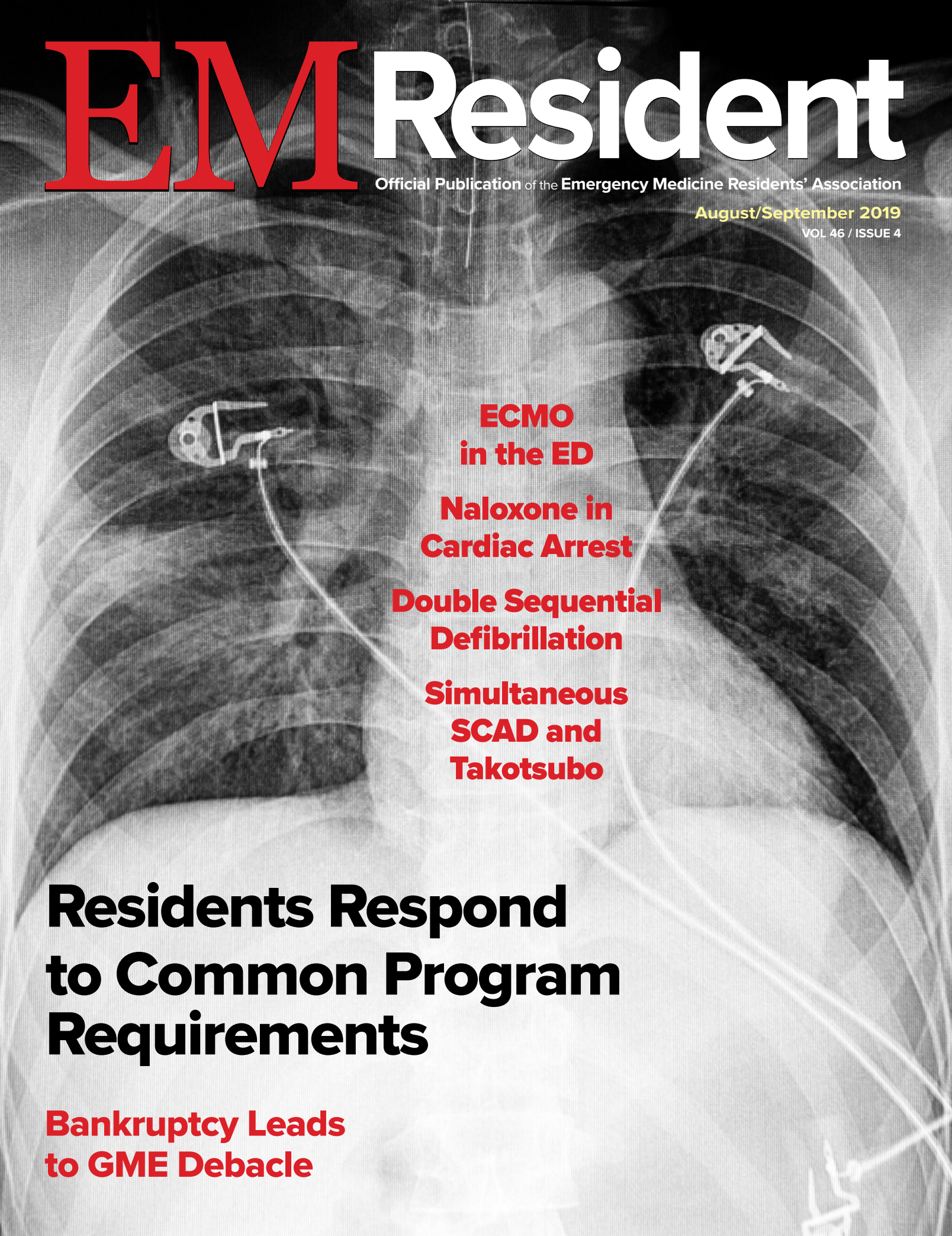
EM Resident
Official Publication of the Emergency Medicine Residents' Association
August/September 2019
VOL 46 / ISSUE 4



ECMO in the ED

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Bankruptcy Leads to GME Debacle

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HAHNEMANN CLOSURE

The Worst GME Debacle in U.S. History

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Editor-in-Chief, *EM Resident*
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In late June, Philadelphia's Hahnemann University Hospital, the main teaching hospital for Drexel University, declared bankruptcy and announced plans to cease all patient care operations by September. The abrupt statement came just days ahead of a new academic year for the 570+ residents and fellows training in Hahnemann's 36 graduate medical education (GME) programs.[1]

The closure has not only abandoned patients in need, but also forced the largest stranding of medical residents in U.S. history.[2]

Chaos and confusion have followed. Based on previous program closures, it was assumed the trainees would be released from their contracts and tasked to secure positions for themselves elsewhere. In an outpouring of support, programs in the Philadelphia area and throughout the country began developing plans to accommodate the soon-to-be displaced trainees. The ACGME invoked its Extraordinary Circumstances Policy[3] and the Education Commission for Foreign Medical Graduates began posting updates to help trainees navigate J1 visa challenges.[4]

But then Hahnemann announced plans to transfer its GME funding to neighboring Tower Health, a community health system with only a fraction of the medical specialties and positions held by Hahnemann.[5] This prompted court filings by the AAMC, ECFMG, and ACGME.[6] Yet a bankruptcy judge ruled in mid-July that Hahnemann can, indeed, sell its residency and fellowship slots to the highest bidder — regardless of whether that institution is capable of completing the training of the actual doctors currently filling those slots.

This has sparked immediate outrage in the medical education community.

When a GME program closes, Medicare regulations and ACGME guidelines provide a framework for hospitals to proceed in terms of placing trainees. Usually, displaced trainees are permitted to temporarily transfer their Medicare funding to other hospitals for the duration of their remaining training. If the receiving institution intends to keep that position and its associated funding past the transferred resident's training, a section of the Affordable Care Act allows for the permanent transfer of GME funding via a competitive redistribution process.

Before any of that can happen for the Drexel residents, however, their contracts and Medicare funding must be released by Hahnemann. Yet that funding instead is being sold off in a bidding process — keeping hundreds of trainees in limbo even as the hospital closure creates a health care crisis for Philadelphia's underserved communities.[5]

Court filings continue, with no clear resolution in sight as of press time. ***Crystal* clear, however, is the need for every medical student, resident, and practicing physician to engage in advocacy to protect not only patients but also the house of medicine itself from ongoing threats to GME.** Residency slots must not be treated as a commodity, and corporate money games cannot be allowed to supersede crucial medical education and patient care.

EMRA Supports Drexel Residents

EMRA is saddened by the impending closure of Hahnemann University Hospital, Philadelphia's historic safety net hospital and training home to hundreds of Drexel University resident and fellow physicians, including 45 emergency medicine residents and 3 toxicology fellows.

As of 2019, there are 2,552 annual EM residency spots including NRMP and AOA positions. While abrupt residency closures are uncommon, we have seen a few closures of ACGME accredited programs in the past years related to natural disasters such as Hurricane Katrina, hospitals filing for bankruptcy, and abrupt physician staffing changes such as Summa Health.

We urge EM programs around the country to accommodate displaced trainees where possible. EMRA applauds the work of the ACGME, CORD, AMA, and the EM community in supporting these residents, and has been in touch with each of these groups to advocate for residents and fellows. We also call for flexibility in transferring resident funding so that residents have the ability to choose the best option for them.

EMRA has also reached out to the Drexel EM community to offer its full support as the program and trainees find a solution to continue their commitment to education and patient care. We consider each of the Hahnemann EM residents affected as a co-resident despite having different home institutions because the bond between emergency medicine residents extends beyond the hospitals where we work. We stand ready to assist in any way, shape, or form. ✶

References available online

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OSTEOPATHIC MEDICINE
What Exactly is a DO?

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A BRIEF HISTORY OF OSTEOPATHIC MEDICINE

Osteopathic medicine was founded on the principle that all body systems are connected and interdependent on one another for good health. The philosophy was conceptualized and coined in 1874 by Dr. Andrew Taylor Still, MD. A Virginia born man, Dr. Still studied medicine and completed an apprenticeship under his father Dr. Abraham Still. He then moved to Missouri where he obtained his medical license in 1854. He completed additional coursework at the College of Physicians and Surgeons in Kansas City, MO. Dr. Still was a servant of the people, first as a member of the Kansas Legislature in 1857 and then as a surgeon within the 21st Kansas Militia of the Union Army during the Civil War; achieving the rank of Major.

After losing his three children to meningitis in 1864, Dr. Still pursued methods of treating disease outside of the realm of traditional medicine of the time. His research led him to believe all aspects of the human body are interconnected and the body itself has an inherent ability to heal. He developed a series of manual techniques which would come to be known as osteopathic manipulative medicine (OMM) that allowed him to both diagnose illness as well as restore the body back to a more relaxed state which serves to improve the body's ability to heal itself. He opened the first school of osteopathic medicine (now known as A.T. Still University) in Kirksville, MO, in 1892 incorporating all aspects of modern medicine taught in allopathic medical schools while also integrating a more holistic philosophy of treating mind, body, and spirit rather than disease alone.[1]

Health care in 19th century United States (US) was fragmented by several competing approaches including naturopathy, chiropractic, osteopathy, and many others. The Flexner Report of 1910 helped define modern scientific medicine,

and while osteopathy was often included in the realm of "nonconformist" approaches in medicine, osteopathic medicine, which included the current practice of medicine, surgery and obstetrics with its traditional principles, survived into the modern era of medicine.[2]

A BRIEF HISTORY OF OSTEOPATHIC EM

The American College of Osteopathic Emergency Physicians (ACOEP) was founded in 1975 and the American Osteopathic Association (AOA) approved the first osteopathic emergency medicine (EM) residency programs in 1978.[3] Leading up to Single Accreditation, there were 62 osteopathic EM residency programs.[4] Of the 2,488 EM positions available in the 2019 National Resident Matching Program (NRMP) Match, osteopathic students filled 648 (26%) of those positions.[5] There have been two DO Past Presidents of ACEP: Robert E. Suter, DO, MHA, FACEP, and Alexander Rosenau, DO, CPE, FACEP.[6] Dr. Suter is the current ACOEP President-elect as well.[7] There are DOs on the current EMRA Board of Directors and MDs on the current ACOEP — Resident Student Organization (RSO) Board of Directors.[8-9] On the surface, there are few differences between osteopathic and allopathic EM physicians. However, there are some important caveats to be aware of.

Osteopath versus Osteopathic Physician

Osteopathic medicine grew and perpetuated in the US at the end of the 19th century when many medical therapies were more harmful than beneficial. When osteopathic medicine spread abroad, particularly to Europe, the principles remained the same but the training was very different. Osteopathic physicians in the US earn a Doctor of Osteopathic Medicine (DO) degree, while European osteopaths earn a Diploma of Osteopathy (DO). They both call themselves DOs, though US trained osteopathic physicians are medical doctors who practice the entire scope of modern medicine, ie. prescribing medications and performing surgery. However, foreign-trained osteopaths are primarily trained to practice osteopathic manipulative techniques and are not medical doctors who prescribe medications and perform surgery.[10] Similarly, osteopathy is sometimes used to refer to the limited practice of osteopathic diagnostic and manipulative techniques, whereas osteopathic medicine is the preferred term for the medical care delivered by US osteopathic physicians that combines osteopathic philosophy with the current practice of medicine, surgery and obstetrics.[11] Therefore, calling an osteopathic physician an osteopath discredits the years of medical school and residency that person has completed.

The Differences Between

MDs and DOs work side by side in the same residency programs and emergency departments all across the nation every day, providing quality care and strong teamwork.

DO and MD Physicians

Medical School

There are 35 accredited colleges of osteopathic medicine with over 30,000 students.[12-13] US osteopathic students complete very similar curriculums to our allopathic counterparts. The biggest difference is the addition of Osteopathic Manipulative Medicine (OMM). OMM is a manual form of medicine which includes somatic, orthopedic, and visceral treatments to help reset the body to a neutral state in support of the body's inherent ability to heal itself.[11]

Board Exams

For medical students, osteopathic trainees are required to take the Comprehensive Osteopathic Medical Licensing Examination of the United States (COMLEX) board exams – our counterpart to the United States Medical Licensing Examination (USMLE) board exams. COMLEX has a different question style and often incorporates OMM physical findings into the question stems. Overall, the COMLEX and USMLE exams cover the same material just in slightly different ways. Osteopathic students can choose to take the USMLE in addition to the COMLEX and often many students do.

Pre-Single Accreditation Residency Training

Osteopathic EM residency programs were accredited by the AOA and participated in a separate match process from the Accreditation Council for Graduate Medical Education (ACGME) programs. These osteopathic EM programs were 4 years long to incorporate a traditional rotating internship and adequate OMM training at the residency level. Students could choose to apply through either the AOA or ACGME match. Those who applied to both, did so knowing the AOA match would be approximately one month prior to the ACGME match. If an osteopathic student chose to apply to both matches, and matched to an AOA residency, they would have to withdraw their application in the ACGME match.

