potential etiologies may be secondary to a regional problem, such as surrounding infection or hematoma. These conditions are more commonly seen directly following placement.

The pacemaker itself can also have problems secondary to lead separation,



dislodgement, or coiling. In rare cases the pacemaker can cause local trauma, leading to thrombus formation or myocardial rupture. When these types of problems occur, they often lead to downstream electrical problems, such as failure to capture or pace.

Thorough history-taking is vital in these patients, as external interference can also occur. The National Institutes of Health recommends against extended exposure in close proximity to objects such as cellular phones, household appliances such as microwaves, high-tension wires, metal detectors, industrial welders, or electrical generators because these objects can disrupt signaling and cause dysfunction. Some recent literature also supports that cellular phones cause no disruption, and that a pacemaker will only alert airport security to a metal device, but have no risk of dysrhythmia. Almost all pacemakers are appropriate for MRI testing, but it is recommended that the patient’s cardiologist is contacted prior to testing. If a patient requires

cardioversion, it is recommended that cutaneous pads are placed anterior-posterior, with at least 8 cm between pads and patient’s pacemaker.

Case Resolution

After it was determined that the patient was stable, requiring no acute interventions, pacemaker interrogation was performed. The generated report from the supplier displayed that the patient’s pacemaker had gone in and out of “magnet mode” multiple times just prior to her arrival. The patient had no acute events of dysrhythmia.

On further questioning, and in an effort to investigate for electromagnetic interference, it was found that the patient was wearing a magnetic nametag over her left chest while in a meeting. Her nametag had already been removed, and her pacemaker was no longer in magnet mode and acting appropriate. After appropriate counseling, she was discharged to follow up with her cardiologist and avoid any potential objects that can cause electromagnetic interference. ★

Eggplant Emoji

EMERGENCIES

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While approximately 49% of us have male genitalia, nearly all emergency physicians are uncomfortable with penile problems and procedures. Moreover, it is not easy obtaining volunteers or male colleagues to be the simulated patient during penile procedure day during grand rounds. Because these emergencies occur so infrequently, emergency physicians typically encounter their first penile aspiration or paraphimosis reduction on an actual patient who needs help emergently. But this is why we became emergency physicians – to save life and “limb.”

For lack of a better term, a “penile fracture” is defined as a tear in the tunica albuginea of the corpus cavernosum, even though there is no anatomical bone within the penis. This “fracture” typically occurs when an external force is applied to an erect penis, leading to immediate pain and rapid detumescence. Mounting intercourse and aggressive masturbation have been discovered as common causes for a ruptured tunica albuginea.¹ The practice of taghaandan is another common cause in Western Iran wherein men forcibly “snap” an erection to force detumescence.²

Eggplant Sign

The eggplant sign is the classic presentation because the penis looks just like the subtropical vegetable due to swelling and extravasation of blood into the superficial tissue of the penis. Ironically, this common distinction has been around long before the invention of emojis. As emergency physicians, there is not much we can do for these patients other than assessing for urethral injury, providing adequate analgesia, and – most important – emergent consultation with urology. If hematuria is present, a cystourethrogram to rule out urethral injury should be completed prior to surgical repair of the penile fracture.

The term priapism is derived from Priapus, the Greek God of fertility, lust, viticulture, and the protector of horticulture (explaining the association

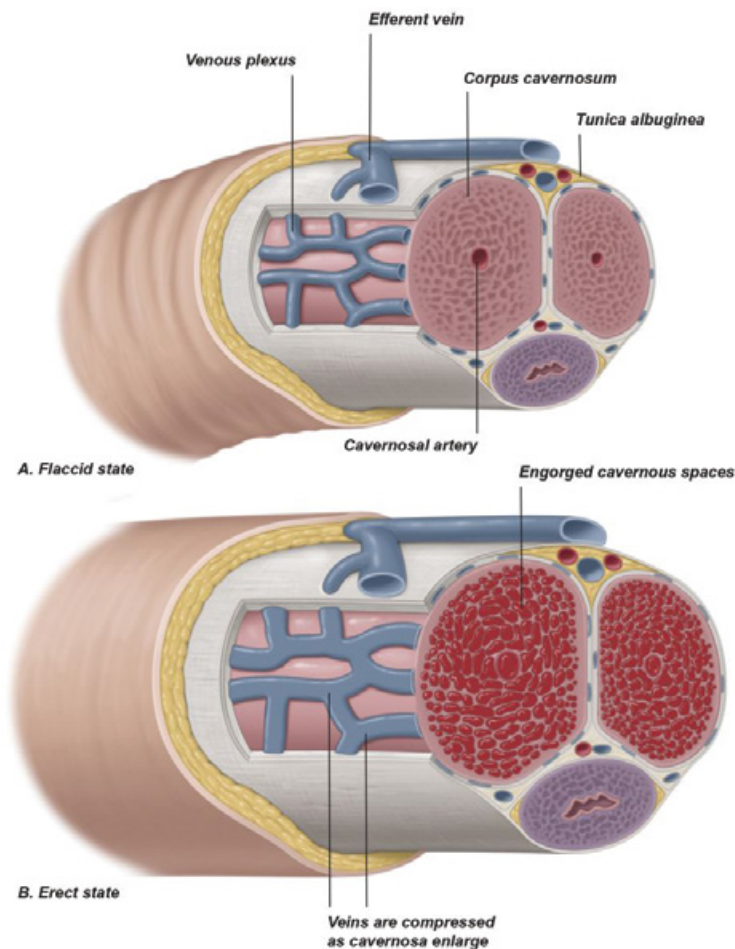


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with eggplants). Priapism is defined as a persistent erection for at least 4 hours in the absence of sexual stimulation. It can be further classified as either ischemic or non-ischemic, and the distinctive features are further clarified in Table 1.

To help distinguish these two types, you can think of ischemic priapism as compartment syndrome of the penis, but please do not attempt a fasciotomy in this case. Stuttering priapism is a subtype of recurrent ischemic priapism that occurs over an extended period of time and is almost exclusively seen in the setting of sickle cell disease.

Non-ischemic priapism is far less common and is most commonly caused by penile or perineal trauma. Non-ischemic priapism is generally self-limiting and resolves within several hours to days. Additional treatments include embolization and surgical ligation, which is beyond the scope of emergency medicine.

Management of Priapism

Ischemic priapism can be managed in the ED with corporal aspiration with or without irrigation and intracavernosal injections of phenylephrine. Aspiration/irrigation involves inserting an 18-22 gauge needle connected to tubing and a 3-way stopcock. A butterfly needle tends to work the best as it provides stability of the needle within the corpora cavernosa. Remove up to 20-40 mL or until bright red arterial blood is aspirated and sterile water can then be injected. For intracavernosal injections, 100-500 mcg of phenylephrine is injected directly into the corpora cavernosa.³ In order to obtain the 500 mcg/mL mixture, mix 0.5 mL of phenylephrine (10 mg/mL) in 9.5 mL of normal saline.⁴ Inject up to 1 mL of the phenylephrine solution every 3-5 minutes for up to one hour until detumescence is achieved.⁵ Less than 1 mL could be injected for children or patients with coronary artery disease. If these techniques are unsuccessful, urology should be

TABLE 1. Characteristics of Priapism

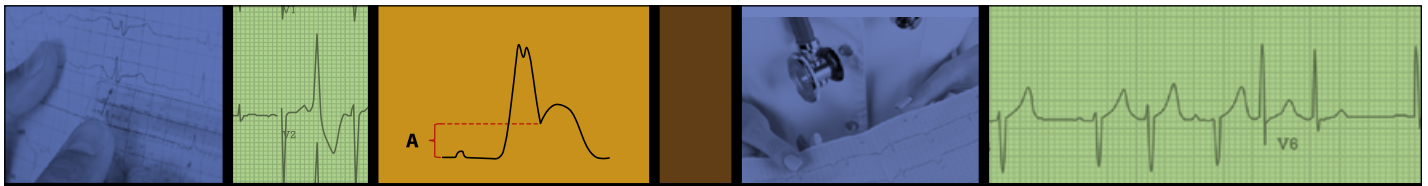
Ischemic	Non-Ischemic
Exquisitely painful with shaft tenderness	Classically nonpainful
Fully rigid shaft with sparing of the glans	Fully or partially erect penile shaft and glans
Corporal Blood Gas: pH<7.25, pO ₂ <30mmHg, and pCO ₂ >60mmHg	Corporal Blood Gas: PH >7.30, pO ₂ >50mmHg, and pCO ₂ <40mmHg

consulted immediately. Patients with sickle cell disease should also receive disease specific treatments such as blood transfusions and exchange transfusions if necessary.

Paraphimosis occurs when the penile foreskin in an uncircumcised male becomes entrapped behind the coronal sulcus and cannot be returned to its normal, protracted position. Emergent reduction of the foreskin must be attempted in order to prevent further injury to the glans. Local skin necrosis is the most common outcome, but untimely management could lead to infarction, gangrene, and two words that no man would ever want associated with their penis: auto-amputation. Analgesia with parenteral opiates, penile dorsal nerve

block, and even procedural sedation should be implemented prior to manual reduction of the foreskin. Various methods to reduce swelling of the glans to assist in foreskin protraction include manual compression or wrapping a compressive dressing such as coban around the glans.⁶ Osmotic agents such as granulated sugar, 50% dextrose, or 20% mannitol could be placed on the glans with application of a compressive dressing to further facilitate swelling reduction.

Overall, penile emergencies are quite uncommon but require rapid treatment. It is imperative that we all routinely familiarize ourselves with these problems and procedures pertinent to the penile emergencies.★



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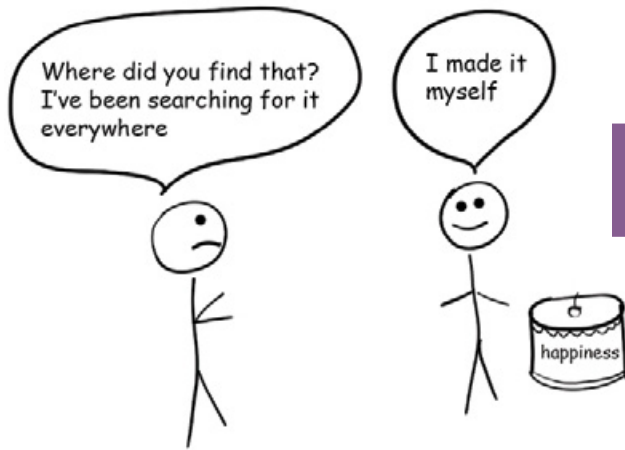


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I used to believe happiness was something you earned. Happiness was an emotion you felt after some good fortune happened to you or after you achieved a goal. Once you achieve all your goals, *then you get to be happy*.

As a pre-medical college student, I thought to myself, “I’ll be happy when I get accepted to medical school.” As a pre-clerkship medical student, it turned into “I’ll be happy when I do well on USMLE Step 1.” During clerkships, I moved on to “I’ll be happy when I match in one of my top 3 programs.” As a resident, I found myself postponing happiness even further by thinking “I’ll be happy when I’m an attending and I actually have more money and time.” That last one got pushed even further into the distance as I debated whether I wanted to pursue a fellowship, as if a fellowship would just delay the start of my happiness even more. This is how I saw happiness on the grand scale of my life.

How did I try to find happiness on a day-to-day basis? It was when I finished a project, the end of a shift, when the waiting room was cleared out (which was almost never), my next day off, the end of a relentless intensive care unit rotation when I could finally go back to my “home” in the emergency department, or the countdown of days until my next vacation. Between each of these “accomplishments” there was happiness, but it was fleeting. When I was far away from reaching any of these “things that made me happy,” I felt exhausted and frustrated that I was working so hard and wasn’t fulfilled. Burnout was a foreign concept I never thought could happen to me, but quickly realized I wasn’t as resilient as I thought.

MAKING MY Happiness

Being happy is a skill; you have to practice it and make it a habit.

Success doesn’t fuel happiness — happiness fuels success.