Post-Single Accreditation Residency Training

However, with the completion of Single Accreditation set for 2020, much is changing. For osteopathic students, the merger means they only have to submit one application to apply to all of our potential residency programs. For osteopathic EM residents, major changes include the research paper is no longer required, the traditional rotating internship is no longer required except in a few states where they may need to apply for exemption through Resolution 42, and all emergency medicine residency graduates will be eligible to pursue board certification through the American Board of Emergency Medicine (ABEM). By 2020, all residency programs are expected to be solely accredited by the ACGME. Programs have the option to apply for Osteopathic Recognition which includes OMM training in the curriculum.[14]

CONCLUSION

Despite the nomenclature, MDs and DOs are both physicians. They work side by side in the same residency programs and emergency departments all across the nation every day. While there are some differences between the training philosophies and content, there are more similarities than differences. ✶

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Emergency Medicine Residents Oppose ACGME Changes to the Common Program Requirements

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In July 2019, limits on clinical work hours will be taken away from core residency program faculty. The impact of this change is far-reaching and has provoked action by emergency medicine trainees across the country. Core faculty are the foundation of emergency medicine, charged with training the future generation while pushing the specialty forward through scholarship and academic productivity—all while working clinically in the most intense, stressful specialty of modern medicine. Without protected time, core faculty will be unable to complete their mission. The future of emergency medicine hangs in the balance as the emergency medicine community grapples with the new Common Program Requirements.

Upcoming Changes to the Common Program Requirements

At its core, residency training represents the transformation of a medical student to an independent physician. The purpose of the Accrediting Council for Graduate Medical Education (ACGME) is to improve health care by standardizing this process. The general guidelines created by the ACGME for accreditation purposes are known as the Common Program Requirements (CPR). Specifications in these program requirements range from limits on resident duty hours to faculty responsibilities. For unique issues, the ACGME also maintains a dedicated Review Committee for each specialty.

To adapt over time, the CPR undergoes regular revision. Historically, the CPR and Review Committee for Emergency Medicine (RC-EM) requirements have met the needs of emergency medicine by allowing the specialty to protect academic time for core faculty.

This has been accomplished through a specific provision that allows the RC-EM to limit clinical hours for the program director, associate program directors, and core faculty.[1]

When the new CPR changes go into effect on July 1, this protection will no longer exist.

The greatest threat stems from removal of the provision allowing for the RC-EM to create requirements specific to core faculty.[2] This prevents the specialty from creating formal limitations on clinical hours for core faculty and represents a major ideology shift away from protected faculty time. Emergency medicine residents are disturbed by this revision and the downstream impact it will have on the specialty.

Challenges to On-Shift Education

The nature of emergency medicine demands minute-to-minute care, which rapidly diminishes the ability to do anything other than direct patient care on shift. While faculty from other specialties may be able to briefly step away from clinical responsibilities after rounds, the dynamic environment in the emergency department requires that emergency physicians directly care for patients throughout the entire shift.[3]

The challenge of bringing core education into the emergency department cannot be understated. Unlike inpatient teaching services with mandated caps, there is no way to cap the number of patients presenting to the emergency department. Even in maximum-capacity scenarios in which ambulances are temporarily diverted to other hospitals, the doors remain open for walk-in patients to receive care.

Like unlimited patient caps, the increasingly prevalent phenomenon of emergency department overcrowding places stress on education. While this has led to increased clinical experience for emergency medicine residents and forced the development of innovative teaching methods on-shift, overcrowding results in decreased faculty supervision and further decreases on-shift education.[4,5] To make matters more complicated, the literature is clear that both emergency department patient volume and complexity are increasing at a steady pace.[6]

Finally, clinical productivity measures represent a unique challenge to on shift education. Although important to maximize patient care capacity, pressure to satisfy administrative metrics disincentivizes emergency physicians from dedicating on shift time to resident education. While residents in other specialties often decrease productivity, emergency medicine residents have been shown to both increase productivity and reduce emergency physician staffing

requirements.[7,8,9,10] By decreasing emergency physician hours and adding resident hours, emergency departments are maximizing productivity while decreasing opportunity for on shift education. Without protected time, there is no way to compensate for this.

Emerging Role of Off Shift Clinical Education

While all residents have dedicated classroom time, there is substantial evidence that emergency medicine education is best done off-shift.[4] One potential explanation for this is that the breadth of the specialty is massive. As the name implies, emergency medicine encompasses a wide scope of low frequency, high risk situations. As other specialties continue to sub-specialize and reduce their individual scope, the emergency medicine scope of practice continues to expand.

Although emergency medicine residents routinely perform *most* critical procedures in the emergency physician scope of practice, the expectation is that they will be able to perform *every* possible procedure safely at a moment's notice once working independently. This level of mastery can only be achieved through off shift education. In fact, simulation training has become a critical adjunct to emergency medicine education for this reason.[11,12,13,14] The only way that core faculty can provide this detailed level of training is through protected time.

Impact on Burnout Culture and Wellness

Protected core faculty hours are dedicated to clinical education, research, and administrative work. These activities represent the core of academic emergency medicine and serve as the guiding force for the specialty. Innovation in each of these respective areas is the direct result of protected time. In its absence, emergency medicine stands to lose the momentum it has worked so hard to generate since its inception.

Requiring core faculty to teach and perform scholarly activity without protected time is an unrealistic burden. In fact, there is evidence that excessive faculty work burdens perpetuate burnout culture and incapacitate the ability to effectively support residents during critical points in training.[15]

The high-intensity shift work of clinical emergency medicine has already proven to cause physician burnout at levels unrivaled by any other modern medical specialty. The unrealistic burden placed by removing core faculty protected time will substantially increase burnout, further contributing to this serious problem at a critically low point in the history of the specialty.[16]

The future of emergency medicine hangs in the balance as the emergency medicine community grapples with the new Common Program Requirements.

Workforce Implications in Academic Emergency Medicine

The future of academic emergency medicine is inherently dependent on protected time for core faculty. Historically, the percentage of emergency medicine residents that enter academic emergency medicine is approximately 26%.[17] With unlimited clinical hours, expectation to spend uncompensated personal time on academic productivity, and lower pay than alternative positions in community practice, it seems likely that this percentage will significantly decrease.[18,19,20]

Limiting time protections impairs the ability of faculty to investigate gaps in program inclusion, to enhance residency program diversity, and to mentor residents and students from underprivileged or underrepresented backgrounds. These are factors associated with increased diversity in emergency medicine residency programs.[21,22]

The loss of protected time will disproportionately impact graduating emergency medicine residents from diverse backgrounds and further exacerbate existing disparities among academic emergency medicine faculty. The cycle will then repeat as emergency medicine trainees from underprivileged or underrepresented backgrounds are less able to find academic mentors from diverse backgrounds.[21,22] Interestingly, the proposed CPR specifically requires that programs engage in practices that focus on systematic recruitment and retention of a diverse and inclusive workforce of residents, fellows, and academic faculty.[23]

To further illustrate this concept, consider the immediate impact that this simple change will have on the livelihood of academic emergency physicians themselves. The Council of Residency Directors in Emergency Medicine (CORD) recently conducted an internal survey designed to assess the opinions of protected time by those directly affected by the change—the program directors, assistant program directors, and core faculty in ACGME-accredited emergency medicine residency programs.

They found that 95% of nearly 200 respondents considered a loss of protected time to be "job threatening" or "career threatening". Not surprisingly, a similar proportion of respondents felt that the loss of protected time would significantly impair their ability to perform academic duties.[24] In this, eliminating protected time for core faculty threatens not only the future of the specialty, but the present, too.

Conclusion

As health care trends increasingly incentivize institutions to redirect priorities toward clinical productivity and revenue, it is now more important than ever for accrediting bodies to uphold the academic duty of residency training. It is imperative to maintain a high standard to ensure that today's residents are prepared to become tomorrow's leaders and innovators. For emergency physicians in training, protected faculty time is an absolute necessity. It is the key mechanism through which emergency medicine learners engage with educators and is the only way to achieve the academic progress that will propel the specialty into the future. ✶

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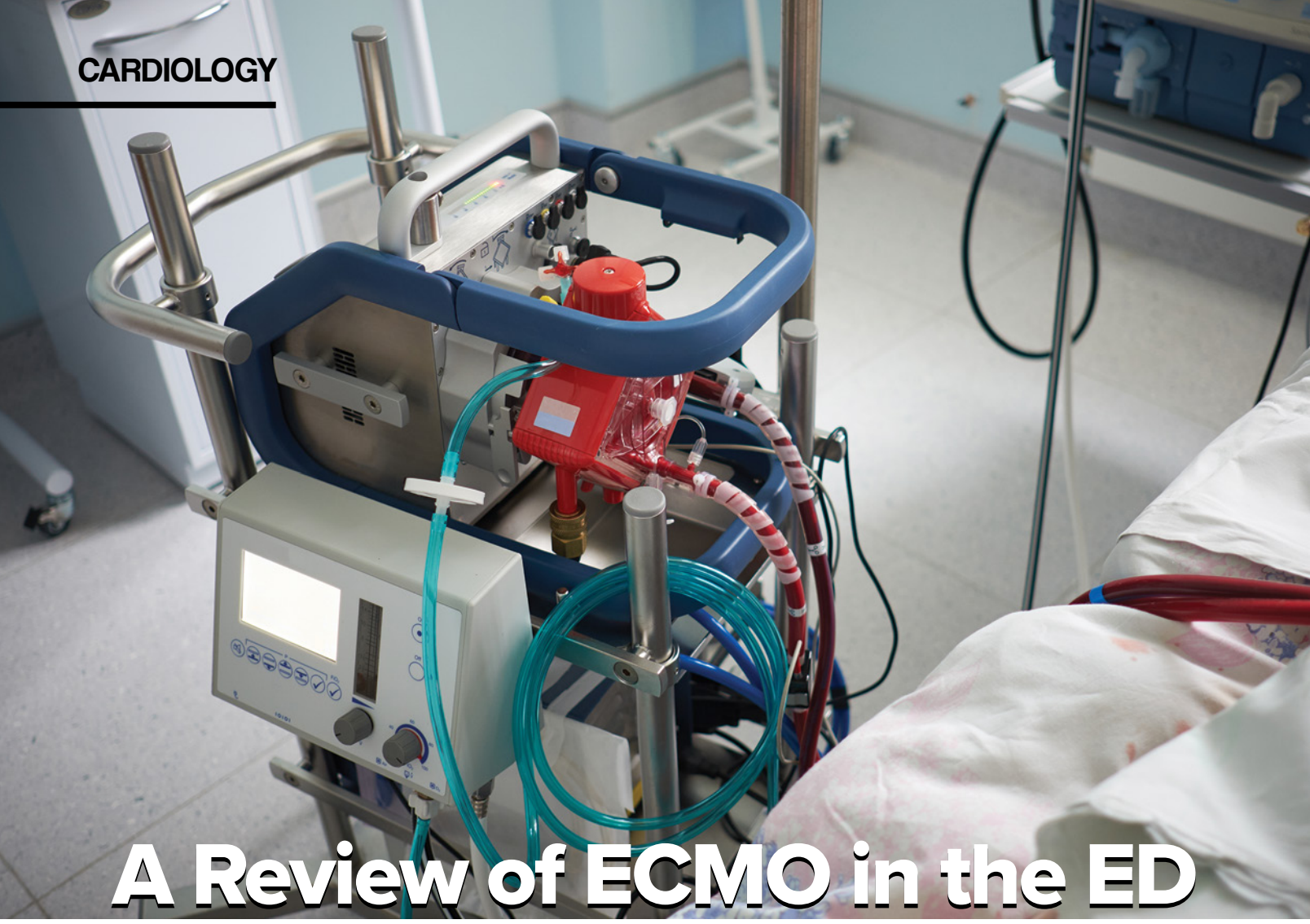

A Review of ECMO in the ED

History, Mechanics, Common Indications, and Future Implications

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The use of extracorporeal membrane oxygenation (ECMO) as a potentially lifesaving intervention is becoming increasingly popular and its application more diverse. Advancements in technology, increased training for intensivists, and its implementation at more health care centers have provided growing opportunities for the use of ECMO in patients with the appropriate indications.[1] An EM provider is tasked with diagnosing patients with acute, life-threatening pathologies and implementing the appropriate interventions in a timely manner. Certain presentations of acute respiratory failure, cardiopulmonary failure, or cardiac arrest may be refractory to early, aggressive resuscitation resulting in death or significant morbidity, for which ECMO may be a solution. Thus, it is imperative that professionals in the ED are familiar with ECMO in order to provide optimal care for the appropriate candidates. The purpose of this article is to review the history of ECMO, how it works, common indications for its use, and its role in the ED.

History of ECMO

ECMO has had an extensive history, initially functioning as a support device for patients in cardiopulmonary bypass operations in the 1950s.[1] As technology progressed, its use expanded, and in 1971, Dr. Solomon Hill successfully treated a patient with acute respiratory failure utilizing an extracorporeal bypass circuit.[2] This finding spurred increased usage of ECMO as a therapeutic option for patients with significant isolated lung injury refractory to optimal medical management, and in that time period, devices similar to ECMO were used in more than 150 patients with approximately 15% surviving the initial insult.[2] A few years later, an infamous case report was published detailing the use of ECMO in a neonate. In 1974, a mother emigrating from Mexico birthed a child at a hospital in Orange County, California, who aspirated meconium

at the time of delivery. This patient remained hypoxic despite maximal ventilator settings and was anticipated to suffer a fatal outcome. The decision was made to initiate ECMO, and the neonate fully recovered after 3 days.[3]

Over the next several years, more anecdotal evidence surfaced reporting increased survival when using ECMO in patients with respiratory failure, but no randomized controlled trials (RCT) had been performed to suggest its efficacy in treating patients with this indication.

In 1979, Zapol et al. published an RCT comparing survival between adult patients with severe acute respiratory failure treated with the medical standard of care including mechanical ventilation (MV) to ECMO. This study found no statistical difference in survival with approximately 90% mortality rate in both groups.[4] Additionally, in 1994, Morris et al. published an RCT that compared MV to veno-venous ECMO (VV ECMO) in patients with acute respiratory failure and reported survival rates of 44% and 33%, respectively.[5] The data up to this point showed the greatest benefit when ECMO was used in the pediatric population but little improvement when used in the adult setting.