It turns out happiness is a *cause* of good things in life, and it itself promotes even more happiness. Positive psychology, an evidence-based science that focuses on studying human thoughts, feelings, and behaviors with a focus on strengths,¹ has found that a positive mindset can raise productivity levels, boost well-being, and that happiness promotes success, not the other way around. It can even be contagious; individuals with happy friends and significant others are more likely to be happy in the future.² Research has shown that doctors specifically who were primed to have a “positive effect” performed better at problem-solving and were better at selecting the correct diagnosis, with less anchoring bias than their peers.^{1,3}

You are responsible for your own happiness.

How can we apply this to our lives to reduce burnout and promote resiliency? First, we have to stop relying on purely external factors to validate our own happiness. Good news is that research has shown us that “the good life” can be taught. You already have the tools to improve your own well-being and happiness, both with external actions and internal thoughts.

External

- **Gratitude** is one factor that contributes to happiness. The more we cultivate it, the happier we will be.⁴ Giving hugs and physical affection can also boost overall well-being.⁵ People who intentionally cultivate a positive mood to match an outward emotion they need to display, genuinely experience the positive mood.⁶ People who perform acts of kindness toward others not only get a boost in well-being, they are also more accepted by their peers.⁷
- **Volunteering** on behalf of a cause you believe in improves your well-being and life satisfaction and may even reduce symptoms of depression.⁸
- **Giving** by spending on other people results in greater happiness for the giver.⁹ In fact, spending money on experiences also provides a boost to happiness rather than spending money on material possessions.¹⁰
- **Building** a strong social network improves your well-being. The more social support you have, the happier you are. Work on building positive and supportive relationships.

Internal

- **Improve your mood** with meditation, finding something to look forward to (a movie, a meal), exercising, and spending your money in ways that make you feel fulfilled.
- **Find meaning and purpose in what you do** by being engaged in what you do. You can have the best job in the world, but if you can't find the meaning in it, you won't enjoy it.
- **Be optimistic** by looking for the positivity in your world. Bad things are temporary and not universal. Burnout is the result of a pessimistic attitude toward your job with negative thoughts such as, "This isn't getting me anywhere. I can't handle this. It's never going to get any better."
- **Think of failure as an opportunity for growth** by creating the local and temporary mindset ("I/it will get better"). Remember: You will fail at something. Give yourself the opportunity to move up not despite the setbacks, but because of them.
- **Believe that your actions have a direct effect on your outcomes.**¹¹

YOUR WELLNESS CHALLENGE

Being happy is a skill; you have to practice it and make it a habit. Shawn Achor created a "21 Day Challenge," suggesting picking one of the following 5 habits to do 21 days in a row in order to improve feelings of happiness and optimism.¹² (In fact, doing all 5 can take as little as 30 minutes a day.)

1. *Improve your optimism:* Write down 3 new things you are grateful for every day.
2. *Scan the world for positivity:* Write for a few minutes a day describing a positive experience you had that day.
3. *Increase feelings of social support and commit kindness:* Write a message (email, Twitter, Facebook, carrier pigeon) at the start of every day thanking or praising a member of your team.
4. *Undo the negative effects of multitasking and decrease stress:* Meditate for a few minutes a day, only focusing on your breathing.
5. *Train your brain that your behavior matters:* Exercise for 10 minutes a day.

With as high as 70% burnout rates in emergency medicine,¹³ we need to focus on how to reduce emotional exhaustion. Happiness can help us to become more resilient and improve well-being, which subsequently can also promote success and further happiness. ★



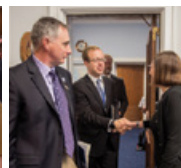
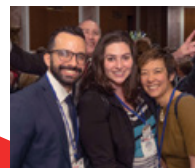
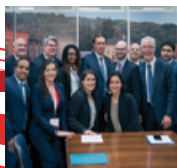
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FOURNIER'S GANGRENE

A Flesh-Eating Infection of the Perineum

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A 54-year-old female with a significant medical history of hypertension, tobacco dependence, and Type II diabetes presents to the ED complaining of right groin pain. She notes fullness to her right labia and upper thigh for the past week, along with subjective fever and chills. She reports a fullness-type sensation moving in her groin and progressively getting worse. The review of systems is negative for any trauma, abdominal pain, vomiting, vaginal bleeding, vaginal discharge, or any history of similar symptoms.

On physical examination, the patient is afebrile, heart rate of 98 bpm, blood pressure of 140/97, respiratory rate of 21 breaths/min, pulse oximetry on room air of 100%, and a weight of 433 lbs. She is in no acute distress, sitting up in bed, and talkative. The cardiovascular and respiratory findings are unremarkable. The patient is found to have moderate induration of the anterior right labia, extending up to the right inferior portion of the mons pubis. No palpable fluctuance, no evidence of skin necrosis, no crepitance, no drainage, and no bullae are noted. Pain appears out of proportion to the physical exam. No other acute findings are found on physical exam.

Given the history and physical exam, code sepsis was activated, antibiotics were initiated (vancomycin, clindamycin, and piperacillin/tazobactam), and general surgery was immediately contacted for suspected necrotizing fasciitis. A complete blood count revealed a neutrophilic leukocytosis of 23.6 and a mild anemia of 12.7 g/dL. On chemistry, she was found to have a hyponatremia of 130 mmol/L, glucose of 188 mg/dL, a blood

urea nitrogen level of 41 mg/dL, and a creatinine of value of 2.30 mg/dL (up from a baseline of 0.70 mg/dL). In addition, she was found to have a lactic acid of 1.5, c-reactive protein of 408 mg/L, and lactate dehydrogenase of 370 U/L. Urinalysis demonstrated cloudy urine, specific gravity of 1.021, 1+ blood, 2+ leukocyte esterase, 11-24 WBC/hpf, and 1+ bacteria. The remainder of the laboratory investigation was unremarkable.

CT abdomen/pelvis without contrast (see Figures 1-3) revealed extensive inflammatory stranding involving the anterior/lateral right pelvic soft tissues with soft tissue gas identified tracking from the lateral right pelvis soft tissues at the level of the iliac crest medially and inferiorly down to the level of the right groin. The inflammatory stranding reached the anterior rectus fascia without intraperitoneal extension. These findings were concerning for necrotizing fasciitis.

The patient was taken emergently to the operating room where she underwent wide excision and debridement of skin, fat, fascia of the right groin and perineum totaling 30 x 15 x 8 cm. She was then transferred to the intensive care unit, intubated, and put on inotropic support. She went back to the OR multiple times over the course of her hospital stay for further wound exploration, washout, debridement, and reconstruction. Anaerobic cultures grew *Prevotella* and *Porphyromonas* species. Her hospital stay was complicated by septic shock and acute respiratory distress syndrome (ARDS); however, she improved and was discharged to subacute rehabilitation on day 24. Her most recent monthly surgery follow-up

revealed a well-healing wound with no complaints from the patient.

Discussion

Necrotizing fasciitis, specifically Fournier's gangrene, is a monomicrobial or polymicrobial severe flesh-eating deep soft tissue infection of the perineum and genitals.¹ This extremely life-threatening and rare necrotizing fasciitis of the perineum carries with it an extremely high mortality rate. Most commonly, Fournier's gangrene appears to have a preference for individuals with a history of diabetes and alcohol abuse. The age of the patient, advancement of the disease, delayed presentation to the ED, and sepsis all contribute to the overall high mortality.

Necrotizing fasciitis progressively destroys muscle fascia and subcutaneous fat as it spreads along the fascia, with the overlying tissue often appearing unaffected by this flesh-eating infection. As a result, this can be a difficult diagnosis to make.⁶ The infection may follow a subacute and progressive course or may explode rapidly over days with multi-system organ dysfunction.



FIGURE 1. CT abdomen/pelvis without contrast

The *British Journal of Surgery* reports that the following classic physical exam findings are only present up to 25% of the time and occur later in the disease process. As such, relying on these signs for a diagnosis can be dangerous.^{3,4}

- Skin necrosis
- Crepitus (subcutaneous emphysema)
- Cutaneous anesthesia
- Bullae
- Severe sepsis/septic shock

Common physical exam findings to help the clinician differentiate necrotizing fasciitis from cellulitis includes:

- Pain out of proportion to physical exam findings
- Absence of lymphangitis
- Indistinct margins of involvement
- Tenderness beyond the location of erythema
- Worsening infection despite antibiotic use.⁴



FIGURE 2. CT abdomen/pelvis without contrast

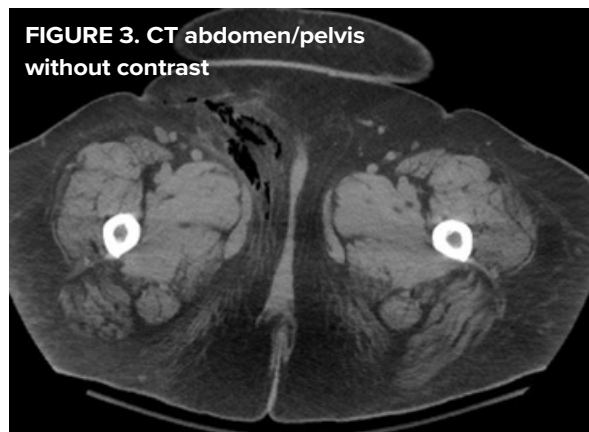


FIGURE 3. CT abdomen/pelvis without contrast

LRINEC Score

A scoring system that is helpful in prognosticating, but not diagnosing, and often helps in aiding clinical decision-making has been the LRINEC score (Laboratory Risk Indicator in Necrotizing Fasciitis). This scoring system utilizes 6 laboratory components (c-reactive protein, white blood cell count, hemoglobin, serum sodium, serum creatinine, and plasma glucose) that are each given a score, and the total score will assess the risk of necrotizing fasciitis. A score of ≥ 6 in the original study was found to have a 96% negative predictive value and a 92% positive predictive value.³ However, the score does have a limited sensitivity, has not been validated, not useful in the early recognition of necrotizing fasciitis, and there are reported cases in the literature of necrotizing fasciitis with a LRINEC score of 0.³ While necrotizing fasciitis can

present with the previously mentioned lab abnormalities, interpreting the score with caution and maintaining a high index of suspicion are keys to identification.

Plain radiographs can show subcutaneous emphysema; however, this finding is only highly specific to *Clostridia* species.⁵ CT is more sensitive but does lack specificity as multiple different disease processes can reveal fluid collections, tissue stranding, and gas formation. If suspicion is high for necrotizing fasciitis, immediate surgical consultation is necessary, even without imaging, as the gold standard for the diagnosis is surgical intervention.

Categorizing Necrotizing Fasciitis

Necrotizing fasciitis is divided into groups based on the type of bacteria. Type 1 infections are polymicrobial in nature and consist of gram-negative organisms (*Klebsiella*, *Escherichia coli*, *Proteus*, *Enterobacter*, and

Pseudomonas) and anaerobes (*Clostridia* species, *Bacteroides*, *Fusobacterium*, *Peptostreptococcus*, and *Prevotella*).^{3,7} Type 1 infections are the most common and are typically found in patients with diabetes mellitus, obesity, chronic liver disease, recent surgery, peripheral vascular disease, omphalitis, and immunosuppression, to name a few.³ The synergistic effect of the bacteria can result in fulminate gangrene, multi-system failure, and death. Type 2 infections are monomicrobial in nature and often caused by *Streptococci* and/or *Staphylococci*. Type 2 infections are typically found in healthy younger patients with a history of surgery, burn, trauma, laceration, or intravenous drug use.^{3,7} Type 3 infections are caused by gram-negative bacteria, specifically marine organisms like *Vibrio* species. These types of infections often occur after a break in the skin or an open wound is exposed to either saltwater or freshwater.^{3,7}

Treatment

Antibiotic therapy should be broad to cover activity against gram-negative, gram-positive, and anaerobic bacteria. A commonly used acceptable regimen for the emergency physician will include broad spectrum coverage for gram positives, gram negatives, and anaerobes (vancomycin + piperacillin/tazobactam + clindamycin). After the results of gram stain, culture, and sensitivity, the antibiotic coverage can then be narrowed.