Among the most important studies regarding the potential benefit of ECMO came from the United Kingdom in 2009. The Conventional Ventilatory Support vs Extracorporeal Membrane Oxygenation for Severe Adult Respiratory Failure (CESAR) trial compared VV ECMO with conventional medical management in patients with acute respiratory distress syndrome (ARDS). The trial's primary outcome was death or severe disability at 6 months or before discharge from hospital. While the design of this study invited significant criticism, it found an improved survival in the ECMO group (patient transferred to an ECMO center) when compared to the group treated with conventional management.[6] In contrast to this, later that year, Jones et al. published an observational study in which VV ECMO was used in patients admitted to the ICU with H1N1 influenza infection. The group reported that of those treated with VV ECMO, 71% survived to ICU discharge and approximately 47% survived to hospital discharge.[7] This finding identified that ECMO can be a viable option in certain patients with consideration to age, gender, medical comorbidities, and etiology of respiratory failure.

In 2018, the widely publicized multicenter RCT, ECMO to Rescue Lung Injury in Severe ARDS (EOLIA) trial, comparing standard medical management to VV ECMO in patients with severe ARDS was published. While this study did show a reduction in 60-day mortality in the group treated with ECMO, this finding was not statistically significant (p-value 0.07) and ultimately, the study was terminated for futility.[8,9] Though this study did not show significant reduction in mortality, it did highlight that patients not responding to optimal standard-of-care management in the setting of severe lung injury may benefit from early consideration of ECMO and showed evidence of clinical improvement within a few hours of its initiation.[10] Since this trial, multiple case studies have continued to document positive outcomes when using ECMO; however, additional RCTs are needed to further illustrate its efficacy and identify optimal candidates for its use.

What is ECMO and How Does It Work?

ECMO works by draining deoxygenated blood from a vein, pumping this blood through a membrane oxygenator which removes carbon dioxide and supplies oxygen, and reintroducing the newly oxygenated blood to a patient's vein or artery.[11] This is performed through a circuit that consists of a blood pump, membrane oxygenator, internal tubing system, heat exchanger, and drainage and return cannulae.[12] ECMO can be divided into two main categories, VV ECMO and veno-arterial (VA) ECMO. VV ECMO is indicated in patients with significant isolated lung injury and provides respiratory support but not circulatory support. Thus, consideration for this modality should be made in patients suffering from respiratory failure, but in whom cardiac function is sufficient.[11] In contrast, VA ECMO is used in patients with cardiac or cardiopulmonary failure and provides circulatory support as well as respiratory support.[11]

ECMO devices use either a roller or centrifugal pump. Roller pumps function through continuous peristalsis of an inner tubing system which moves blood through the distal portion of the compressed region. Deoxygenated blood travels from this pump to the membrane oxygenator, where the blood is decarboxylated and oxygenated, and is then delivered back to the patient.[13] Most well-funded centers, however, use a centrifugal pump, which is smaller and utilizes more advanced technology.[13] Centrifugal pumps work by establishing a pressure differential that drives blood through a revolving impeller which further moves blood through the ECMO unit and return cannulae.[13] Ironically, while centrifugal pumps require lower doses of heparin and are associated with decreased levels of hemolysis, they have been shown to result in higher rates of gastrointestinal, pulmonary, and intracranial hemorrhage.[14] Another important component of the ECMO circuit is the membrane oxygenator which functions in lieu of a patient's lungs to remove carbon dioxide and oxygenate blood. Currently, most ECMO oxygenators use a polymethylpentene membrane, which is a more durable material, allows for better gas exchange, and results in reduced rates of anemia and coagulopathy than other options.[15]

Cannulation techniques and the associated resources required differ significantly depending on the type of ECMO employed. In VV ECMO, cannulation can be performed with either single lumen or double lumen catheters. When using single lumen catheters, carboxylated blood is typically drained from a cannulated femoral vein and decarboxylated blood is returned to the right internal jugular vein (IJV).[15] In comparison, a single dual lumen catheter can be inserted into the right IJV with one lumen functioning to drain blood and the other to return it.[16] Use of

It is imperative that professionals in the ED are familiar with ECMO in order to provide optimal care for the appropriate candidates.

dual lumen catheters has been increasing in adult ECMO as this method results in decreased recirculation phenomenon.[16] Recirculation results when, instead of providing support to systemic circulation, oxygenated blood is infused into the patient through a return cannula and exits immediately back to the ECMO circuit through the drainage cannula, creating a closed loop of circulation within the ECMO system.[16] Other advantages of this approach include reduced sites for potential infection and increased mobility for the patient.[16] Both single and dual lumen cannulation can be performed percutaneously by a trained provider at the bedside; however, transesophageal echocardiography or fluoroscopy is needed for accurate placement of the dual lumen cannula.[15]

VA ECMO cannulation can be performed either centrally or peripherally, each with its own potential risks, benefits, indications, and resources required. In central cannulation, blood is typically drained from the right atrium and returned to the proximal ascending aorta. A surgeon, anesthesiologist, and staffing for the operating room are typically required to perform this type of cannulation as direct access to the right atrium and aorta involves a sternotomy.[17] In contrast, peripheral cannulation can be performed using a percutaneous approach by a medical provider at bedside.[18] This involves cannulation of the proximal femoral or jugular vein for drainage of deoxygenated blood and carotid, femoral or axillary artery cannulation for delivery of newly oxygenated blood back to the patient.[19] One major complication in femoral arterial cannulation is distal limb ischemia.[20] To mitigate this risk, providers may choose to place a distal perfusion catheter on the same side of femoral artery cannulation most often in the superficial femoral artery.[20] Another important consideration in peripheral VA ECMO is coronary and cerebral hypoxia as a result of blood mixing. Blood mixing occurs when oxygenated blood from the ECMO device combines with blood ejected from the patient's left ventricle.[21] When myocardial function is significantly impaired, this mixing point typically occurs at the proximal ascending aorta and does not result in clinically significant cerebral hypoxia.[21] However, as myocardial function improves, the mixing point moves to the aortic arch, resulting in deoxygenated blood being pumped to coronary and cerebral circulation.[21] For this reason, it is important to frequently monitor arterial blood gases from the right upper extremity in order to identify and address cerebral or coronary hypoxemia.[22]

When using VA ECMO, there is also risk of increased left atrial and left ventricular end-diastolic pressures resulting in left heart distention.[23] A prophylactic approach to this involves placing an Impella to decompress the left ventricle or a transseptal drain to preserve normal left atrial pressure.[23] For this reason, pressures in the left atrium and left ventricle and the chambers sizes should be closely and regularly followed using echocardiography. These are just a few examples of how physiology is manipulated with ECMO and some of the associated complications. Thus, it is imperative to carefully assess risks, cost, and resources required before implementing this intervention.

Common Indications for ECMO

Consideration to initiate ECMO depends largely on patient risk factors, response to resuscitative efforts, and pathology being addressed. In the setting of cardiogenic shock or cardiac failure refractory to optimal medical management, VA ECMO may be appropriate.[24,25] Certain pathologies that seem to benefit from VA ECMO include "acute coronary syndrome (ACS), cardiac arrhythmias, sepsis with cardiac depression, drug toxicity with profound cardiac depression, myocarditis, pulmonary embolism, cardiac trauma, acute anaphylaxis, post-surgical cardiac complications, primary cardiac allograft failure, and cardiac cardiomyopathy."[26]

In contrast, patients with acute respiratory failure not responsive to initial aggressive interventions may benefit from VV ECMO. Common pathologies that have shown benefit with the use of VV ECMO include "ARDS (bacterial or viral pneumonia, aspiration events, alveolar proteinosis), lung rest from pulmonary contusion, smoke inhalation and airway obstruction, post-lung transplant with primary graft failure, bridge to lung transplant, lung hyperinflation in setting of status asthmaticus, pulmonary hemorrhage or massive hemoptysis, congenital diaphragmatic hernia, and meconium aspiration."[26]

Role in the ED

In ED setting, physicians are tasked with initiating the appropriate treatment plan for their patients in a timely manner. Patients who present with severe illness often require immediate intervention with frequent reassessments for improvement or worsening of their initial presentation. In some scenarios where patients continue to clinically deteriorate despite vigorous resuscitative efforts, further cardiorespiratory support in the form of ECMO may be useful.

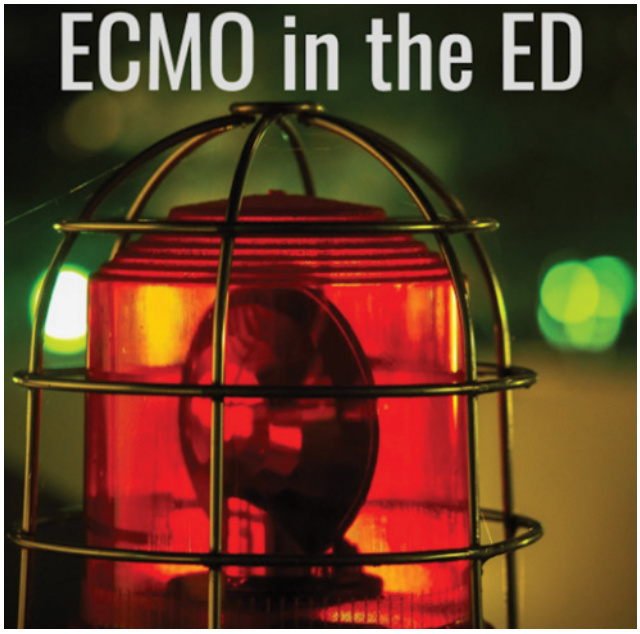
While the underlying mechanics remain the same, ECMO performed in the ED is termed either extracorporeal life support (ECLS) or extracorporeal cardiopulmonary resuscitation (ECPR). ECLS describes the use of ECMO as a temporizing measure in the critically ill patient.[27] ECPR is the term applied when VA ECMO is initiated in the setting of cardiac arrest.[27] Even when optimal cardiopulmonary resuscitation (CPR) is performed, only a fraction of normal cardiac output is achieved, rendering the patient susceptible to significant anoxic brain injury and multi-system organ failure.[27] The fraction of normal

cardiac output (CO) attained in the setting of CPR is approximately 30%. This is associated with a cardiac index of 0.6L/min/m^2 compared to 2.0L/min/m^2 when using ECPR (normal cardiac index is 2.5-4L/min/m^2).[28] In the setting of out-of-hospital cardiac arrest, global survival rates are 2-11%.[29] In contrast, between the years 1990 and 2012, the Extracorporeal Life Support Organization (ELSO) reported survival rates of 27% when ECPR was initiated.[30] In 2017, Wang et al. published a meta-analysis of six studies comparing ECPR to conventional CPR and found significant improvement with regards to survival rate to discharge and neurologic outcome when ECPR was employed.[31] This study highlighted a number of additional factors contributing to the better outcomes including early recognition of pulselessness, time to CPR, immediate defibrillation, initial rhythm, etiology of cardiac arrest, and time to ECPR.[31] Kuroki et al. further posits that a unique advantage of ECPR is the ability to use this technology to simultaneously performing coronary angiography and fibrinolysis which may also contribute to treating the underlying pathology leading to the cardiac arrest.[32] While more studies are needed to further identify the suitable candidates for whom positive outcomes can be achieved and the appropriate timelines for such interventions, present data suggests that ECPR may play an essential role in effective management of cardiac arrest in the ED.

ECLS is another therapeutic option available for use in the ED. In 2016, Allen et al., in collaboration with The American College of Emergency Physicians commented on the use of ECMO and ECLS in the ED and specified patients who may be appropriate and ethical candidates.[33] In this paper, Allen proposed that the decision to initiate ECMO should be made quickly and with the primary goal of being used as a bridge to definitive therapy for patients in whom the provider anticipates a meaningful outcome.[33] This article also reported that the current data would support clinical consideration for ECLS or ECPR in patients who "are 18 to 70 years old, have a witnessed arrest, have ventricular fibrillation or ventricular tachycardia as their initial rhythm, have a presumed cardiac cause, and have received high-quality CPR delivered with minimal interruptions."[33] While this publication recommends that eligibility for ECMO should be restricted to a rather small cohort of patients, it also suggests that candidacy is ultimately best determined by a clinician's overall gestalt and comfort with initiating this intervention.

Conclusion

The use of ECMO has been expanding in the advent of more advanced technology and increased training opportunities for intensivists, surgeons, and emergency medicine physicians, leading to its implementation at more health care centers. Access to ECMO centers is growing, and its early initiation has demonstrated significant positive impact on patient outcomes in a number of clinical trials and case reports. Emergency physicians are considered experts at resuscitating patients with the most severe presentations of both acute and chronic pathologies, and are tasked with maintaining a broad knowledge of available interventions that may be life-saving or life-altering. For this reason, ED physicians should consider ECMO for patients in cardiac arrest or those suffering from acute cardiopulmonary failure refractory to conventional methods of resuscitation and in whom a meaningful recovery is anticipated if they can be bridged to definitive therapy. Future studies are needed to further identify the appropriate candidates for ECMO in the ED, its efficacy in certain pathologies, and its usefulness as a therapeutic modality in the initial intervention of a patient in cardiac arrest. ✶



Access to ECMO centers is growing, and its early initiation has demonstrated significant positive impact on patient outcomes in a number of clinical trials and case reports. It's imperative for health care professionals in the ED to be familiar with this intervention.