Summary

For the clinician, having a high index of clinical suspicion, prompt recognition, initiating broad-spectrum antibiotics early, and immediate surgical consultation are of paramount importance in potentially saving a life. Despite aggressive treatment, the mortality rate still ranges from 12-45%, and without surgery, 100%.²

Case Conclusion

This case report presents a patient diagnosed with Fournier's gangrene who received prompt treatment and surgical intervention and endured a complicated hospital stay, including septic shock and ARDS; however, she made a complete recovery. ★

Blunt Cerebrovascular Injuries

A REVIEW

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Blunt injuries to the carotid or vertebral arteries, collectively referred to as blunt cerebrovascular injuries (BCVI), may be clinically silent at first, but the implications can have devastating effects – including a mortality rate approaching 43%.

Introduction

Blunt injuries to the carotid or vertebral arteries, collectively referred to as blunt cerebrovascular injuries (BCVI), may be clinically silent at first, but the implications can have devastating effects for patients. BCVI is associated with increased incidence of stroke if not identified in a timely manner and treated appropriately. Mortality may even reach as high as 43%.¹ The purpose of this review will be to discuss the relevant anatomy, imaging modalities, grading of injuries and subsequent treatment.

Anatomy

The cerebrovascular system can be divided into the anterior and posterior circulation, supplied by the carotid and vertebral arteries respectively. The common carotid arteries (CA) bifurcate into the internal carotid artery (ICA) and external carotid artery (ECA) at approximately the level of the third and fourth vertebral bodies. The ICAs are then further divided into four segments based upon their anatomic location: cervical, petrous, cavernous, and cerebral. The significance of these segments relates to the potential for injury with certain skull fracture patterns due to the anatomical constraints of the ICA. The vertebral arteries (VA) travel in the vertebral foramina of the sixth through second cervical vertebrae, and are also divided into four segments based on their anatomic location in relation to the cervical spine and skull base.²

Incidence, Injury Pattern, and Screening

BCVI is a relatively rare diagnosis, but this does not take away from the importance of considering it during the initial evaluation of all trauma patients. One study has found the incidence of BCVI to be only 0.64% of admitted blunt trauma patients, but other studies have reported an incidence as high as 2.5%. The CAs or VAs were equally likely to be involved, with mortality being higher when the CAs were involved.¹

Injuries are felt to be caused by cervical hyperflexion/extension causing a stretching of the vessels, but injuries may also occur with fractures/dislocations of the cervical vertebrae lacerating the vessels or direct cervical blows.³

Due to the low incidence of BCVI, emergency physicians must have a high suspicion in order to recognize this dangerous diagnosis with these injury patterns. Patients may present to the ED initially with symptoms of stroke due to BCVI; however, more commonly there is a latent period with symptoms developing 10-72 hours after injury.⁴

Various trauma institutions have attempted to develop screening guidelines, with one of the most highly utilized being the Denver Grading Scale for BCVIs.⁵ The Denver group has developed recommendations to screen for BCVI in high-risk patient populations, identified in Table 1. Of these risk factors, skull base fractures have been identified as the highest risk traumatic injury associated with BCVI.⁶

Imaging

Many imaging modalities exist to help evaluate BCVI including CT-angiography (CT-A), arteriography, magnetic resonance angiography (MR-A), and duplex ultrasonography. CT-A has been recommended as the best screening modality for BCVI in trauma patients. Arteriography can provide a definitive diagnosis; however, it is invasive, has limited availability, and is not ideal to obtain from the ED. CT-A is not perfect, with a sensitivity of approximately 80% and specificity of 97%, but it has been deemed the most cost-effective screening strategy to prevent strokes. MR-A and duplex have no data to support their use as screening modalities for BCVI.²

Pathophysiology and Stroke

Injuries to the cerebrovascular system typically cause intimal disruption regardless of the mechanism. This intimal tear then becomes a source for platelet aggregation that has the potential to cause downstream effects such as embolic stroke or vessel occlusion.⁴ A grading scale has been developed to provide a description of injury as well as categorize the associated stroke risk, seen in Tables 2 and 3.

Treatment Options

Due to the risk of stroke in these patients, treatment with antithrombotics, operative repair, or endovascular stenting is important and may decrease the stroke rate considerably. Antithrombotic agents,

primarily heparin, are utilized to promote clot stabilization with prevention of further vessel thrombosis and promotion of resolution via intrinsic fibrinolysis and are extremely effective at preventing stroke.⁵ Difficulty arises, however, when many of these patients have multisystem trauma and are not amenable to systemic anticoagulation therapy due to bleeding risks.

Operative therapy may not be an option in patients who have vascular injuries in inaccessible areas such as the carotid canals or vertebral foramen; therefore, endovascular stenting may also be considered.

Grade I injuries only require antithrombotic treatment while Grade V injuries require operative repair, via open or endovascular techniques. Grade II-IV injuries may be considered for operative/endovascular repair or antithrombotic treatment.

Arguments exist as to which treatment is best, with questionable benefit to endovascular stents due to many of the associated complications such as post-stenting stroke, stent occlusion, or procedural complications. One prominent study by Burlew et al from the Denver Health Medical Center finds that administering heparin for BCVI is an effective treatment for stroke prevention and that routine endovascular stenting provides no

TABLE 2. Injury Grading Scale⁴

Grade	Injury
I	Intimal irregularity or dissection < 25% luminal narrowing
II	Dissection or intraluminal hematoma with ≥ 25% luminal narrowing, intraluminal clot, or visible intimal flap
III	Pseudoaneurysm
IV	Complete occlusion
V	Transection with active extravasation

additional benefit, but should be reserved instead for rare symptomatic BCVI or patients with a markedly enlarging pseudoaneurysm.⁸ In a study of asymptomatic patients with BCVI treated with heparin infusion, stroke rate was decreased to 0.3%.⁹ Recommendations have been made to treat grade I-IV injuries with low-dose anticoagulation via heparin infusion. Antiplatelet agents, such as aspirin with or without clopidogrel, have also been suggested and potentially have similar efficacy in stroke prevention; however, there have not been large studies to support this currently.

Many low-grade injuries (ie, Grade I and II) heal within 7-10 days, therefore early repeat CT-A is recommended to evaluate, as this may allow for discontinuation of antithrombotics at that time. Otherwise, treatment is continued typically for 3-6 months, at which point re-imaging is recommended to evaluate for resolution.⁵

TABLE 3. Stroke Risk⁵

Injury	Grade	Stroke Rate (%)
Carotid Artery	I	3
	II	14
	III	26
	IV	50
	V	100
Vertebral Artery	I	6
	II	38
	III	27
	IV	28
	V	100

Conclusion

BCVI is a relatively rare condition associated with significant morbidity and mortality that can be prevented if identified early. Emergency physicians are incredibly important in diagnosing this disease and must know which trauma patients require advanced imaging so they may identify BCVI early in the hospital course. Key factors in identification involve recognizing high risk injury patterns and utilizing the appropriate screening imaging modality. With prompt diagnosis and treatment, trauma patients with BCVI may be spared from long-term neurologic disability. *

BCVI is a relatively rare condition associated with significant morbidity and mortality that can be prevented if identified early.

TABLE 1. High Risk Injury Patterns⁷

Signs/Symptoms of BCVI	Risk factors for BCVI
<ul style="list-style-type: none"> • Arterial hemorrhage from neck/nose/mouth • Cervical bruit in patient < 50 years old • Expanding cervical hematoma • Focal neurologic defect • Neurologic defect inconsistent with head CT findings • Stroke on CT or MRI 	<ul style="list-style-type: none"> • High energy transfer mechanisms • LeFort II or III fractures • Mandible fracture • Complex skull fracture/basilar skull fracture/occipital condyle fracture • Closed head injury with GCS < 6 • Cervical spine fracture, subluxation, or ligamentous injury at any level • Near hanging with anoxic brain injury • Clothesline type injury or seat belt abrasion with significant swelling, pain or altered mental status • Traumatic brain injury with thoracic injuries • Scalp degloving • Blunt cardiac rupture • Upper rib fractures

From Bedside to Curbside

Prehospital Clinical Research Update

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PARAMEDIC-2 Trial

The efficacy of advanced airways, drug therapy, and even traditional advanced life support (ALS) care in cardiac arrest have been challenged in recent years. Now, the use of epinephrine is again being questioned.

A July 2018 *New England Journal of Medicine* article by Perkins, *et al.* studied epinephrine use in 8,014 out-of-hospital cardiac arrest (OHCA) cases in the United Kingdom, randomized to either epinephrine or placebo.¹ The Prehospital Assessment of the Role of Adrenaline: Measuring the Effectiveness of Drug Administration in Cardiac Arrest (PARAMEDIC-2) trial is the first randomized, controlled trial in recent years to reexamine the efficacy of epinephrine in OHCA. The article first reviews the theory behind the use of epinephrine: constricting arterioles and increasing aortic diastolic pressure during CPR, hopefully improving coronary blood flow in the process. Other theoretical benefits include cardiac inotropy and chronotropy as well as increased cerebral perfusion pressure.

The majority of historical studies were retrospective or observational and demonstrated increased return of spontaneous circulation (ROSC) with epinephrine, but not necessarily an improved neurologic outcome. These studies were done mostly using animal models where assessing neurological outcome was difficult and extrapolation of data to humans was problematic. A 2015 *Journal of Critical Care* article by Loomba, *et al.* presented a meta-analysis of 14 studies involving 655,853 patients, which concluded that epinephrine does not increase survival to discharge but does make it more likely for those discharged to have poor neurologic outcomes.² A 2014 *Resuscitation* article by Lin, *et al.* also presented a meta-analysis of 14 randomized controlled trials drawing a similar conclusion: there is no benefit of epinephrine use in survival to discharge or neurological outcomes, but there were improved rates of survival to admission and ROSC with epinephrine versus placebo.³

The primary end goal for Perkins, *et al.* in the most recent study was survival at 30 days with a secondary endpoint of a favorable neurological outcome at 30

days (ie, Modified Rankin score of ≤ 3). Those with a non-favorable neurological outcome had a Modified Rankin Score of 4 or 5. 130 of the 4015 (3.2%) in the epinephrine group survived to 30 days as compared to only 94 of the 3999 (2.4%) in the placebo group.

Modified Rankin Score

- 0 – No symptoms at all
- 1 – No significant disability despite symptoms; able to carry out all usual duties and activities
- 2 – Slight disability but able to look after own affairs without assistance
- 3 – Moderate disability; requiring some help, but able to walk without assistance
- 4 – Moderately severe disability; unable to walk without assistance and unable to attend to own bodily needs without assistance
- 5 – Severe disability; bedridden, incontinent and requiring constant nursing care and attention
- 6 – Dead

While the use of epinephrine did lead to higher rates of 30-day survival, there was no difference in favorable neurological outcome because more survivors had severe neurological

impairment in the epinephrine group. While some patients did have a favorable neurological outcome after receiving epinephrine, the numbers were similar in the non-epinephrine group as well. These results are congruent with previous studies. However, this new study is not only the most recent, but is also randomized and controlled, adding validity and supporting the conclusions of previous studies.

These results have significant implications in the ongoing debate regarding OHCA pharmacotherapy. However, they also raise new social, ethical, economic, and moral questions that are less frequently discussed. For example, what would you say to a physician who told you, “We can give your mother a medication for her heart problem that would increase the chances of her living by almost 1%. But, if she did survive, she would likely be unable to walk or take care of herself and may be bedridden and require a diaper?”

Is it worth it? Would the increased rates of (at least, initial) survival allow a family to say goodbye before their loved one passes away? Would it allow for an increased likelihood of viable



organ donation? Or, would it lead to a prolonged ICU stay, giving the family a false sense of hope because the patient has a pulse and brainstem reflexes but little hope of neurological and functional recovery?

Further complicating this analysis is that the data seem to suggest that patients who end up walking out of the hospital neurologically intact after suffering cardiac arrest may have done

so without ever receiving epinephrine anyway. Loomba, *et al.* also found that the NNT for epinephrine in OHCA was 7, and there was no benefit to hospital discharge or survival at 1 month.

Epinephrine is also recommended regardless of underlying rhythm -- whether it be PEA, VFib, VTach, asystole, torsades de pointes, or bradycardia in a pediatric patient. While the authors of this study did mention that about 17% of

patients presented in VFib, they did not discuss if outcomes were different based on which rhythm the patient had when receiving epinephrine versus placebo. Given recent discussions of withholding epinephrine (and even giving esmolol in refractory VFib⁴), it would have been interesting to see if, in this study, patients in VFib who were randomized to the placebo group happened to do better than those receiving epinephrine.