References available online

FROM NARCAN TO ECMO
A Case of Opioid Reversal-Induced Acute Respiratory Distress Syndrome

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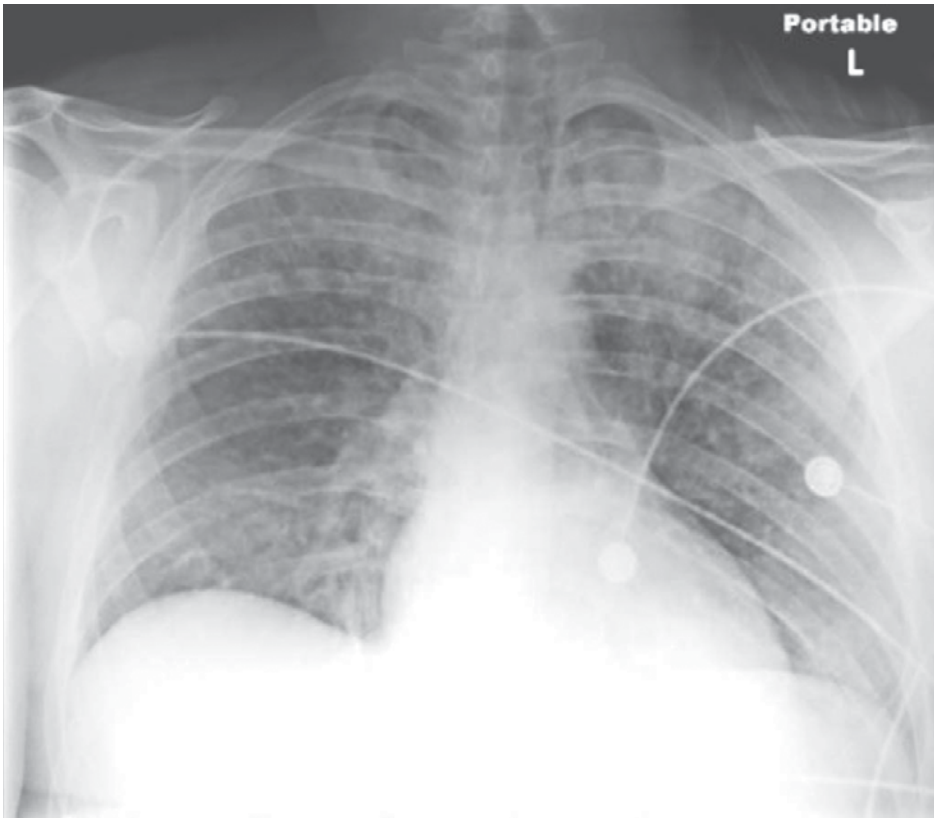


The rise in heroin and opioid overdoses throughout the country demands an awareness of the sequelae of naloxone-related pulmonary edema that can occur in opiate overdose reversal.

Case Description

A 32-year-old male visiting from out of state was brought in by EMS after a friend found him passed out in the bathroom. The patient was given 8 mg of Narcan by EMS with no response and the patient arrived in the ED with EMS using a bag-valve mask to assist in respiration.

Initial vital signs were HR 116, RR 22 (by EMS), SpO2 86%, and BP 118/80. Shortly after arriving in the ED, with no further intervention, the patient became awake, alert, oriented, and began speaking in full sentences. Repeat vital signs were HR 103, SpO2 94%, and BP 125/66. The patient admitted to marijuana, cocaine, and IV heroin use. The physical exam was pertinent for an alert patient with dried blood in the bilateral nares and with equal and reactive dilated pupils. The patient was tachycardic, but in normal rhythm with no murmurs, rubs, or gallops. His pulmonary exam was significant for initially slow, but then normal respiratory rate, with rhonchi bilaterally, and he had an unremarkable abdominal and extremity exam. Once the patient became alert, no focal neurological deficits were noted.

Initial laboratory work was significant for a leukocytosis with WBC 14.28, and a toxicology screen positive for benzodiazepines, cocaine, cannabinoids, and opiates. His initial lactic acid was 5.7, the Basic Metabolic Panel was significant for a potassium level of 5.5, and his blood gas on arrival was pH 7.34/PaCO2 48/PaO2 97.

While being observed in the ED, the patient's SpO2 began to decrease to the low 90s. Repeat ABG demonstrated a developing respiratory acidosis with pH 7.21/PaCO2 66/PaO2 65. A chest X-Ray revealed bilateral patchy infiltrates, concerning for ARDS. He was placed on bipap, with initial improvement in oxygen saturation. However, when the patient was evaluated by the MICU team, he was in increasing respiratory distress, and the decision was made to intubate. Per ARDS protocol, the patient was placed on the ventilator with low tidal volumes and high PEEP, and he was admitted to the MICU.

During his third day following ARDS protocol on mechanical ventilation, the patient developed pneumomediastinum and subcutaneous emphysema in the setting of worsening hypoxemia. The patient was placed on nitric oxide treatment overnight as a bridge to ECMO. His respiratory failure did not respond to inhaled nitric oxide, paralysis, or to steroid treatments. He underwent multiple bronchoscopies for mucous plug removal, and cultures showed evidence of Pasteurella *bacterial infection, indicating aspiration pneumonia. He continued to clinically deteriorate with worsening hypoxemia, and on day 5 of admission, he was placed on V-V ECMO.*

After 4 days of ECMO therapy, he was successfully weaned off, and he was extubated 5 days later. The patient was discharged home 6 days later with no oxygen requirements and in a stable condition.

Discussion

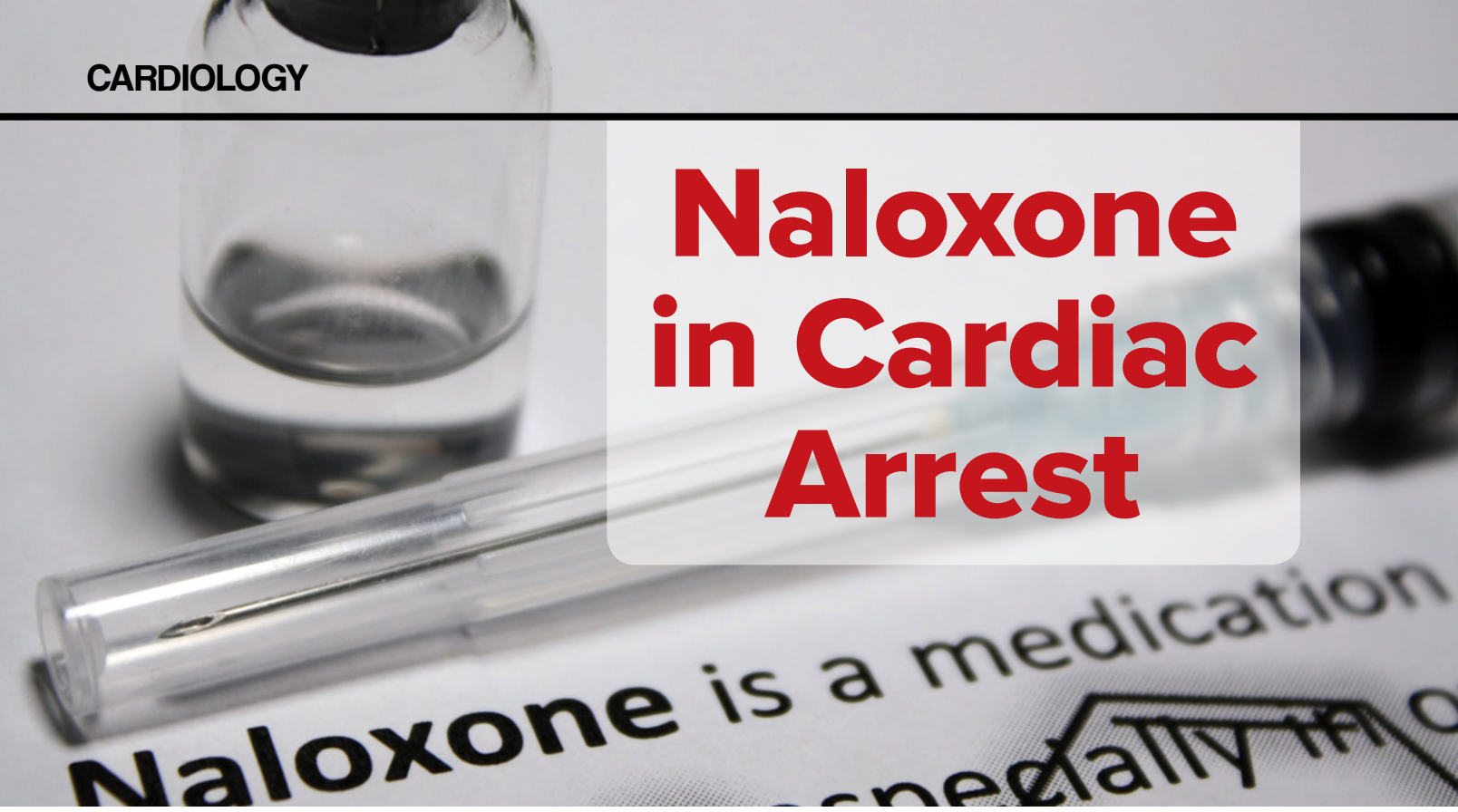
The literature on the subject of non-cardiogenic pulmonary edema and that regarding ARDS in the context of opiate overdose reversal is slowly developing; however, the explanation is most likely multifactorial. While animal studies have discussed the effects of Narcan on pulmonary capillary permeability and the phenomenon of pulmonary edema, robust literature has not well described the phenomenon in humans.[1,2] Some of the theories suggest that a catecholamine release occurs after opioids are reversed by Narcan, leading to a rapid increase in cardiac output and pulmonary pressures, ultimately increasing the risk for ARDS. Other theories describe leaking of pulmonary capillaries and subsequent accumulation of protein-rich pulmonary fluid also increasing the risk for ARDS. The negative pulmonary pressures that result from an upper airway obstruction, due to obstructive secretions or mucous plugs, a closed glottis, or glottis laxity, can also complicate the issue.[3]

The most common etiologies for ARDS include sepsis, aspiration, near-drowning, pneumonia, and barotrauma. In our case, bronchoscopy cultures showed evidence of *Pasteurella* bacteria, suggesting aspiration pneumonia as a precipitating cause of the patient's ARDS. It is important to realize, however, that ARDS can result independent of aspiration in this population as well.

A case series conducted over 4 years by Sporer and Dorn analyzed 27 patients who developed non cardiogenic pulmonary edema within 24 hours of their presentation for heroin overdose. 74% of these patients were hypoxic on arrival to the emergency department and 22% developed symptoms of respiratory distress within the first hour. While 33% of patients required mechanical intubation, the majority were able to be extubated after 24 hours. In order to optimize a patient's respiratory status in the setting of opioid induced noncardiogenic pulmonary, endotracheal intubation should be considered early in the course of presentation.[4]

The use of ECMO in patients with refractory hypoxemia on mechanical ventilation secondary to opiate overdose is rarely needed. Yet, as we have seen in this case report, it can be a lifesaving alternative when a patient is failing standard ARDS treatment on mechanical ventilation.

ARDS requiring ECMO in opiate-overdose patients is becoming an increasingly recognized treatment strategy. A recent case report describes a similar case requiring V-A ECMO for ARDS secondary to aspiration pneumonia after an opioid overdose.[2] In another case study from Egypt, a patient with ARDS secondary to opiate overdose was successfully weaned off V-V ECMO and extubated after 12 and 14 days, respectively. Similar to our case, both patients were successfully discharged without supplemental oxygen requirements.[5,6] Therefore, it is important to note that, similar to other recommendations in ARDS treatment, transfer to an ECMO center should be considered in the case of opioid-induced ARDS refractory to mechanical ventilation. ✶



Naloxone in Cardiac Arrest

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On an EMS rotation, you are dispatched to a call for a 26-year-old male, cardiac arrest — CPR in progress. On arrival, you note a man supine on the porch of a rundown house. He is apneic and pulseless, skin is ashen. No rigor is noted. A bystander on scene is attempting to perform CPR by 911 dispatch instructions, but he appears intoxicated. Paramedics take over CPR, placing the patient on their cardiac monitor. His initial rhythm is asystole, but after two rounds of epinephrine converts to pulseless electrical activity. He is intubated, with an EtCO2 of 23. While paramedics continue to work the code, bystanders state that the patient was recently released from jail and was using heroin, and that it was a "bad batch." They used half an hour prior and found their friend unresponsive upon awakening. The paramedics turn to you for medical direction — should they administer naloxone?

The Centers for Disease Control and Prevention found that more than 70,000 people died from drug overdose (OD) in the United States in 2017 — more than those who died from gun violence or motor vehicle collisions.[1] More than 47,000 died from opioid overdose, with approximately 28,000 involving synthetic opioids (other than methadone). This staggering rise in mortality corresponds to increased access to powerful synthetic opioids such as fentanyl. Fentanyl began trickling into the illicit drug supply a decade ago, but many municipalities have found fentanyl-associated deaths now exceeding those due to oxycodone and heroin.[2] National mortality due to fentanyl nearly doubled from 2016 alone.[1] Nationally, opioid overdose ED visits increased by nearly 30% from the third quarter of 2016 to the third quarter of 2017, even more so in the U.S. Southeast.[3] (Figure 1) One large public hospital in Miami saw a nearly fivefold increase in naloxone use between 2015 and 2016,[4] while EMS use of naloxone in the same county increased more than tenfold.[5]

The main approach to combating this overwhelming rise in mortality has been through needle exchange programs, health departments, and even hospital emergency departments and EMS distributing naloxone to people at greatest risk of witnessing or experiencing an opioid OD.[6,7] Previously a medication only available in the medical setting, increasing naloxone availability has complicated Good Samaritan resuscitation for the layperson rescuer when faced with an unresponsive patient.