PAMPer Trial

The Prehospital Plasma During Air Medical Transport in Trauma Patients at Risk for Hemorrhagic Shock (PAMPer) trial is a pragmatic, multicenter, cluster-randomized study that included patients at risk for hemorrhagic shock who were transported via air medical transport.⁵ Patients included in the study were those transported from the scene of an injury or referred from an outside emergency department to a participating trauma center. They were eligible for the study if they were between 18-90 years old and sustained blunt or penetrating trauma. Patients included were those who experienced at least one episode of hypotension (SBP <90 mm Hg) and tachycardia (HR >108) or severe hypotension (SBP <70) prior to arrival at the receiving trauma center.

The intervention group received two units of either group AB or group A with a low anti-B antibody titer (<1:100) thawed plasma, which was initiated during transport by the air medical team. The study protocol included infusion of both units of plasma even if administration was ongoing upon arrival to the trauma center. Plasma administration was followed with standard care as defined by the provider's local protocol.

The control group was composed of the same population and was provided with standard-care resuscitation. This included infusion of crystalloid as primary resuscitative fluid, although 13 of the participating 27 air medical bases had the ability to administer 2 units of universal donor red cells in accordance with local protocols.

Upon study completion, a total of 501 patients were evaluated; 230 patients were included in the plasma group and 271 patients were in the standard-care group. The primary outcome was 30-day mortality. This was significantly lower in the plasma group when compared with the standard-care group (23.2% vs. 33.0%; 95% confidence interval, -18.6 to -1.0; $P=0.03$). Secondary outcomes that favored the intervention group included 24-hour mortality, in-hospital mortality, amount of blood transfused in 24 hours, and INR (1.2 in the plasma group and 1.3 in standard-care). Of these, only the difference in INR was found to be statistically significant after adjustment for multiple comparisons. There were no documented cases of transfusion-

related lung injury. Transfusion-related reactions occurred in 2.2% of patients in the plasma group, and personnel at blood bank services at the sites where they were reported assessed them as minor. There was no significant difference between the two groups in multiorgan failure, nosocomial infection, or vasopressors received in the first 24 hours.

This is a potentially practice-changing study in the management of hemorrhage in the prehospital air transport setting. While it is logistically challenging to provide thawed plasma to prehospital providers, it appears it will help to decrease mortality in patients suffering from hemorrhagic shock. The number needed to treat in regard to 30-day mortality is staggering at 10. It seems possible that the early administration of this product helps to prevent downstream coagulopathy, perhaps than if it were to be administered immediately upon hospital arrival.

Large-scale administration of plasma in the prehospital setting would come with challenges. There would need to be agreements between prehospital



providers and the facilities receiving patients. Administration of plasma is also not likely to be useful in all settings. This was a study that was restricted to air medical transport during which the transport time was 40 minutes in the standard care group and 42 in the plasma group. This is not representative of what many ground EMS services experience in terms of transport time. It would likely be impractical for urban prehospital providers with short transport times to begin these interventions; another study has shown that there is no significant mortality benefit to prehospital plasma use in an urban ground transport setting.⁶ The relatively short shelf life of thawed plasma must also be taken into account, meaning that this may only be a viable treatment option for transport services that experience a high volume of trauma patients. Nonetheless, this provides further evidence that severe hemorrhagic shock needs to be treated with blood, not "salt water" (saline). Not only is it important to replace oxygen-carrying red blood cells (RBCs) but also the clotting factors that are critical in hemostasis. ★

A CASE REPORT

Acute Myocardial Infarction in a 29-year-old Male

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Cardiovascular disease (CVD) is currently the leading cause of death in both men and women across the United States. The term describes a group of diseases that includes coronary artery disease, myocardial infarction (MI), stroke, and heart failure. There are both modifiable and non-modifiable risk factors that can increase the risk of a person's susceptibility to CVD. Non-modifiable risk factors include ethnicity, family history of CVD, sex, and age. Historically, studies regarding CVD use 40-45 years old as the lower limit defining a "young" patient with CVD. These studies have found that 5-10% of patients experiencing an MI are younger than 40.¹

When a patient presents to the ED with chest pain, the HEART score is often used to assess the risk of an acute coronary syndrome (ACS). The parameters of the HEART score include a suspicious history, ischemic ECG findings, age, number of cardiac risk factors, and initial troponin levels. Each category will receive a value of 0, 1, or 2 for a maximum score of 10; a patient under 45 years old receives 0 points for the age category. A patient's total HEART score can classify their 6-week risk of a major cardiac event as low, moderate, or high. In the case below, the patient received a HEART score of 3, a low score representing 0.9-1.7%² risk of having a major cardiac event.

CASE REPORT

A 29-year-old Caucasian male with history of acute lymphoblastic leukemia (ALL) treated with full body radiation, marrow transplant and chemotherapy 12 years ago, presents to the ER with acute chest pain and SOB that began 3.5 hours prior to arrival, while delivering food. He then walked into the local CVS to check his blood pressure, which was "150s/90s." Although unable to describe the quality of the pain, he is writhing in pain on the stretcher and rates it a 10/10. Nothing seems to be giving him any relief. He denies any similar past episodes. The patient does not currently use any medications, denies a history of smoking or illicit drug use. The patient's father has a history of CAD in his 50s, and his mother's health is unremarkable. The patient has been in complete remission from ALL for 12 years; he has no other medical conditions.

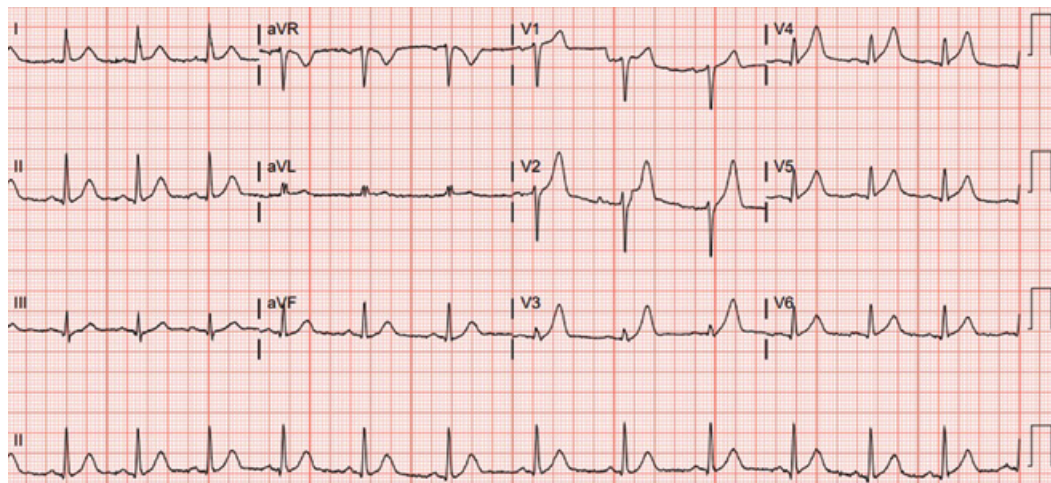


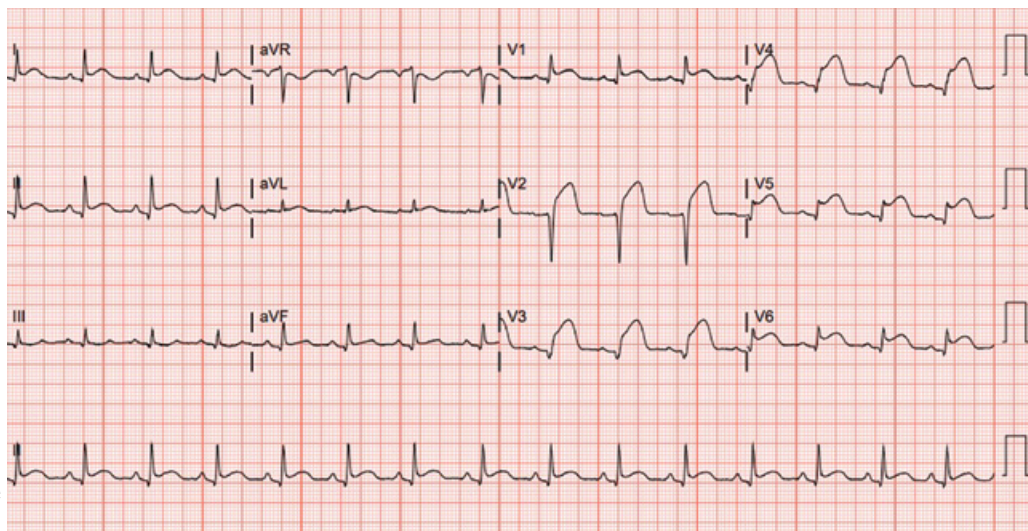
FIGURE 1. Initial EKG Reading

The patient's initial workup in the ED included an ECG, chest X-ray, complete blood count, comprehensive metabolic panel, cardiac enzymes, lipase, and coagulation studies. His initial vitals were unremarkable. His initial EKG (Figure 1) performed in triage, showed normal sinus rhythm with hyperacute T wave changes in V2-V6; his potassium was 4.3. His other labs were unremarkable except for a troponin-I of 0.41. A repeat ECG (Figure 2) was obtained 1.5 hours after the first, which showed normal sinus rhythm with significant ST segment elevation in leads I, II, and V1 through V6, suggesting an anterolateral MI. The patient was immediately given aspirin, morphine, nitroglycerin, and heparin and was taken for emergent cardiac catheterization. Upon catheterization, he was found to have a 100-percent occlusion along the middle portion of the left anterior descending (LAD) artery, no occlusions were found in the right coronary artery or the circumflex artery. Stents were placed along the first and second diagonal branches of the LAD. During the procedure, the patient had several episodes of ventricular tachycardia which resolved with deep coughing and also had an episode of ventricular fibrillation requiring cardioversion to sinus rhythm. The catheterization lab report also noted that patient had an anterolateral apical hypokinesia and a left ventricular function of 40%. Following percutaneous coronary intervention, the patient was admitted to the intensive care unit for further monitoring.

FIGURE 2. Repeat EKG Discussion

As a patient with low CVD risk factors, it is important to consider the history of pediatric cancer. This patient had been in remission from ALL for 12 years. As a child, he had undergone numerous combinations of chemotherapy, radiation, and eventually a bone marrow transplant. Studies have examined the development of atherosclerosis in patients who have received radiation.³ In pediatric cancer patients, individuals who live more than 5 years following their radiation have shown an eightfold increase in their risk of developing atherosclerosis later in life.³

Also consider the T wave, which is a reflection of the repolarization of the ventricles of the heart. Most T waves have a positive direction and should be asymmetric with a slow upstroke and a rapid downstroke.⁴ The T waves in the limb leads should be less than 5 mm, and those in the chest leads should be less than 10 mm.⁴ Upon the initial ECG of this patient, it was important to rule out any abnormalities in T wave presentation that could reflect underlying pathology of the heart. No evidence of hyperkalemia in



the form of peaked T waves that could lead to fatal arrhythmias were seen at the time. However, hyperacute T waves were seen in leads II and V2 through V6. Hyperacute T waves are often, but not always, known to precede ST segment elevation. They are the earliest available electrical event that can be detected on an ECG during an acute ischemic event and can rapidly progress into ST segment elevation.⁴ While a hyperacute T wave is not always associated with ST elevation, if detected on an ECG, consider ordering serial ECGs with the cardiac biomarkers to watch for acute cardiac event. *

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A Simplified Approach Reading a Journal Article

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Introduction

Evidence-based medicine is the foundation of how we care for our patients. Every medical decision we make on shift stems from some level of evidence. As physicians, we need to feel confident with critically appraising new research.

Undergraduate and graduate medical education provides minimal preparation for scrupulous literature review. Complex statistical analyses and long-winded articles can quickly become overwhelming without a simplified and consistent approach to article review.

What's the Question?