The 2015 American Heart Association (AHA) Emergency Cardiac Care (ECC) guidelines address this complexity directly, recommending that despite a lack of evidence that "administration of naloxone will help a patient in cardiac arrest... empiric administration of IM or IN naloxone to all unresponsive opioid-associated resuscitative emergency patients" may be a reasonable adjunct to standard first aid and non-healthcare provider basic life support protocols, but that "CPR should take precedence over naloxone administration as patients without a palpable pulse may be in cardiac arrest or may have an undetected weak or slow pulse."[8]

While instructive for the layperson rescuer, this guideline references a historical conundrum surrounding naloxone use in patients suffering cardiac arrest secondary to opioid OD. For professional rescuers in either the prehospital or hospital settings, naloxone remains a constituent of the AHA Advanced Cardiac Life Support (ACLS) Algorithm and is found in the list of interventions to correct reversible causes of cardiac arrest as an adjunct

to correct the hypercarbic respiratory failure caused by opioid OD. Although naloxone's efficacy in treating respiratory depression in patients with a pulse is well established, its utility in cardiac arrest remains controversial.

History of Naloxone in Cardiac Arrest

Prior to the advent of EMS equipped with ventilatory equipment and naloxone, respiratory failure in the out of hospital setting frequently resulted in death.[9] μ-opioid receptor agonists such as morphine, heroin, and fentanyl induce significant respiratory depression, responsible for the toxicity of opioid OD. Naloxone was developed in 1961 and has played a varying role in the resuscitation of opioid OD victims once the drug became available a decade later.[10] Heroin and other opiates were responsible for significant morbidity and mortality in the first half of the 20th century.[11] However, the earliest iterations of ACLS referenced naloxone only for use in the resuscitation of neonates with respiratory depression "induced by narcotics given to the mother before delivery."[9,12] It was not until the 2000 ECC guidelines that opioid OD was recognized in ACLS as a "pre-arrest poison" with the recommendation to "try to reverse respiratory insufficiency with naloxone before inserting an endotracheal tube."[13] In the years since, an epidemic of opioid OD has overtaken the United States, making naloxone one of the most important drugs in the prehospital pharmacopeia.

Many studies and practice guidelines have recommended the use of naloxone in cardiac arrest patients. However, in the 2015 ACLS guidelines, the AHA endorses specific indications for empiric naloxone administration in the peri-arrest setting:

- **"It may be reasonable to administer IM or IN naloxone based on the possibility that the patient is not in cardiac arrest. (Class IIb, LOE C-EO)**
- **Standard resuscitative measures should take priority over naloxone administration, with a focus on high-quality CPR (compressions plus ventilation). (Class I, LOE C-EO)**
- **We can make no recommendation regarding the administration of naloxone in confirmed opioid-associated cardiac arrest. Patients with opioid-associated cardiac arrest are managed in accordance with standard ACLS practices."[14]**

In the absence of a clear recommendation on naloxone in confirmed cardiac arrest patients, physicians are left to the literature base to support decision-making when faced with patients in cardiac arrest secondary to presumed or known opioid OD.

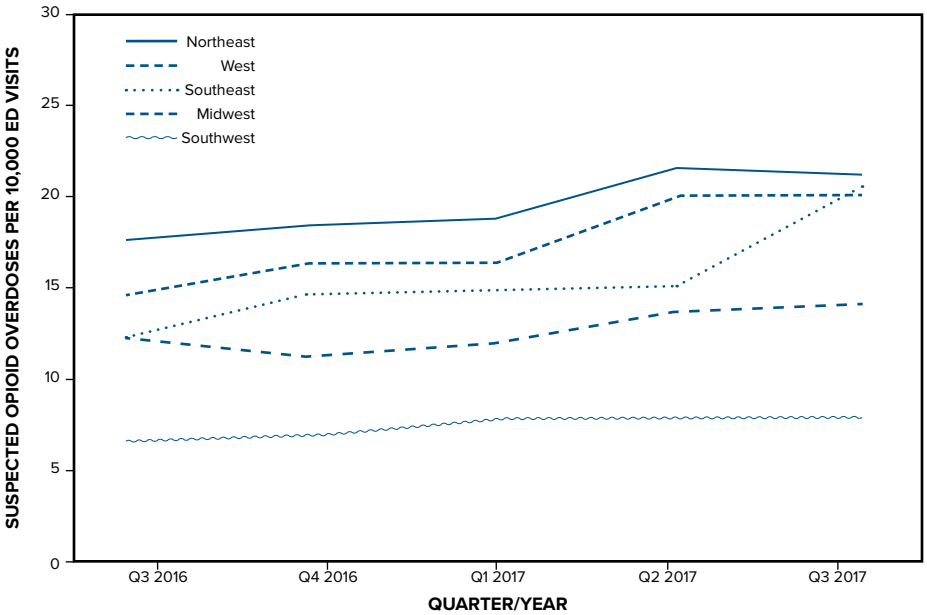

FIGURE 1. Quarterly rate of suspected opioid OD, by U.S. region — 52 jurisdictions in 45 states, National Syndromic Surveillance Program, July 2016–September 2017[3] (Public Domain).

Evidence Supporting Naloxone Use in Cardiac Arrest

Naloxone is a potent opioid receptor antagonist with an excellent safety profile and ability to reverse opioid-associated respiratory depression in patients. It can be administered via intravenous, intraosseous, intramuscular, subcutaneous or intranasal routes, as well as nebulized for inhalation. There are few adverse events associated with naloxone. The most prominent adverse event is the precipitation of acute withdrawal, with signs and symptoms including agitation, hypertension, tachydysrhythmias, and vomiting.[15,16] Precipitated withdrawal is particularly dangerous in opioid overdoses in the setting of polypharmacy overdose.

Reversal of opioid overdose in the presence of stimulants can trigger unopposed catecholaminergic activity with subsequent demand ischemia. Reversal of overdose in the presence of CNS depressants is particularly dangerous as patients who remain obtunded but with an iatrogenically induced predisposition for emesis are at increased risk for aspiration. Although several reports exist in the literature describing flash non-cardiogenic pulmonary edema occurring following naloxone administration, this phenomenon is rare at a rate of 0.2-3.6% in patients transported to ED following opioid overdose.[17] Documented since the 1970s, the etiology of naloxone associated non-cardiogenic pulmonary edema is still poorly understood and potentially caused by rapidly stimulated ventilatory drive in the presence of a closed glottis or a sequelae of the histaminergic activity of opioid use itself.[18]

Theoretically, administration of naloxone prior to ROSC could result in a patient who can protect their own airway upon obtaining a pulse, thereby abrogating the need for aggressive airway management, including intubation.

Several case reports and animal studies describe antiarrhythmic and positive inotropic effects of naloxone

in cardiac arrest. [19,20] Likewise, a retrospective chart review of emergency medical dispatches for cardiac arrest in which patients received naloxone found that 42% of patients who received naloxone in the prehospital setting had an improvement in electrocardiogram (EKG). Furthermore, "of the participants who responded to naloxone, 47% demonstrated EKG rhythm changes immediately following the administration of naloxone." These rhythm changes were inconsistent and included sinus tachycardia, accelerated junctional rhythm, PEA, ventricular tachycardia and ventricular fibrillation.[21]

Two other studies on demographics of out-of-hospital cardiac arrests (OHCA) found that patients in cardiac arrest secondary to a presumed opioid OD had improved odds of survival to ED and survival to hospital discharge. The first found an odds ratio of 1.21 (P=0.66) for survival to ED, while the other found a higher rate of survival to hospital discharge (19% vs. 12%, p=0.014) versus non-overdoses.[22,23] These findings support the recommendation to administer naloxone in opioid associated OHCA, because given "low rates of [ROSC] and survival during cardiac arrest, any potential intervention leading to rhythm improvement is a reasonable treatment modality."[21] Baseline prognostic factors in cardiac arrest secondary to opioid overdose are poor and outcomes are often dismal. However, this is not an acceptable reason to limit use of a pharmacologic agent with potential mortality benefit: A review of patients who survived OHCA secondary to OD with or without naloxone found that outcomes were "no worse than after non-OD OHCA, and among survivors a majority had a good neurological outcome."[22]

Evidence Opposing Naloxone Use in Cardiac Arrest

While the results of the previous studies are compelling, it is important to consider the strength of the evidence. The pharmacological data underlying a purported efficacy of naloxone in achieving ROSC is ambiguous, and contradictory: the animal model demonstrating higher rates of ROSC in the presence of naloxone also noted ROSC duration to be 1.5 times shorter versus epinephrine alone or saline.[19] While groundbreaking as the first human cohort observed, Saybolt et al.'s results are low-powered and a prospective trial with fewer confounders would be necessary to demonstrate effect. Although in many cases observed rhythm changes occurred immediately following naloxone administration, the number of pharmacologic agents involved in ACLS, each with distinct pharmacokinetics, makes causality of the naloxone intervention difficult to establish. Likewise, within the epidemiologic literature, there are many confounding factors that may have played a greater role in opioid-associated OHCA patient outcomes; specifically, OD OHCA patients were on average significantly younger, had fewer medical comorbidities, were more likely to present with non-shockable rhythms and had worse baseline neurological function as measured by Glasgow Coma Scale. [22] Further, one of the demographic studies notes that among OD OHCA patients, 40 (47%) received naloxone and naloxone use was not associated with survival (P = 0.54).

The AHA asserts that in most settings, it is difficult to establish that the patient's "clinical condition is due to opioid induced CNS and respiratory depression toxicity alone, and [providers] might therefore misidentify opioid-associated cardiac arrest as unconsciousness or vice versa… particularly where determination of the presence or absence of a pulse is unreliable." [8] This difficulty may further confound providers when considering administration of naloxone, as what may have appeared to be an effective treatment for cardiac arrest merely reversed severe CNS depression. While naloxone is an uncertain treatment in patients without a pulse, it is clear is that naloxone reverses respiratory depression in patients with a pulse. A patient in cardiac arrest due to apparent OD overdose should be treated for a respiratory cause of cardiac arrest. The treatment for respiratory failure centers around airway management, which necessitates controlling the patient's oxygenation and ventilation. This may be achieved via endotracheal intubation or placement of a supraglottic airway to ensure adequate ventilation and oxygenation. Given the many harms associated with intubation, it may be prudent to reserve placement of an advanced airway until later in a resuscitative effort. Empiric naloxone use does not improve oxygenation or ventilatory drive in a pulseless patient, and as such does not improve not add anything to the treatments the patient is already receiving. By placing a focus on rapid administration of naloxone in cardiac arrest patients with presumed opioid OD, providers may be predisposed providers to diagnostic inertia and distracted from evidence-based methods to improve care in cardiac arrest: namely, early recognition, high quality CPR, and early defibrillation.

Conclusions

With the rapid rise in opioid-associated deaths and widespread dissemination of naloxone, emergency care professionals are faced with the decision whether or not to administer naloxone to patients in cardiac arrest after a presumed OD. With scant evidence to support or refute the empiric use in opioid OD-associated cardiac arrest, health care professionals should tailor their approaches to cardiac arrest to the etiology of arrest, rather than empiric intervention. Even so, the evidence supporting naloxone may point towards a possible role for the drug. Further investigation, including a prospective clinical trial, would be necessary to investigate this role. In the meantime, it is critical to focus attention to the treatments we know work best.

Case Conclusion

You recommend against administering naloxone because the patient is already intubated. Following the third round of epinephrine, the patient develops a perfusing rhythm with palpable peripheral pulses, and an initial GCS of 3. He is loaded into the ambulance and paramedics transport him to the local hospital ED. After 16 days in the intensive care unit he demonstrates significantly improved mental status. He is extubated and transferred to a rehabilitation floor for physical and occupational therapy. ✶

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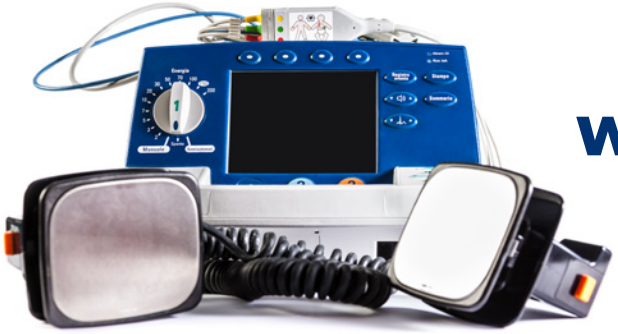

What to Consider with Double Sequential Defibrillation in Refractory VF

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A 54-year-old male suffered witnessed cardiac arrest while shoveling snow. Bystanders initiated CPR; EMS administered 4 defibrillations and maximal doses of antiarrhythmic agents before bringing the patient into the hospital in refractory ventricular fibrillation (VF). Double sequential defibrillation (DSD) was attempted, with successful return of spontaneous circulation (ROSC) after cardiac arrest. The patient was discharged home neurologically intact.

Introduction

In out-of-hospital cardiac arrest, the administration of rapid, high-quality CPR dictates outcomes of Adult Cardiopulmonary Life Support (ACLS). Patients in arrest with a shockable rhythm (ventricular fibrillation [VF] or ventricular fibrillation [VT]) have a higher rate of survival.[1] However, a subset of patients with VT/VF do not respond to CPR, medications, or defibrillation; these are considered to be in refractory VF. A recent chart review demonstrated that up to 0.5% of patients receiving out-of-hospital CPR are found to be in refractory VF.[2] Improved out-of-hospital CPR technique and increased bystander CPR have increased chances of survival for out-of-hospital arrest. Patients have a longer window in shockable rhythms with good neurological outcomes, which has created opportunities for initiation of external defibrillation and potentially treatment with double sequential external defibrillation (DSD). For this case we define DSD as the use of 2 sets of defibrillation pads: one set in the standard positioning and a second set either lateral or orthogonal (anterior/posterior) to the first. While standard defibrillation involves the delivery of electrical current via a single asynchronized electrical cardioverter, DSD uses the 2 separate defibrillators to fire nearly simultaneously, allowing for greater energy transmission and augmented vectors of energy to travel through the heart.

Multiple case reports and review articles have described Refractory VF.[3] The use of DSD has been postulated as a method for improvement of treatment for these patients. Multiple theories suggest that this method may benefit select patients. During DSD, the patient's myocardium receives broader and multiple energy vectors, which could help reset the electrical rhythm. In addition, it may lead to more complete depolarization of the myocardium or decrease the defibrillation threshold, making a second shock more effective. A recent retrospective cohort analysis showed there was no added harm from DSD and single defibrillation in patients with refractory VF after three 200 J shocks.[4] In a recent retrospective chart review of 12 patients in refractory VF, 9 out of 12 were converted and 3 out of 12 had ROSC.[2]

Although DSD has not demonstrated direct harm to the patient, a recent case report has shown that DSD may lead to damage of each defibrillator.[5] Such reports raise concern for patient safety if damage is not identified prior to the next attempted use. Reported damage to defibrillators may be due to positioning of defibrillation pads; the pads may be placed too close together, resulting in diversion of current between the devices instead of through the myocardium.[5] In this index case report, the two devices involved were from different manufacturers with proprietary programming, which may also play a role in the resultant damage. Currently, DSD is considered off-label use as manufacturers do not test devices in this manner and cannot ensure reliable functionality during DSD. The off-label use of DSD may lead to voiding of warranties if there is any damage. Due to these concerns, explicit policies should be developed to specify appropriate indications for DSD, correct pad placement, and post-DSD testing of devices prior to returning to service.

Conclusions

With improvements in out-of-hospital emergency systems and emphasis on high quality CPR, patients have the potential for good neurologic recovery despite longer arrest times. Given the high likelihood of demise in cases of refractory VF and the potential for meaningful neurological recovery with ROSC, clinicians should consider DSD for patients who are found to have failed conventional antiarrhythmic and standard defibrillation. Institutions should create protocols for the use of DSD, including specific indications, pad placement, and device testing after use. ✶

SUMMARY POINTS

- DSD should be considered for patients with refractory VF.
- DSD is the use of 2 separate defibrillators to deliver nearly simultaneous shocks.
- The pads can be placed with one in the conventional location and a second beside it, or in the anterior/posterior arrangement.
- EM teams should work with their hospital to create a protocol for the use of DSD.

References available online

Differentiating Acute versus Chronic Right Heart Failure with Bedside Echocardiography

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Case

A 65-year-old female presents to the ED with shortness of breath for 2 days. She has a mild productive cough but denies fevers or chills. She reports a history of pulmonary embolism (PE) 10 years prior, for which she was treated, but cannot remember the details of her treatment. She has not established care with a PCP in over 20 years. She is a nonsmoker and occasionally drinks alcohol. Her vital signs are as follows: Respiratory rate is 25 BPM, pulse oximeter 92%, Temperature 99 F, blood pressure 130/90, and heart rate 115 BPM.

To help determine the etiology of her symptoms, you perform a bedside echocardiogram. You place the phased array (cardiac) probe in a parasternal long axis and upon obtaining an adequate image, you are concerned that her right ventricle (RV) is dilated, which suggests pulmonary embolism. Her lab results are significant for a creatinine of 2.7 mg/dL with an unknown baseline, mild troponin T elevation at 20 ng/L, and a white blood cell count of 13 x 10^9 cells/L. Chest X-ray demonstrates mild cardiomegaly and no obvious infiltrates. Because of her elevated creatinine, you are hesitant about ordering a CTPE study. The radiology technician tells you VQ scans cannot be done on the weekends and therefore this modality is not an option for you.

Where can you go from here? Her right ventricle dilation is concerning for a pulmonary embolism (PE) but given her history, she may also have chronic pulmonary hypertension or right heart failure from another etiology. Are there ultrasound findings that can help differentiate acute versus chronic right heart strain in this patient?

Overview

There are a number of ways to evaluate for right heart function using ultrasound. There are both **qualitative** and **quantitative** methods.[1] Commonly, **qualitative** or 'eyeball' methods are used. These qualitative methods include evaluating for RV dilation, for septal flattening, and for paradoxical septal motion.[1] To evaluate for RV dilation *qualitatively*, the most commonly used views are the parasternal long axis (PSLA) and the apical 4 chamber (A4) views. In the PSLA view, the RV size is compared to that of the aorta and the left atrium; all of which should be approximately equal in size (Figure 1). In the A4 view, the RV:LV ratio should be approximately 0.6:1. An RV:LV ratio >1:1 indicates

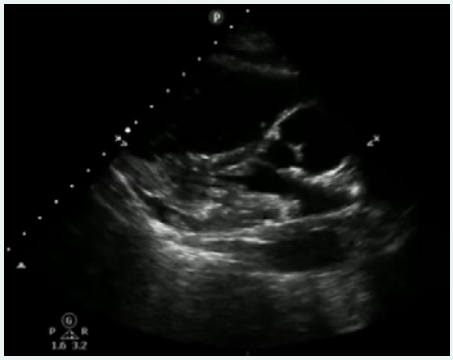

FIGURE 1. Parasternal long axis view demonstrating RV dilation. RV is notably larger than the ascending aorta and LA, which should normally appear approximately equal in size.

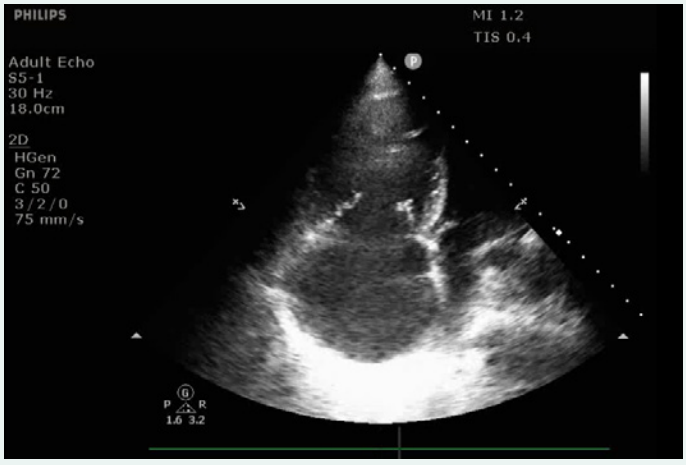

FIGURE 2. A4 view, above, with RV enlargement. Note the left heart is only partially seen given size of the right heart. The RV: LV ratio is >1:1.

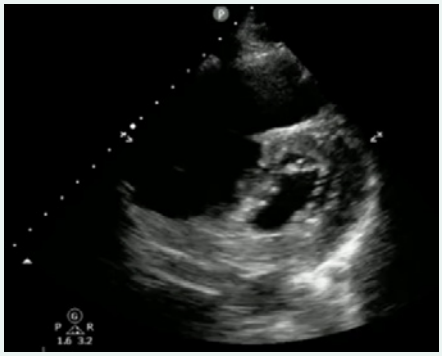

FIGURE 3. Parasternal short axis view, left, demonstrating RV dilation and 'D' sign

RV dilation and should raise suspicion for elevated pulmonary pressures (Figure 2). Additionally, evaluating for septal flattening, or the 'D' sign, can be identified in the parasternal short axis (PSS) mid-papillary view (Figure 3). Paradoxical septal motion can be visualized in both the PSS, subxiphoid, and A4 views, caused by a dilated RV that bows into the LV during LV end diastole.

There are also **quantitative** methods that can be used to evaluate right heart function.[1] Quantitative size measurements include measuring the diameter and the area of the right ventricle, the area of the right atrium, and the thickness of the right ventricle.[2] Right ventricle systolic function can be evaluated and is equally as important as the above listed measurements[2]. Though not covered in depth here, Tricuspid Annular Plane Systolic Excursion (TAPSE), Systolic Excursion Velocity (s'), and Fractional Area of Change (FA) can be used to measure right heart systolic function. The presence and severity of tricuspid regurgitation is also important to evaluate. This value is needed to calculate right ventricular systolic pressure (RVSP) and pulmonary artery systolic pressure (PASP), in conjunction with Inferior Vena Cava (IVC) diameter as an estimate of right atrial pressure.

Acute vs Chronic RHF

There are a number of quantitative and qualitative assessments that can be used to evaluate right heart function. However, when specifically evaluating for acute versus chronic right heart failure, there are two measurements that can be useful in making this distinction. These measurements are right ventricular wall thickness and pulmonary artery systolic pressure (PASP); both of which were valuable in the diagnosis of the above-mentioned case.

RV Hypertrophy

Right ventricular hypertrophy is fairly easy to evaluate. Right ventricle hypertrophy occurs over time with chronically elevated PA pressures[1]. RV hypertrophy is not typically seen in the setting of acute right heart failure.[1]

1. How to Diagnose RV Hypertrophy on ECHO
    a. In a PSLA or subxiphoid view, measure the RV free wall from inside to outside in **end diastole**. A value > 5 mm likely indicates hypertrophy of the RV free wall. This is indicative of a more chronic process.[2,3]

A thickened RV >5mm should raise suspicion for chronic causes of right heart failure[1]. An additional measurement that can help us differentiate acute versus chronic right heart failure is pulmonary artery systolic pressure (PASP).[4,5] A PASP value of less than 35 mmHg is typically normal, and PASP of 40-60 mmHg can be indicative of pulmonary hypertension.[2] A PASP **greater than or equal to 60 mmHg** is generally consistent with longstanding pulmonary hypertension (PHTN). PASP ≥ 40 mmHg has been shown to be sensitive and specific when ruling in pulmonary hypertension.[6] PASP can be estimated with echo by utilizing continuous wave (CW) doppler and knowledge of the modified Bernoulli equation.[4]

Measuring the Pulmonary Artery Systolic Pressure (PASP)

1. Using a phased array probe, obtain either an apical A4 or PSS axis view at the level of the tricuspid valve (TV). The key is to get good visualization of the TV.
2. Place a color doppler window over the TV and RA, looking for a tricuspid regurgitant (TR) jet (Figure 5).
3. Using CW doppler, align your doppler beam so it passes through the regurgitant jet.
4. Once the beam is aligned, toggle CW mode to spectral display (typically you'll press the CW mode button a second time to toggle to this). (Figure 6)
    - Your x-axis displays time, your y-axis displays velocity. Velocities greater than zero, indicate movement towards the probe, less than zero indicate movement away from the probe.
    - Your TR velocity tracing will be negative indicating flow away from the probe. The tracing typically takes on a parabolic shape.
5. Measure the maximum TR velocity ($V = V_{TR\ Max}$). (Figure 6)
6. Use the Modified Bernoulli equation ($\Delta P = 4V_{TR\ Max}^2$) to calculate the pressure gradient (ΔP) across the tricuspid valve.
    - $\Delta P = 4V_{TR\ Max}^2$
7. RVSP is obtained by adding right atrial pressure (RAP) to ΔP. RAP is equivalent to central venous pressure (CVP)
    - $RVSP = 4V_{TR\ Max}^2 + CVP$
    - Estimate CVP by measuring the diameter and respiratory variability of the IVC from the subcostal view[2]
8. Assuming no significant pulmonary valve stenosis exists, pulmonary artery systolic pressure (PASP) approximately equals RVSP. $PASP \approx RVSP$

Summary

Find the tricuspid regurgitant jet and measure the peak velocity ($V_{TR\ Max}$). Estimate the **pressure** gradient **(**ΔP) between the right ventricle and the right atrium using the modified Bernoulli equation ($\Delta P = 4V_{TR\ Max}^2$). Add ΔP to the RAP to obtained the estimated PASP.

a. $RVSP \approx PASP$ (assuming no pulmonic stenosis)
b. $PASP = \Delta P + RAP$
c. $\Delta P = 4V^2$= Pressure gradient across tricuspid valve (Modified Bernoulli equation)
d. $V = V_{TR\ Max}$= Peak Tricuspid Regurgitant Jet Velocity
e. $RAP \approx CVP$
    i. $CVP \approx$ IVC diameter/variability
        1. See IVC/CVP table[2]

Bonus: 60/60 Sign

After evaluating for RV hypertrophy and measuring the PASP, there is one additional evaluation that can help in determining acute versus chronic right

CVP	Normal 0-5 (3) mmH	Intermediate 5-10 (8) mmHg		High (≥ 15) mmHg
IVC diameter	≤ 2.1 cm	≤ 2.1 cm	≥ 2.1 cm	≥ 2.1 cm
Collapse with Sniff	> 50%	< 50%	> 50%	< 50%

References available online

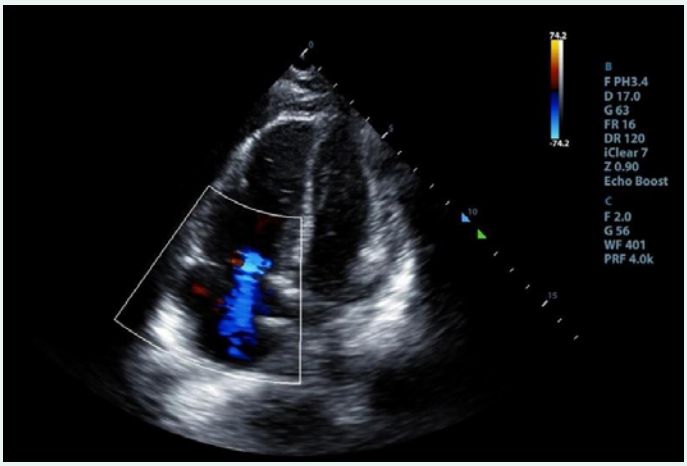

FIGURE 4. An A4 view with color doppler window centered over tricuspid valve demonstrating the presence of tricuspid regurgitation.