Instead of reading an extensive introduction, skip to the punch line. Find out immediately what the question is the authors are trying to answer. This will provide you a frame of reference for the entire study and will allow you to determine if the introduction justifies the importance and originality of the question being asked. Specifically, look at the objective, hypothesis and primary outcome. This is important because the purpose of the study is to evaluate the objective and/or hypothesis through measurement of the primary and secondary outcomes. Some journals have begun to incorporate an editor's "capsule summary," which can be helpful to ascertain the study question.

The Devil Is in the Details

The hardest sections to read are often the most important sections to understand. A paper with poorly designed methods and/or inaccurately reported results can invalidate the entire study. Take your time here. Read it more than

once to have a good understanding of how the data was collected.

1. Begin with answering who, what, where when and how?

Who: Consider inclusion and exclusion criteria. Is a selection bias present?

What: What intervention is being performed and what measurements were taken? Is this a patient centered outcome or a laboratory centered outcome?

Where: What is the study setting? Is it similar to the one I practice in?

When: Was the study performed a significant amount of time prior to publication? Have patient care practices changed since then? Review the references for any older references and out-of-date citations. For example, has more current research on the topic been published recently and not included in the references?

How: Analytic strategy and data analyses used.

2. Consider internal validity. In other words, are the study design and collection methods appropriate for the question they are trying to answer?
3. Consider external validity. Do the results translate to my practice? Are the results generalizable?

Results and Statistical Analyses: Do the Numbers Answer the Questions?

Stay organized in the results sections and keeps things simple. It's easy to get overwhelmed with statistical tests, sensitivity analyses and subgroup analyses.

1. Do the results parallel the methods? Are the specified outcomes measured and reported appropriately?
2. Carefully compare the patient cohorts. Are they similar? Are there confounders potentially influencing the results?
3. Is the size of the study population appropriate? Was a power calculation reported? Remember that a study

is usually only powered based on the primary outcome. Interpret the secondary outcome variables and other a priori or post hoc analyses with this in mind.

4. Were the right statistical tests utilized? We went to school to be Emergency Physicians, not statisticians. Most of us do not have an epidemiology background or expertise in statistics. However, we should be able to determine if the statistical tests used were appropriate based on study design and types of variables measured.
5. Check the math! Recreating a linear regression curve may be unreasonable, but take the time to make your own 2x2 table and confirm reported sensitivities, specificities, and more. You may be surprised that some published studies have discrepancies here. And you may need to perform these calculations on board examinations.

Does This Change My Practice?

Finally, when reviewing the discussion section, pay careful attention to the authors' conclusion based on the results presented and the existing literature.

1. Consider limitations in the study that could severely affect the results, validity and generalizability.
2. Do you agree with the authors' conclusions? Remember that the conclusion should be based on the objectives and hypotheses. Be skeptical of magnanimous statements. Studies that report extreme results are often poorly designed and prone to inherent bias, or they answer a question we already know to be true or nobody finds relevant.
3. Is this relevant to emergency medicine? Can I take the study to the bedside?
Critical appraisal of research does not require a PhD or background in statistics. Stay organized, use a consistent approach, and keep things simple. Lastly, take pride in critical appraisal of the literature; our patients and your career are depending on it! ★

Profiles in Toxicology

Ken Katz, MD, FACEP, FAAEM, FACMT

Aaron S. Frey, DO

Lehigh Valley Health Network
 Chair, EMRA Toxicology Committee
 @AFrey1776

To highlight the varied opportunities in the field of medical toxicology, the EMRA Toxicology Committee will be publishing a series of Q-and-A discussions. You will hear from a variety of people: from recently matched residents to current fellows to seasoned attendings. It is our hope that these interviews will not only deepen your understanding of medical toxicology, but also increase your interest in it. Our inaugural interview is with Ken Katz, the editor-in-chief of the recently published *EMRA and ACMT Medical Toxicology Guide*.



Where did you attend residency?

Christiana Care Health System in Newark, DE

Where was your medical toxicology fellowship? Why did you choose that program?

Banner University Medical Center in Phoenix. I chose that program because of the outstanding medical toxicology attendings and unique educational opportunities.

Why did you pursue a career in medical toxicology?

I've always enjoyed the logical, mechanistic aspect of medical toxicology. Moreover, understanding the basic science of a drug or toxin and its interaction with the body is fascinating. Also, it requires good diagnostic skills at the bedside and an opportunity to truly think about clinical presentations and treatments.

Where do you currently practice medical toxicology? How have you divided your time between emergency medicine and medical toxicology?

Lehigh Valley Health Network in Allentown, PA. I spend approximately half my time as an emergency physician and the other half practicing medical toxicology.

What are some of the misconceptions about medical toxicology that you have encountered?

I was once rounding in the hospital, and someone asked me what a taxidermist was. For once in my life I had no reply.

What are some of the challenges that medical toxicologists will face in the next decade?

I think medical toxicologists are seeing their role grow in the field of addiction medicine and as providers for medication assisted treatment both in the inpatient and outpatient settings.

You are the creator of the EMRA and ACMT Medical Toxicology Guide. What made you want to write this publication?

After nearly 15 years of answering literally thousands of questions from all different health care providers both on the phone and in the hospital, it became evident that providing an efficient, pragmatic guide for health care providers at the bedside was needed to help clinicians care for the poisoned patient.

Why should someone consider a career in medical toxicology?

I think it offers a variety of opportunities in many different arenas — from industry and government to inpatient and outpatient clinical settings and much more.

What advice do you have for those considering a career in medical toxicology?

Honestly, if you are thinking of completing a medical toxicology fellowship, don't wait — do it right after residency.

How does a resident become a strong applicant for fellowship?

I would advise participating in a medical toxicology rotation or offering to assist with any research opportunities, for starters. Showing interest and participating are paramount. Also attending a medical toxicology conference is another opportunity to meet other toxicologists and find out more about fellowship programs.

There are almost 30 medical toxicology fellowships. How does one determine which program is a good fit for him/her?

Honestly, this is a personal decision based on many factors — much the same as those used to determine residency. Personally, I looked for unique or interesting curriculum offerings that set a certain program apart from others. But a resident will be well-trained in any accredited fellowship program.

FIND OUT MORE

EMRA resident and alumni members received the *EMRA and ACMT Medical Toxicology Guide* as a benefit of membership. To order additional copies, visit emra.org/guides.

To learn more about toxicology fellowships, read the chapter in the *EMRA Fellowship Guide* at <https://emra.org/books/fellowship-guide-book/27-toxicology>. ★

MedSpanish

Language Immersion and Medical Outreach

Darren Cuthbert, MD, MPH
Rutgers Robert Wood Johnson
@DarrenCut

When Haywood Hall, MD, FACEP, founded the Pan American Collaborative Emergency Medicine Development program — PACE MD — in 2002, his goal was to improve health care, emergency systems, and medical education in Mexico and Latin America.

To that end, PACE has established a variety of courses and programs advocating for better patient care on many fronts: out-of-hospital efforts, OB-GYN, emergency, advanced life support, and more. Physicians, nurses, and other health care providers — many from the U.S. — volunteer as mentors and course instructors, helping train local health care teams and thus creating lasting improvements.

But for as much as they give, these

volunteers gain even more. PACE's MedSpanish participants, for example, get a Spanish language immersion experience specific to health care, as they endeavor to learn the language while teaching medical concepts.

It's a symbiotic experience that can make you a better doctor in many ways, according to one participant.

Medical Education in San Miguel de Allende

While nearing the end of residency, I wanted to do something different with my elective time, so I decided to head south of the border to San Miguel de Allende, Mexico, with PACE MD's MedSpanish program. Not only did this rotation exceed all of my expectations, but it changed me as an individual, broadening my horizons while affording me a new skill set that will help me throughout my career.

To start, San Miguel is a place like no other. Its heart, character, and history have made it a UNESCO World Heritage cultural site where a vibrant downtown frequently features live music, festivals, and celebrations on nearly every corner. The food is richly diverse, the people are proud, and it creates a culture that is full of life. Meanwhile the city is surrounded by incredible nature and history.

I was fortunate to begin my MedSpanish rotation when PACE was holding its Global Forum for Emergency Care and Obstetrics. This was a conference like no other I've attended. There was an intrinsic goal among all providers to expand knowledge and improve care throughout the places they love in Latin America. From Chile to Brazil to the U.S. and our neighbors in Mexico, everyone came together in training. Thousands of practitioners were participating in various courses, including ACLS, Helping Babies Breathe, PALS, BLS, and ALSO (Advanced Life Support in Obstetrics). Throughout the week, training also continued on the use of diagnostic bedside ultrasound. Lectures were provided by Judith Tintinalli, Robert Suter, Terrence Mulligan, Jeff Solheim (president of ENA), Ken Iserson, and many other world-renowned medical educators.

Following the conference, my days included a few hours of one-on-one instruction in medical and basic Spanish. My tutor was friendly and fun, with a wealth of experience in teaching. Fluent in both English and Spanish, she quickly recognized my knowledge gaps that required closer attention and improvement, and I looked forward to meeting with her daily. After my language lesson, I would head to one of my 4 clinical rotations: working with the Mexican Red Cross (Cruz Roja Mexicana), Rural Health Brigades, CAISES public health clinic, and the public hospital ED.

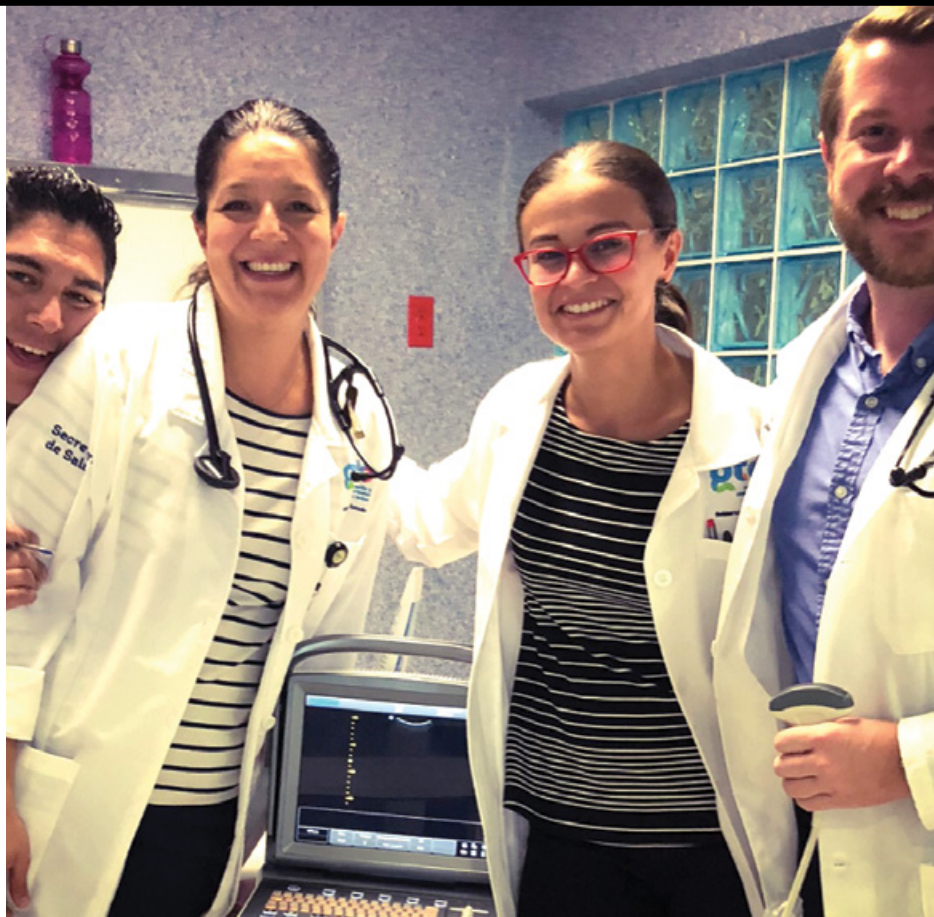
I began by working with the Cruz



Roja Mexicana, focusing on prehospital care in Mexico. Their providers were highly trained and equipped with familiar tools such as chest tube and cricothyrotomy kits. They integrated me seamlessly into their team, eager to learn whatever I knew that could help them improve their practices. Together we developed straightforward lectures on airway, electrocardiogram, and ACLS for their team.