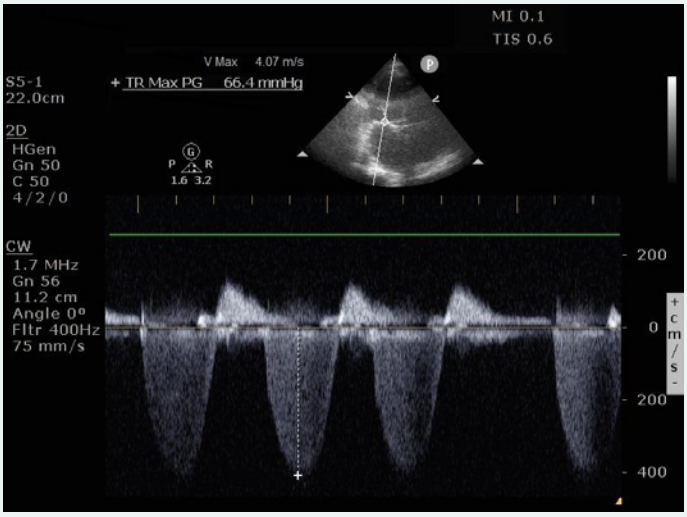

FIGURE 5. CW doppler spectral display of TV regurgitant jet velocity. Applying the modified bernoulli equation to the maximum velocity, VTR Max of 4.07 m/s, we see the pressure gradient (ΔP) across the TV is 66.4 mmHg. Add CVP to obtain PASP.

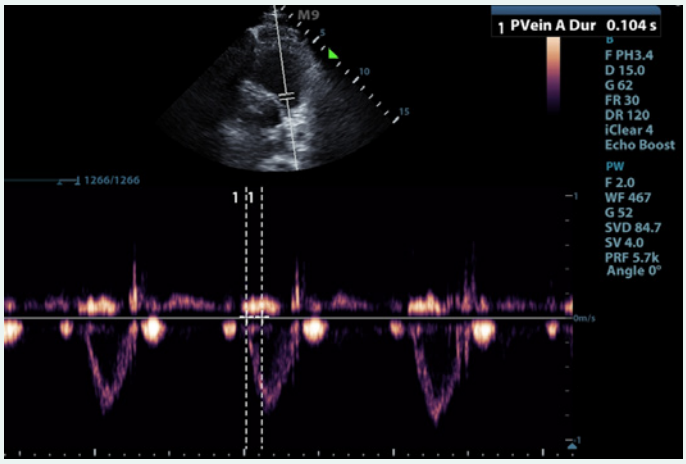

FIGURE 6. Measurement of pulmonary acceleration time in parasternal short RV inflow/outflow view. Note this example is normal with PAT >104msec.

heart failure. This evaluation is called the **60/60 sign** and requires two values: the tricuspid insufficiency pressure gradient ($\Delta P=4V_{TR\ Max}^2$) and the Pulmonic Valve Acceleration Time (PAT).[7] The PAT is the time interval from pulmonic valve opening until maximum blood flow velocity is reached. The tricuspid insufficiency pressure gradient measurement is described above in the calculation of PASP.

The PAT can be measured by first obtaining a PSS axis view at the level of the mitral valve. Once at this level, fan the probe towards the base of the heart (typically the tail of the US probe will fall towards the left hip) until the RV outflow view comes into plane. This view is characterized by visualization of the tricuspid valve, the pulmonic valve, and the "Mercedes Benz" shaped aortic valve. Place a pulse wave doppler (PW) gate centered over the pulmonic valve. Once the beam is aligned, toggle PW mode to spectral display (typically you'll press the PW mode button a second time to toggle to this). To obtain the PAT, measure the time interval from the start of blood flow to its peak velocity.

The 60/60 sign suggests that if ΔP ($=4V_{TR\ Max}^2$) and PAT are *BOTH* less than 60, acute causes of right heart failure are more likely. This is a positive 60/60 sign. One study demonstrated that in patients where acute PE was suspected, ΔP < 60 mmHg and PAT <60 msec had a specificity and positive predictive value of 94% and 90% respectively for acute PE. Greater pressures and longer times, ΔP and PAT >60, were more commonly found in patients with chronic pulmonary hypertension[7].

Case Resolution

In our patient, her $V_{TR\ Max}$ was 4.07 m/s, thus ΔP was ~66.4 mmHg. CVP was 8 mmHg.

$$PASP=\Delta P + RAP$$

$$\Delta P= 4V_{TR\ Max}^2$$

$$PASP= 74.4\ mmHg$$

TAPSE was also measured at 0.9cm indicating reduced RV systolic function (normal >1.6cm).[8]

In this acutely hypoxic patient with qualitative bedside ECHO views demonstrating right ventricular dilation and septal flattening, as discussed above, there are additional quantitative ultrasound findings that can help us differentiate acute versus chronic right heart failure. These include measurement of the pulmonary artery pressures, in conjunction with assessment of right ventricular wall thickness. Our patient had right ventricular wall thickness of 6 mm, TV gradient of 66.4 mmHg ($\Delta P = 4V_{TR\ Max}^2$) and PASP of 74.4 mmHg. Altogether, the values make chronic pulmonary hypertension and right heart failure, from her prior PE, the more likely etiology of her symptoms. Thrombolytics were not given and she was treated for acute decompensated pulmonary hypertension. Her symptoms improved while in the ED but she was admitted to the cardiology service for further monitoring.

Bottom Line

When differentiating acute versus chronic RV heart failure, think about using right ventricular hypertrophy, PASP, and the 60/60 sign. Keep in mind that there may be some exceptions but generally, RV hypertrophy >5mm, PASP >60 mmHg, and negative 60/60 sign (PAT >60 ms, ΔP >60 mmHg) are generally consistent with chronic right heart failure. ✶

A Quarrel, a Broken Heart, and a Dissection Walk into an ED...

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A healthy, 35-year-old female with a past medical history of anxiety and rheumatoid arthritis presented to the emergency department for 8/10 substernal chest pressure and dizziness that began 45 minutes prior to arrival. Her chest discomfort began while arguing with her brother at a family gathering. She initially attributed her symptoms to anxiety. She developed more severe chest discomfort and dizziness while preparing to leave the party and yelling at her son to put on his shoes. She denies nausea, diaphoresis, or dyspnea. There was no pain radiation or tearing quality. The patient has no history of diabetes, hypertension, tobacco abuse, or family history of premature coronary artery disease. She denies alcohol or illicit drug use.

On initial evaluation, the patient was mildly hypertensive (BP 141/93), but otherwise hemodynamically stable. The initial ECG showed ST-elevation in leads II, III, aVF, V5, and V6 (Figure 1), indicating inferolateral wall injury pattern. An immediate point-of-care echocardiogram was performed that demonstrated normal chamber size and contractility. There was no pericardial effusion or obvious wall motion abnormality, including apical ballooning, which was highly suspected based on the patient's age and history of present illness. A cardiologist was consulted, and the patient was taken immediately to the cardiac catheterization laboratory. Her initial troponin-T was 0.037 ng/mL (just 0.007 above the upper limit of normal). Other routine labs, including a creatinine, were unremarkable. Left ventriculography revealed an ejection fraction of 30–35% with left ventricular apical ballooning, consistent with Takotsubo syndrome.

Diagnosis: Simultaneous spontaneous coronary artery dissection (SCAD) and Takotsubo syndrome! To date, there have only been 15 published case reports of these two diagnoses occurring simultaneously.

Following her cardiac catheterization, the patient was admitted for further workup including cardiac MRI which revealed transmural infarction of a portion of the inferolateral wall. A comprehensive echocardiogram 30 hours after arrival showed resolved wall motion abnormalities and an improved ejection fraction of 55%. Her ECG changes resolved. She was then discharged with cardiac rehabilitation.

PEARLS

- Look for risk factors for SCAD and Takotsubo in women presenting with chest pain without a history of atherosclerosis.
- Use POCUS to look for apical ballooning or wall motion abnormalities.
- Don't delay ECG and troponin.

DISCUSSION

This case demonstrates two important causes of ST-elevation in women without atherosclerosis: spontaneous coronary artery dissection (SCAD) and Takotsubo syndrome. While Takotsubo syndrome causes ACS and ST elevation, infarction is often not found. It is estimated that Takotsubo syndrome is present in 5-10% of women with acute coronary syndrome. SCAD most often causes both ST-elevation and myocardial infarction (STEMI). A recent study showed that 24% of acute MI's in women <50 years old were due to SCAD. The most recent evidence also shows that both conditions

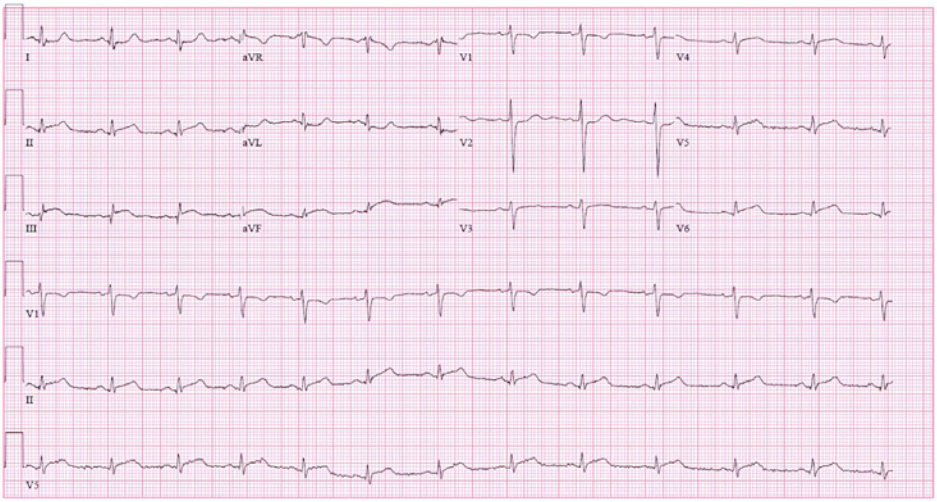

FIGURE 1. Initial ECG
Coronary angiography revealed an abrupt occlusion of the distal branch of the obtuse marginal branch of the left circumflex. A linear lucency was noted, suggesting a dissection and hematoma.

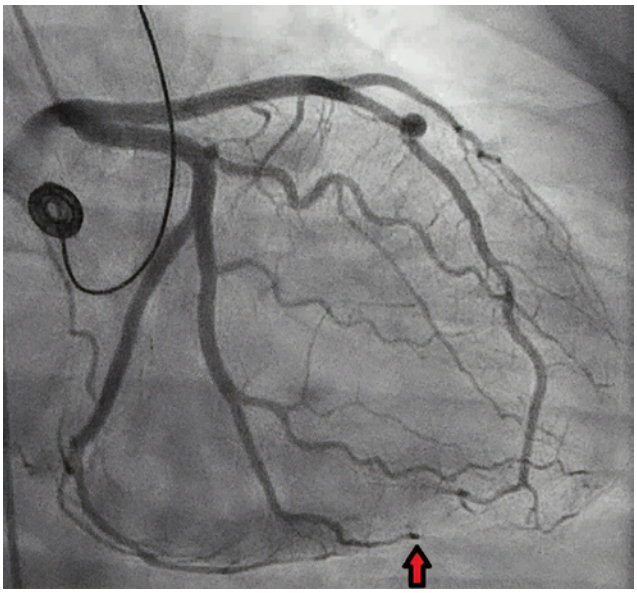

FIGURE 2. Still image from left coronary angiogram showing spontaneous dissection.

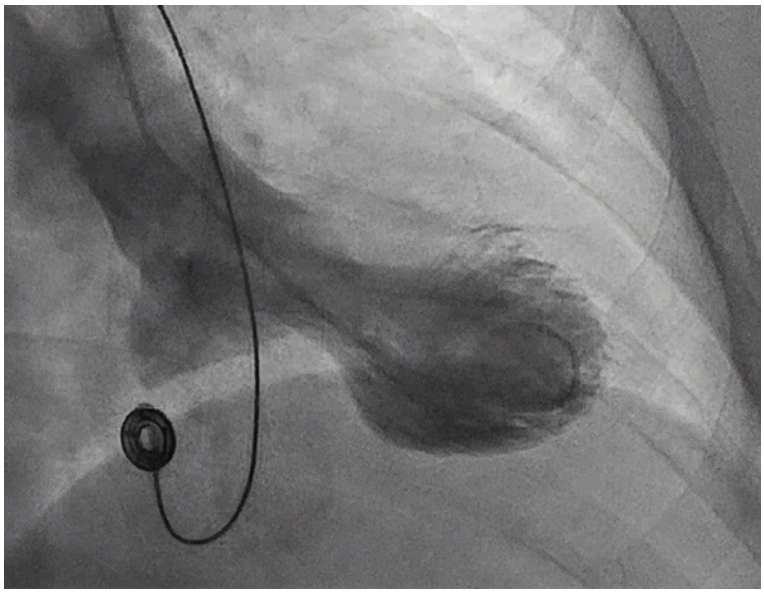

FIGURE 3. Still image from left ventriculogram demonstrating apical ballooning.

are under recognized. Both conditions disproportionately affect women and are linked to acute emotional stress.