Next, I headed to the government-run CAISES health clinic where general practitioners care for those with government insurance (Seguro Popular) throughout Mexico. Their spectrum of care far exceeded what I expected. While working alongside my friend, Dr. Diego Elias, I witnessed him utilize ultrasound to diagnose a long list of complaints, while managing various complex cases outside the hospital. Most patient presentations echoed what we see in EDs back home, though without the resources normally available to us — making the history and physical exam that much more important. For MedSpanish participants, it's a golden opportunity to practice Spanish in the context of emergency medicine.

MedSpanish participants also take part in community health brigades multiple times per week. Mobile health clinics are set up outside of schools and other highly trafficked areas, with the focus on seeing as many patients as possible. For instance, we set up in a poverty-stricken ranching neighborhood, not far from the outskirts of San



Miguel. While seeing patients alongside physicians who run this initiative, we encountered another team of PACE educators teaching CPR to parents and community members.

During my ED rotation, I saw our specialty as it began in the U.S.: clinically rich but resource-limited — so using those valuable resources requires a higher level of justification. In some aspects this

creates a more highly skilled practitioner when it comes to putting hands on the patient. At the same time, we had a high-quality ultrasound to help with bedside diagnosis — and lots of interest from the house staff in learning any tips we could share for using it. My favorite part of rotating through the ED was the culture among the staff. Every day I was welcomed genuinely, and the whole staff took pride in spending their days together helping members of their community.

Since leaving Mexico, I've found the knowledge I gained through the MedSpanish program to be highly useful. No matter if you're a junior resident or a senior physician, if you're passably fluent or you lack any Spanish vocabulary — I strongly advocate for you to find a way to participate in this program. As a residency elective, it was a once-in-a-lifetime opportunity to learn and grow in a beautiful culture. I now plan to continuously return to Mexico and embrace this opportunity throughout life.

For more information on PACE MD and its outreach programs, visit <http://www.pacemd.org>. *





A RESIDENT'S PERSPECTIVE

Guns, Politics, and the Emergency Department

What is our role as physicians in confronting the intersection of politics and health? We must be willing to engage in honest dialogue and consider politics as a part of medicine.

Noah Berland, MD, MS
 Kings County Hospital –
 SUNY Downstate Medical Center
 @Making_Noah

After the recent workplace shooting and killing of one of our emergency physician colleagues, a Facebook group called EMDocs exploded in controversy over a discussion regarding gun violence. Some argued that it is not our place to inject politics into such a forum, which caused a large number of members to leave the group. It has additionally sparked much debate and conversation among our community.

This discussion occurred during a campaign by many physicians using the Twitter hashtags #ThisIsMyLane and #ThisIsOurLane in response to the National Rifle Association (NRA) telling doctors to “stay in your lane” instead of adding our voices to the gun debate. There are a few sides to this debate in the EM community. One side seeks to look at gun violence from an academic perspective, advocating for the federal government to fund research on firearm injuries before staking a position in the debate. Another side argues that we already know guns kill and maim

(anecdotal evidence and from other countries) and that the only way to prevent these deaths and injuries is by restricting access to guns. Yet another group believes *people* - not guns - commit violence and the only possible solution is to address the human factor. (*Full disclosure, I fall into Camp 2. I believe the most effective way to address our high rate of gun-related injuries and deaths, among our peer nations, is to better limit access to firearms.*)³

I'll never forget my first patient during residency who died after being shot. I'll always remember the look of horror that was frozen on his face. I can't forget seeing the scene of his shooting on TV, thinking to myself that this is something no one should have to go through in life or in death, not something any family or community should have to deal with.

No short piece seems capable of shifting opinions on this topic, however evidenced-based it is, but I think the more important conversation that has been sparked, and that is inherent to the “#ThisIsOurLane” movement, is just what is our role as physicians in confronting the intersection of politics and health. We must be willing to engage in honest dialogue and consider politics as a part of medicine.

Certainly, the beginning of medicine focused on individuals and their diseases, but public health and medicine started to intersect almost immediately. The Roman empire dominated not only because of its advanced weapons of war, but also — and arguably more importantly — because of its aqueduct systems and access to clean water.⁴⁻⁶ Public health and political initiatives coming from physicians, often the people in the best position to understand an issue and its implications on human life, continue to have broader reach than one doctor can ever have on the treatment of individual patients. In our modern world, the ultimate responsibility of identifying, implementing, and enforcing public health initiatives falls squarely on governments, ultimately intertwining physicians with politics. In the U.S., with the ACA expanding Medicaid, our very livelihood is even more dependent

on the government and the specific policies it enacts and enforces, with real implications for our patients.⁷ These are inescapable realities; medical societies (including our own) have huge wings devoted to lobbying the government. How can we argue that practicing medicine today isn't inherently political?

You're still skeptical. You believe as doctors we should only deal with the known facts, and what's in front of us; you think we owe it to our patients to be anything but political. Such a point of view isn't without merit. But where do we start drawing the line between medical and political? These two areas are so intertwined, and it can interfere with open, honest discussion of issues. Is gun control political or medical? It's both. If you work in an ED where no one gets shot, whether self-inflicted or inflicted upon them, then I can see how this might appear to be a foreign idea, but for those of us who treat the victims of gun violence on a regular basis, this is a medical problem confronting our patients no different than substance use, hypertension, or diabetes, and it requires the same focus on intervention, treatment, and prophylaxis. Because of research and advocacy, seat belts, airbags, and smoke and carbon monoxide detectors are no longer optional but required. Why should we approach firearm injuries differently than our predecessors approached motor vehicle collisions, fire, and indoor air?

Kings County Hospital, where I train, was dubbed the “Knife and Gun Club” in the 1980s. Though the incidence of penetrating trauma has drastically reduced, in no small part to the strong gun control policies of Mayor Michael Bloomberg, we deal with victims of violence every day. It is important to note how many young minority men are disproportionately being killed and injured because of gun violence. If you work in an area serving minority populations, it is even more important to advocate for policies to address the social determinants of health. I was involved in my medical school's White Coat For Black Lives movement, and I think this is something we should all hold close to our hearts as providers.⁸⁻¹⁰

To try to prevent the continued cycle of violence in our predominantly black community, one of our amazing attendings, Dr. Robert Gore, runs the Kings Against Violence Initiative (KAVI), a youth intervention program. It's the same concept as programs that aim to prevent diabetes through education and outreach. It's aimed at controlling the disease at home, rather than waiting until critical illness arises. Violence is a disease, and we as physicians can play a role in treating and any all disease of the mind, body, and community. Is it political when I ask patients about their smoking or substance use? Is it political when I talk to patients about their access to medications and physicians based on their insurance? Or what about when I talk to patients in the ED about stresses in their life, including concerns about their immigration status in this rapidly changing and fraught political atmosphere? ACEP has even taken a political stance on the Trump Administration's Public Charge policy for immigrants, saying that it will hurt our patients and discourage them from seeking care.¹¹

We are not just black boxes seeing, treating, and dispo-ing patients. We have to look at what brings our patients in to truly treat them and our communities. I surely think I'm correct in my opinion,¹² and though I don't think there is need to do unique-to-the-U.S. research to come to the same answer other countries have discovered, I am open to having this discussion with my colleagues who might disagree with me in a mature and thoughtful manner. I think we would all benefit from an open and mature discussion. To simply shut down discussion because it is a politically charged topic will get us nowhere and will only hurt our patients.

It shouldn't require us to have our own family affected by something to motivate action and discussion, but now it's personal, and when one of our own is a victim of gun violence, we will have failed if we don't respond. This doesn't mean we all need to agree, but we should all agree to discuss the topic and to advocate for the health of our patients. ★

Thank You!

Alameda Health System – Highland

Aria Health

Arrowhead Regional Medical Center

Carolinas Medical Center

Doctors Hospital-Ohio Health

Eastern Virginia Medical School

Genesys Regional Medical Center

Good Samaritan Hospital Medical Center

Henry Ford Allegiance Health

Henry Ford Wyandotte Hospital

Lehigh Valley Health Network

Lincoln Medical & Mental Health Center

Madigan Army Medical Center

Maricopa Medical Center

Medical College of Wisconsin

Naval Medical Center Portsmouth

Ohio Valley Medical Center

Oregon Health and Science University

San Antonio Military Medical Center

St. John Maccomb-Oakland Hospital

St. Joseph's Regional Medical Center/NY Medical

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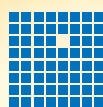
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Calling All Medical Students!

EMRA is launching a new event: the **EMRA Spring Medical Student Forum** will help reduce the stress and anxiety associated with matching in EM.

A panel of EM faculty and program directors will share advice on the many aspects of matching into the specialty. It's a must-attend event.

Date: Saturday, May 4

Location: Washington, D.C., metro area

Registration: FREE — but limited spots available

Held the day before the ACEP Leadership and Advocacy Conference, this event complements our longstanding Fall Forum. **Attendance is free for EMRA members but attendance is limited!** Register at <https://emra.org/be-involved/events--activities/medical-student-forum>. ★

Spring Resolutions Due

The deadline to submit EMRA resolutions for the next Representative Council meeting is **Feb. 15!** If you have an idea about EMRA policies and practices, or you want your association to take a stand on a topic near and dear to you, write a resolution before the deadline.

If there's a topic addressed by other groups within the house of medicine, consider whether EMRA needs to take a stand. If you think the EMRA bylaws need a change, submit it! You set the direction of the association, and you do it most effectively by submitting resolutions to your Representative Council.

- **Resolution Submission Form:** <https://bit.ly/2VTOUi3>
- **Guidelines for Writing a Resolution:** <https://bit.ly/2M9qpJt> ★

UPCOMING EVENTS

Feb. 20: NRMP rank order lists due

March 6: EM Residents' Appreciation Day

March 11-14: NRMP SOAP

March 15: NRMP Match Day

March 31 – April 3: CORD Academic Assembly and EMRA Spring Meetings, Hyatt Regency Seattle

May 4: EMRA Spring Medical Student Forum, Washington, D.C., area

May 5-8: ACEP Leadership & Advocacy Conference @ Grand Hyatt, Washington, DC

May 13-16: Essentials of EM @ The Cosmopolitan, Las Vegas

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EM Residents' Appreciation Day

March 6, 2019

Make plans now to for EM Residents' Appreciation Day! How will you celebrate?

It doesn't take a massive investment of time or resources to make sure the residents who help provide 24/7/365 coverage in the ED know they're vital members of the team. Free food, public recognition, social shout-outs, even a sincere word in passing — these gestures matter. Make appreciation part of your culture this year! ★

Drop the Mic at LAC

EMRA and the ACEP Young Physicians Section will shine a spotlight on engaging, talented speakers during the Drop the Mic Advocacy Lectures event held in conjunction with ACEP LAC.

Approximately 5 speakers will present mini-lectures (~7 minutes) focusing on timely legislative topics. Selected speakers will be given a topic and lecture objectives. The Drop the Mic Advocacy Lectures will take place during the EMRA/YPS Health Policy Primer on Sunday, May 5.

Application: https://docs.google.com/forms/d/e/1FAIpQLScybmKdBazqeCmTX3WKMOVI_qCUTrzljFzQMYBfB1bHXbJRUw/viewform
LAC Registration: <https://www.acep.org/lac>

Questions? Please contact YPS Chair Jessica Best, MD, at Jessica.a.best@gmail.com or EMRA Health Policy Director Angela Cai, MD, MBA, at healthpolicydir@emra.org. ★



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10pm - 2am

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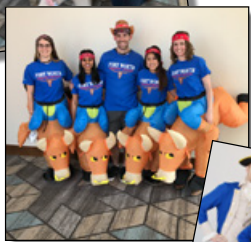
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MONDAY, APRIL 1

SEATTLE

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Questions about the event: slewis@emra.org.

ECG Challenge

Lauren Briskie, MD

Christiana Care Health System
@DrBriskEM

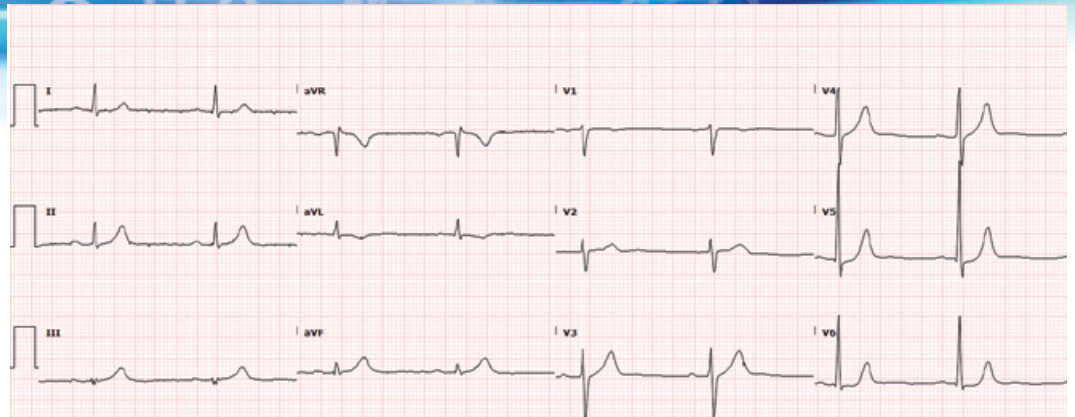
Jeremy Berberian, MD

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

CASE.

A 52-year-old male presents to the ED with chest pain.

What is your interpretation of his EKG?



See the ANSWER on page 43

Visual Diagnosis

Kathryn Braseth, PA-C

Department of Emergency Medicine
UT Health San Antonio

Luke Husby, MD

Global Health Fellow
Department of Emergency Medicine
UT Health San Antonio

Browning S. Wayman, MD, DTM&H

Director, Global Health Fellowship
Department of Emergency Medicine
UT Health San Antonio
@UTHHealthSA

A 22-year-old vaccinated male with no past medical history presents to an ED in South Texas with a rash to his right inner thigh for 3 days. He reports the rash started as a large red bump, progressing into a pruritic “ring-like” appearance minimally relieved by hydrocortisone. Review of systems is positive for nausea otherwise without systemic symptoms. He recalls feeling a bite to his inner thigh the day prior to the rash developing but denies seeing any arthropods at that time. He denies camping, hiking, or travel outside of South Texas in the past 6 months, though his apartment complex has woods surrounding it where he frequently walks his dog along the perimeter. He also endorses having indoor/outdoor cats.

Physical exam is significant for 12 cm x 15 cm circular erythematous rash with central clearing to right medial thigh. Blanchable, flat, nontender patch. No arthropod present.



Diagnose this condition

See the ANSWER on page 44

45 EMRA years

established 1974

That's how long EMRA has supported physicians like you with resources in emergency medicine. You are the doctors on the front lines, holding hands and saving lives.

1974

Joseph F. Waeckerle, MD and others had an idea to form an organization for emergency medicine residents. EMRA was born.

1975

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

1982

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

1988

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

1994

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

2000

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

2005

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

2013

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

2016-PRESENT

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





ECG Challenge

TWI in aVL

Sinus bradycardia with T-wave inversion (TWI) in aVL, hyperacute T waves in the inferior leads, and slight STD in the lateral precordial leads. In a patient presenting with chest pain, these findings are concerning for ischemia and/or impending infarction.

A new isolated TWI in aVL is becoming more commonly recognized as a concerning EKG finding in patients presenting with chest pain. It is critical to be observant for this finding when interpreting an EKG. One study found that 74.9% of physicians missed the finding in their interpretation. Reciprocal changes, which include TWI and/or STD, can be the earliest EKG finding for an AMI.

The isolated TWI in aVL is associated with both impending inferior wall MI and mid-LAD lesions. Multiple studies have demonstrated that an isolated TWI in aVL has a high specificity for mid-LAD lesions, and one study one found it to be the only ECG variable significantly predictive of mid-LAD lesion. When seen with an inferior MI, reciprocal changes in aVL have a high sensitivity, specificity, and PPV for right ventricular involvement which is associated with a higher morbidity and mortality.

The “New Tall T-wave in V1” or “Too Tall T-wave in V1” is another EKG finding that can be an early sign of ischemia and/or infarct in patients presenting with chest pain. It can be thought of as an early hyperacute T-wave concerning for impending anterior MI. The T-wave is typically inverted in V1 so a new upright T-wave in V1 (when compared to a prior EKG) or the T-wave amplitude in V1 > V6 are considered abnormal in the absence of LVH, LBBB, or high voltage (eg, young athletes). As with any hyperacute T-waves, serial EKG’s should be obtained to see if it evolves. ★

LEARNING POINTS

Ischemic T-wave Inversions (TWI)

GENERAL FEATURES

- TWI ≥ 0.1 mm in ≥ 2 contiguous leads with prominent R-wave or R/S ratio >1

CLINICAL SIGNIFICANCE

- New TWI (compared to prior) in the setting of a clinically suggestive presentation should prompt concern for ACS
- New upright T-wave ≥ 0.15 mm in V1 is abnormal in the absence of LVH or BBB
 - Called “Too Tall T-wave” in ACS presentations and suggests impending anterior ACS
 - Upright T-wave in V1 > V6 is concerning for CAD or acute ischemia
- TWI in aVL
 - Can be seen with occlusion of mid-LAD
 - Potential early indicator of impending inferior MI
 - Suggests RV involvement when seen with inferior MI
- When in doubt, obtain serial EKG’s

Hyperacute T-waves

GENERAL FEATURES

- No universally accepted definition of hyperacute T-waves

EKG FEATURES

- Tall, prominent T wave with broad base
 - Hyperkalemia produces tall, peaked, symmetric, narrow based T-waves
- T wave size is relative to QRS, so small T wave can be hyperacute if paired with a small QRS
- Asymmetric with gradual upstroke (ascending) and abrupt return to baseline (descending)
- J-point elevation

CLINICAL SIGNIFICANCE

- Suggests acute vessel occlusion with ischemia and impending AMI
- In appropriate presentation, EKG indication for reperfusion therapy
- Hyperacute T-waves + ST depression in reciprocal leads \Rightarrow early AMI

Assessment



Southern Tick-Associated Rash Illness

This is a case of Southern Tick-Associated Rash Illness (STARI), also known as Master's disease, which occurs after the bite of the Lone Star tick, *Amblyomma americanum*. This tick feeds on humans, cats, dogs, and other animals. The saliva from this tick causes irritation and redness, but the exact cause of the rash is unknown. There is evidence to show the Lone Star tick does not transmit *Borrelia burgdorferi*, the cause of Lyme disease.

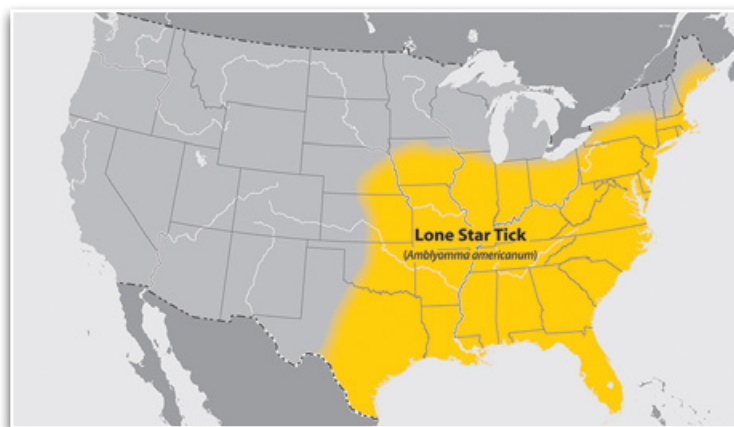
Diagnosis

The clinical presentation includes a bull's-eye lesion (erythema migrans) similar to that of Lyme disease (commonly smaller in size than Lyme disease). Patients may also have systemic symptoms such as fever, fatigue, nausea, headache, myalgias, and arthralgias mimicking Lyme.

Clinical diagnosis based on history of possible tick bite, matching geographic location, and physical exam consistent with bull's eye lesion with or without tick found on exam. Geographic distribution of the Lone Star tick appropriately begins in Texas, spreading as far north as South Dakota and expanding eastward to cover Maine to Florida. Unfortunately, this overlaps heavily with the blacklegged tick or deer tick (*Ixodes scapularis*), which is Lyme disease vector on the East Coast — making differentiation difficult. There is no widely used specific blood test to confirm diagnosis, and Lyme exclusion is prudent.

Treatment

Patients are often treated with doxycycline because STARI closely resembles Lyme Disease; however, there is no evidence to suggest that antibiotic use speeds recovery in STARI management. STARI has not been linked with long-term outcomes similar to Lyme disease, and reassurance of prognosis at the time of diagnosis is crucial in preventing downstream mismanagement. ★



Board Review Questions



PEER (Physician's Evaluation and Educational Review in Emergency Medicine) is ACEP's gold standard in self-assessment and educational review.

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

Order PEER at acep.org/peer

1. A 38-year-old man with diabetes presents complaining of recurrent fever for 1 week, measured as 102°F at home. He has no other symptoms. Physical examination reveals a fever of 38.6°C (101.5°F), a grade 2/6 systolic murmur, and linear streaks under several fingernails. Which of the following tests is most likely to diagnose his condition?
 - A. Blood cultures
 - B. Chest x-ray
 - C. Influenza PCR assay
 - D. Lyme serology
2. What is the most likely underlying etiology of cardiac arrest from polymorphic ventricular tachycardia?
 - A. Brugada syndrome
 - B. Electrolyte abnormality
 - C. Myocardial ischemia
 - D. Re-entrant mechanism through scarred myocardium
3. A 27-year-old woman presents with intermittent, sharp, right-sided chest pain of 3 days' duration that is worse with deep inspiration. She says the pain occasionally occurs when she is not active; it is sharp for about 1 minute then dull and is reproducible with palpation along the right sternal border. She is otherwise healthy; lungs are clear with equal breath sounds. Which of the following concomitant conditions, if present, would lessen concern that this presentation represents a life-threatening problem?
 - A. Pregnancy
 - B. Rheumatoid arthritis
 - C. Systemic lupus erythematosus
 - D. Type II diabetes mellitus
4. A 35-year-old man presents comatose. Vital signs include BP 124/68, P 74, R 26, T 35.6°C (96.1°F). Glucose level is normal. Blood gas analysis reveals pH 7.23, Po₂ 96, and Pco₂ 23. Toxicity from which of these agents is most consistent with this presentation?
 - A. Ethylene glycol
 - B. Isopropanol
 - C. Phenobarbital
 - D. Salicylate
5. A 27-year-old man presents by ambulance after a high-speed head-on collision in which he was an unrestrained driver. On arrival, he has decreased breath sounds on the left side and is in severe respiratory distress. A chest x-ray reveals abdominal contents in the thoracic cavity. Which of the following statements about this patient's injury is correct?
 - A. Blunt trauma typically produces smaller tears than penetrating trauma
 - B. It occurs most commonly on the right side of the body
 - C. Mortality rate is higher when the injury is due to penetrating trauma
 - D. Symptoms are related to the degree of herniation of abdominal contents ★

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



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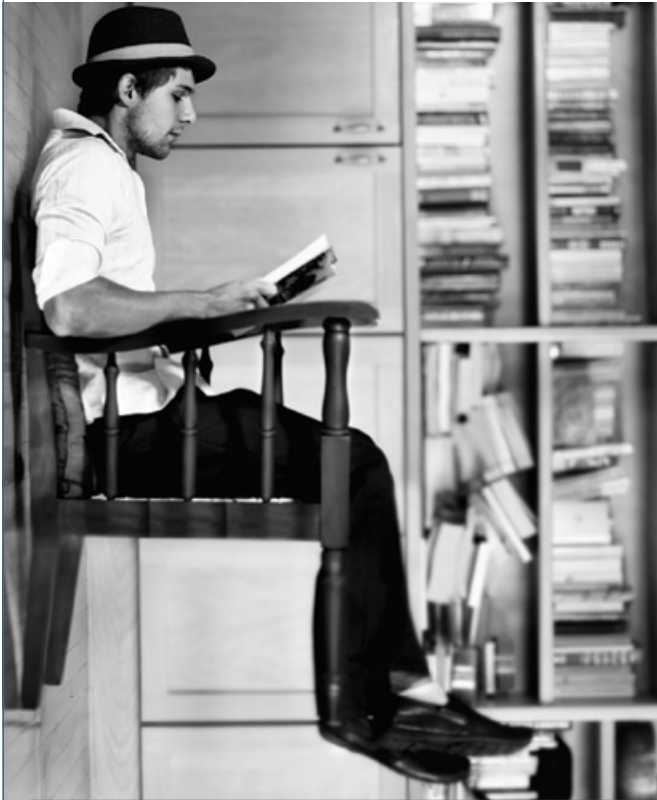
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CALIFORNIA

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TEXAS



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Reading Hospital

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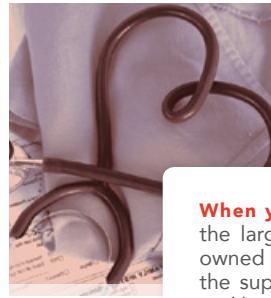
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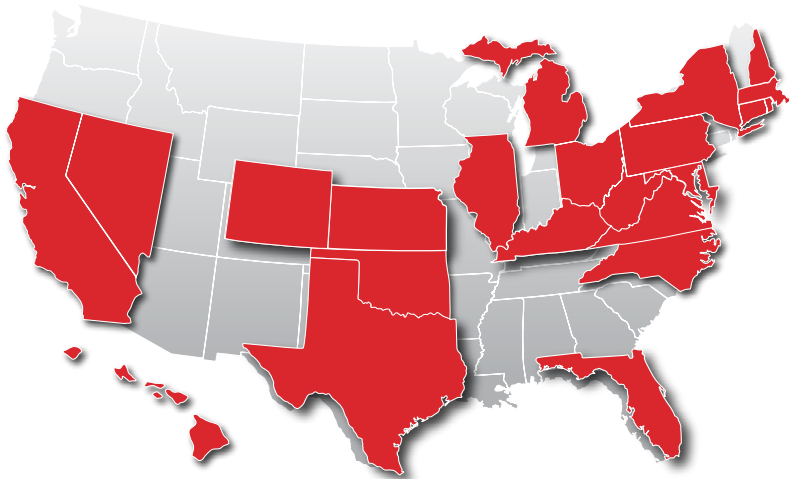


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Email: Lewis.Nelson@njms.rutgers.edu

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HIRING Emergency Medicine Physicians

Kettering Health Network is seeking BC/BE Emergency Medicine physicians to join a highly regarded, regional private group in Dayton, OH.

- Strong, democratic group of 80+ physicians and advanced practice providers
- Group covers 11 of Kettering Health Network's Emergency Departments, including 7 hospitals and 4 freestanding Emergency Centers
- Trauma Level II and III
- Competitive salary, generous benefits package
- \$80,000 Sign-on Bonus
- \$20,000 Early Bonus for residents who sign before they start their final year of residency
- Epic EMR used throughout the network
- Warmth, charm, and work-life balance of the Midwest

Site visits are being scheduled now!

Contact Cindy Corson
Physician Recruitment Manager
cindy.corson@ketteringhealth.org
(937) 558-3475 (office)
(503) 201-8588 (cell)



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{ Job Opportunities }

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EMS Fellowship Director/EMS Medical Director
Assistant Medical Director
PEM/EM Core Faculty
Vice Chair Research Emergency Medicine

What We're Offering:

- We'll foster your passion for patient care and cultivate a collaborative environment rich with diversity
- Salaries commensurate with qualifications
- Sign-on bonus
- Relocation assistance
- Retirement options
- Penn State University Tuition Discount
- On-campus fitness center, daycare, credit union, and so much more!

What We're Seeking:

- Experienced leaders with a passion to inspire a team
- Ability to work collaboratively within diverse academic and clinical environments
- Demonstrate a spark for innovation and research opportunities for Department
- Completion of an accredited Emergency Medicine Residency Program
- BE/BC by ABEM or ABOEM
- Observation experience is a plus

What the Area Offers:

We welcome you to a community that emulates the values Milton Hershey instilled in a town that holds his name. Located in a safe family-friendly setting, Hershey, PA, our local neighborhoods boast a reasonable cost of living whether you prefer a more suburban setting or thriving city rich in theater, arts, and culture. Known as the home of the Hershey chocolate bar, Hershey's community is rich in history and offers an abundant range of outdoor activities, arts, and diverse experiences. We're conveniently located within a short distance to major cities such as Philadelphia, Pittsburgh, NYC, Baltimore, and Washington DC.



FOR ADDITIONAL INFORMATION PLEASE CONTACT:



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



Contact: Myles Jen Kin, DO FAAEM

Wachusett Emergency Physicians
60 Hospital Road, Leominster, MA 01453
Call: (978) 660-9221 Email: mjenkin@wepdocs.com

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Seeking Fulltime BC/BE Emergency Physicians
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Bayfront Health broke ground for a new 37,000 sq ft state-of-the-art
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- 36-hour PA/ARNP coverage

A free-standing ED is also slated to open November 1, 2018.

We offer a comprehensive benefit package including health,
dental, vision, LTC, CME allowance, 401K, profit-sharing,
along with a partnership track.

Requirements: residency trained, ACLS, ATLS and PALS certified.

For more information, contact
the Emergency Physicians of St. Petersburg, PA at
(727)-553-7300, fax (727)-553-7395
or email: aaepp360@gmail.com
Our web site is EpspBayfront.com



Contact us at
recruitment@umem.org
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UNIVERSITY of MARYLAND EMERGENCY MEDICINE

Academic and Community Openings for BE/BC Emergency Physicians

Vibrant and varied career possibilities in academic and community settings in the Baltimore metropolitan area as well as near Washington, Philadelphia and Maryland's coastline.

Live and work in an urban, suburban or rural community, in an atmosphere that encourages work/life balance.

Current EM Practice Opportunities

- Downtown Baltimore – Volumes from 21 to 66K
- North of Baltimore – Volumes from 32 to 65K
- Eastern Shore – Volumes from 15 to 37K
- DC Suburbs – Volumes from 34 to 60K

Our supportive team approach in the delivery of high quality patient care features:

- Dedicated fast track and intake units staffed by Family Practice physicians and PAs
 - ED scribes and medical information systems
 - Stroke centers & STEMI programs
- Ultrasound programs with bedside US machines
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- Additional incentive compensation
- Medical, dental, vision and life insurance
- Employer-paid CME, PTO and 401K safe harbor retirement plan
- Employer-paid malpractice insurance with full tail coverage

SEEKING EMERGENCY DEPARTMENT PHYSICIANS

The busiest ED in North Carolina, and one of the top 15 busiest in the nation, treats 95k adult and 35k pediatric cases annually in its 92 beds. We are currently seeking residency trained BC/BE emergency physicians to work in the 75 bed adult ED. This ED serves a high acuity patient population with 28% annual admission rate. There are over 90 hours of adult physician coverage daily and over 110 hours mid-level coverage daily. It is a Level III Trauma Center with robust hospitalist service, interventional cardiology 24/7, cardiac surgery, neurosurgery, etc. The facility is Chest Pain and Stroke accredited. The EMS system is hospital owned and managed with an award winning paramedic program. Of note, the Pediatric ED is separate and has 17 dedicated beds with an additional 24 hours of physician coverage and 20 hours of mid-level coverage. We welcomed our inaugural class of Emergency Medicine Residents in July 2017. Opportunities exist for both clinical and academic emergency physicians.



EXPECTING TO BE EXCITED AND CHALLENGED?

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TOP TIER COMPENSATION

The cash compensation package is valued at over \$250/hour, including evening, night, and holiday differentials, as well as a quarterly incentive bonus. We offer a generous sign-on bonus plus moving stipend. The comprehensive benefits package includes Malpractice Insurance Paid; CME Time and Allowance; 403(b) match and 457(b); and health, dental, and other desirable benefits.

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.

Known for its many golf courses (Pinehurst is located only 30 minutes away), our central location provides easy access to beautiful beaches to our east and to the majestic Blue Ridge Mountains to our west. Our mild climate, low cost of living, and patriotic spirit makes our location ideal for rising healthcare professionals and families.



CAPE FEAR VALLEY HEALTH

Please contact Ashley Dowless, Corporate Director, Physician Recruitment
at 910-615-1888

or adowl@capefearvalley.com for additional information.





EMSOC, Emergency Medicine Specialists of Orange County

Hiring both Emergency and Pediatric Emergency Physicians
Partnership Track Available

Ideal Emergency Medicine physician candidates are those boarded in emergency medicine or board eligible in emergency medicine working interested in an academic emergency medicine environment at CHOC Children's Hospital of Orange County.

Ideal Pediatric Emergency Medicine physician candidates are those boarded in pediatric emergency medicine or board eligible in pediatric emergency medicine working interested in an academic emergency medicine environment at CHOC Children's Hospital of Orange County.

CHOC Children's Hospital of Orange County is a newly opened ultramodern emergency department of 35 beds with Level II trauma center designation, rapid assessment and treatment area, fellowship and resident involvement. Additionally, all emergency physicians are supported with medical scribes and mid-level providers.

EMSOC and CHOC Children's Hospital of Orange County are nationally recognized for teaching excellence in an independent democratic practice environment.

Contact Information Contact EMSOC@emsoc.net for applications and additional information, or call 714-543-8911.

Emergency Medicine & Toxicology Faculty

Rutgers Robert Wood Johnson Medical School

The Department of Emergency Medicine at Rutgers Robert Wood Johnson Medical School, one of the nation's leading comprehensive medical schools, is currently recruiting Emergency Physicians and Medical Toxicologists to join our growing academic faculty.

Robert Wood Johnson Medical School and its principal teaching affiliate, Robert Wood Johnson University Hospital, comprise New Jersey's premier academic medical center. A 580-bed, Level 1 Trauma Center and New Jersey's only Level 2 Pediatric Trauma Center, Robert Wood Johnson University Hospital has an annual ED census of greater than 90,000 visits.

The department has a well-established, three-year residency program and an Emergency Ultrasound fellowship. The department is seeking physicians who can contribute to our clinical, education and research missions.

Qualified candidates must be ABEM/ABOEM certified/eligible. Salary and benefits are competitive and commensurate with experience. Sub specialty training is desired but not necessary.

For consideration, please send a letter of intent and a curriculum vitae to:

**Robert Eisenstein, MD, Chair, Department of Emergency Medicine
Rutgers Robert Wood Johnson Medical School**

1 Robert Wood Johnson Place, MEB 104, New Brunswick, NJ 08901

Email: Robert.Eisenstein@rutgers.edu

Phone: 732-235-8717 • Fax: 732 235-7379

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EMERGENCY MEDICINE

Health System

BC/BE EMERGENCY PHYSICIANS NEEDED to join current staff of 40+ physicians

- **Level I Trauma Center with 75 beds and fast track**
- **Medical Observation Unit with 16 beds**
- **Pediatric ED with 16 beds**
- **Community hospital ED with 21 beds**

EXCELLENT COMPENSATION PACKAGE!

- **Competitive salary with RVU-based incentives, CME, paid vacation, health/life/malpractice, 401k**

Huntsville Hospital is looking for additional coverage for our progressive Emergency Department. We see approximately 150,000 patient visits per year across our 4 different units (Level I Trauma Center, Medical Observation Unit, Pediatric ED at Children's hospital, community hospital in Madison - plus an OB ED staffed by our OBGYN Hospitalist team. Our physicians work an average of 14-15 shifts per month (9 hours per shift), allowing for an excellent work/life balance. Teaching opportunities with 3rd/4th year medical students from UAB and Family Medicine and Internal Medicine residents at UAB-Huntsville rotate through our ED.

Huntsville Hospital is a Level I Trauma Center and the Regional Referral Center for North Alabama and Southern Tennessee. Huntsville Hospital is Alabama's only Top 50 Heart Hospital by Truven Health Analytics and one of America's 50 Best Cardiac Surgery Programs by HealthGrades.

Huntsville is situated in the fastest growing major metropolitan area of Alabama, and with the highest per-capita income in the Southeast, Huntsville is the best place to live, learn, and work. We are a community on the move, rich with values and creative talents. These unique characteristics will certainly provide a place for you and your family to flourish. With a population of 385K, we are a high-tech, family-oriented, multicultural community with excellent schools, dining, and entertainment - all nestled in the foothills of the beautiful Appalachian Mountains.

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



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