Myocardial infarction in young, healthy women is rare. It would have been reasonable to attribute this patient's chest pain to acute anxiety. This patient's history, however, included risk factors for both SCAD and Takotsubo (Tables 1 & 2). This highlights the importance of maintaining a broad differential diagnosis and avoiding anchoring bias in the evaluation of healthy, female patients that present with chest pain.

The use of point-of-care ultrasound (POCUS) can be used to supplement the workup and evaluation among ED patients. Bedside ECHO can be used to look for pericardial effusion, apical ballooning, RV strain, or wall motion abnormalities. Apical and mid-ventricular ballooning and basal hypercontractility of Takotsubo syndrome can be seen best in the apical 4-chamber view. SCAD can cause more subtle changes including ventricular dyskinesia.

The patient arrived shortly after developing symptoms, and apical ballooning had not yet developed. In patients suspected for apical ballooning, consider performing a repeat echo while trending troponins for early presenters. The onset of takotsubo-induced myocardial dysfunction following an emotional stressor is unknown.

Have a low threshold to obtain an ECG in young patients presenting with chest pain and anxiety. Takotsubo syndrome alone can cause ST-elevation, particularly in the anterior precordial leads. Look closely for T wave inversion in lead aVR without corresponding T wave inversion in lead V1. SCAD may also result in ST segment elevation in the affected vessel distribution on EKG.

Both conditions also result in an elevated serum troponin.

TABLE 1. Risk Factors for SCAD

Young, healthy female (age 35–55)
Prior vascular dissection
Fibromuscular dysplasia
Connective tissue disorders
Hormone therapy
Multiparity
Systemic inflammatory disease

TABLE 2. Risk Factors for Takotsubo Syndrome

Post-menopausal female
Recent physical or emotional stressor
Alcohol abuse
Cardiovascular disease risk factors
Psychiatric disease, especially anxiety

Bottom Line

Both SCAD *and* Takotsubo can result in acute ST-elevation in women without atherosclerosis. A prompt ECG, troponin, bedside ECHO, and a careful medical history will help clinch the diagnosis and avoid premature anchoring bias. A cardiac catheterization is required to confirm the diagnosis. ✶

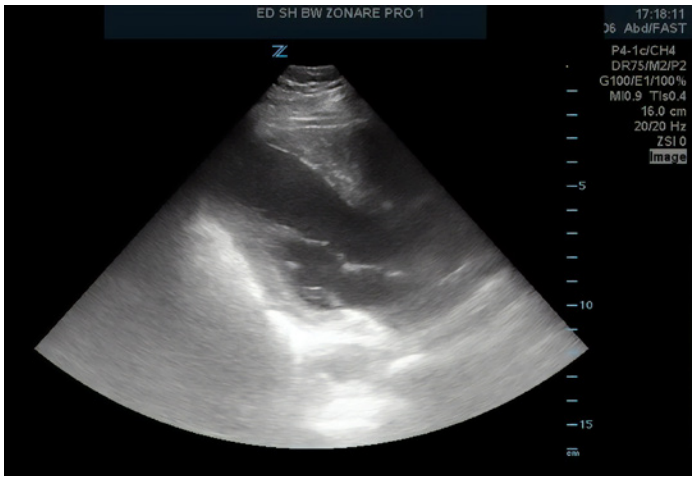

FIGURE 4. Still image from parasternal long axis point of care echocardiogram.

References available online

Jackhammer Dissection

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Case Report

A 28-year-old white male with no significant past medical history presented to the emergency department complaining of dizziness, nausea, and vomiting. He also related a headache and unsteadiness when trying to walk. His significant other noted that he had some slurred speech and right eye deviation as well. He awoke around 5:50 AM feeling normal, fell back asleep, and woke again at 7:00 AM with his presenting constellation of symptoms. He started a construction job about 1 month ago and noted a severe headache while using a jackhammer two days prior to presenting to the ED. Review of systems was otherwise negative. He denied any previous strokes or history of hyperlipidemia or clotting disorders. He had no family history of stroke. He denied alcohol or drug use but noted that he vaped for the past couple years.

On exam, the patient was afebrile, with a heart rate of 98, a blood pressure of 138/74, a respiratory rate of 18, and a pulse oximetry of 100% on room air. He weighed 150 lbs. He appeared anxious and was shaking in bed, but did not have a tremor. Cardiovascular and respiratory findings were unremarkable. He exhibited mild dysarthria, intermittent ophthalmoplegia to both the right and left eyes, and an ataxic gait. No other acute findings were present on physical exam.

Given the history and physical examination findings, a Code Stroke was activated. The patient's NIH stroke scale was 3 due to his gaze, dysarthria, and ataxia. A blood glucose level was 164 and his complete blood count and metabolic panel were unremarkable. A CT head without contrast showed a questionable hyperdensity in the distal basilar artery with no acute intracranial hemorrhage or large vessel territory ischemic changes. CT angiogram of the neck showed a luminal irregularity and narrowing of the V3 segment of the left vertebral artery with a nonocclusive thrombus at the C1-C2 level (Figure 1). These findings were concerning for a vertebral artery dissection. Additionally, CT angiogram of the head showed partial thrombosis of the distal basilar artery and an occlusive thrombus extending into the left superior cerebellar artery (Figure 2). Due to the two-day duration of the problem, the patient was not a candidate for tPA. The neuro-interventional team assessed the patient and quickly performed an emergent thrombectomy which resulted in successful recanalization of his basilar artery occlusion and TICI 3 flow.

Following his intervention, he was admitted to the neurology intensive care unit for close monitoring of his neurologic status per the unit protocol. Dual antiplatelet therapy was initiated using aspirin (325 mg) and clopidogrel (75 mg) daily. Atorvastatin (80 mg) daily was also added to his regimen. His symptoms resolved and his NIH stroke scale returned to zero at the time of discharge. He received physical and occupational therapy prior to discharge, and was discharged home with no residual deficits.

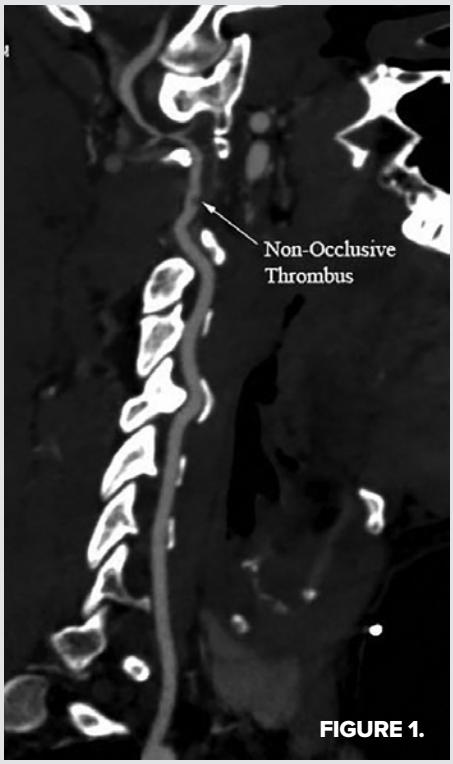

FIGURE 1.

Our standard stroke workup, including bilateral lower extremity doppler studies, hypercoagulability panel, and echocardiogram was performed in order to expose factors contributing to his stroke. The workup was unrevealing with the exception of a patent foramen ovale with left to right shift.

Discussion

The vertebrobasilar arterial system originates from the subclavian arteries. Infarction of this system results in a posterior circulation stroke. While posterior stroke presentation is not always classic, the most common clinical manifestations include vertigo, loss of balance, diplopia, headache, nausea, slurred speech, limb weakness, and altered consciousness. Dysphagia and Horner's syndrome may be seen with proximal lesions. On physical exam, patients may display limb weakness, gait ataxia, nystagmus, and unilateral limb weakness. Some causes of posterior circulation stroke include atherosclerosis, embolism and arterial dissection. Atherosclerosis accounts for 35% of posterior circulation strokes, while embolism accounts about 25% and cervical artery dissections about 15%. Given the etiologies of acute posterior circulation infarct MRI

continued on page 24 ➲

Take a HINT on Central Vertigo in the Emergency Department

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If one were to survey a group of EM physicians on a chief complaint that irks them the most, "dizziness" would probably top that list.

Although dizziness only comprises 3.5% of all ED visits per year[1] it produces anxiety because of its broad, dangerous differential. Residents are taught to dichotomize dizziness by subjectively asking patients to categorize it into "lightheadedness" or "vertiginous" categories. This question often confuses the patient and can end in a misleading diagnosis. For example, almost half the patients diagnosed with cardiac etiologies initially endorsed vertiginous symptoms.[1] Rather than ask "What do you mean by dizzy?", we should seek to delineate dizziness like any other chief complaint—focusing on timing, triggers, associated symptoms, and relevant medical history. We also need an objective exam to delineate peripheral vs. central vertigo.

Differentiating benign peripheral conditions from central nervous system lesions like strokes can be difficult, as the focal neurological deficits that can accompany central causes are essentially inconspicuous.

Enter the HINTS exam—the objective measure.

What is HINTS?

In 2009, Kattah et al. examined the diagnostic accuracy of combining 3 previously established bedside diagnostic tests:

1. Horizontal head impulse testing (**H**ead **I**mpulse)
2. Direction-changing nystagmus in eccentric gaze (**N**ystagmus)
3. Vertical skew (**T**est of **S**kew)

These tests were combined and have since been used as a tool to identify posterior circulation stroke: the Head Impulse, Nystagmus, Test of Skew (HINTS).[2] A single central finding on any of the 3 components "rules-in" a posterior circulation stroke, and further testing/treatment is indicated.

The Kattah study demonstrated the HINTS exam was more sensitive than an MRI in the first 24 hours. Interestingly, patients with a positive HINTS exam and an initial negative MRI were later found to have positive MRI findings for a stroke. Studies have shown sensitivity of the HINTS to be 96-100%, with specificity 96-98%.[3]

Jackhammer Dissection *(continued from page 23)*

is generally the most useful imaging technique; however, CT imaging is more commonly used as the initial imaging modality in patients with symptoms of stroke. As seen in our case report, CT angiogram of the head and neck frequently augment the workup to elucidate vascular abnormalities. The first-line treatment is IV recombinant tissue-plasminogen activator if the patient presents within 4.5 hours of when the patient was last known well. In order to prevent recurrent strokes, risk factors such as hypertension, hypercholesterolemia and clotting disorders must be evaluated and managed.

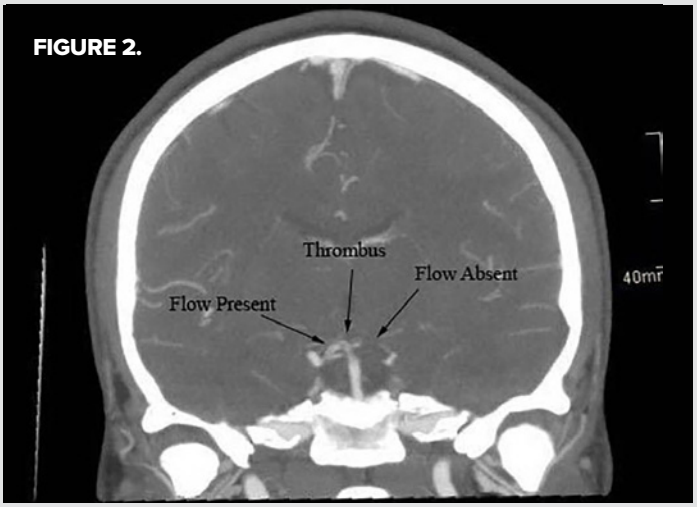

FIGURE 2.

If a patient presents with recent trauma, neck pain, headache or neck manipulation with signs or symptoms of stroke, cervical artery dissection should be considered. While use of heavy machinery seems to be a rare cause of cervical artery dissection such as in this case, we did find one other reported case of a 48-year-old male who suffered a carotid artery dissection while using a jackhammer. Cervical artery dissections may result from cervical trauma or occur spontaneously and account for the cause of about 15% of strokes in young adults. The vertebral artery segments most commonly involved in dissections are V2 and V3, which course through the transverse foramina and then exit the foramen transversarium of C2 before entering the skull.

Conclusion

One less common cause of posterior circulation strokes is cervical artery dissection. In this case report, a young male patient suffered a posterior circulation stroke due to a vertebral artery dissection. The patient had suffered a severe headache two days prior to presentation while using a jackhammer, and this was presumed to have contributed to his dissection and stroke presentation. In young patients presenting with stroke, a thorough history including occupational history and recent trauma should be done to ensure proper diagnosis and treatment. ✶

The most challenging aspect of the HINTS exam is identifying the appropriate patient. First, the patient must present with acute vestibular syndrome (AVS): vertigo, nystagmus, nausea/vomiting, head-motion intolerance, unsteady gait. Second, the patient must currently be symptomatic with nystagmus either at rest or with lateral gaze. If the patient is not currently symptomatic, it can result in false negatives. For example, the absence of corrective saccade on the head impulse test is indicative of a central cause of vertigo, but the saccade will also likely be absent in any patient not currently symptomatic.

Performing the Exam

Horizontal head impulse testing (Head Impulse)

- Hold the patient's head, allowing their mandible to rest and relax into your palms. Ask the patient to fixate on an object (ie, your nose). Then, quickly and gently move the patient's head to the left or right and then back to the neutral position again.
- Central Finding: Absence of saccade (no large beats of nystagmus as the eyes "catch up" to re-fixate on examiner's nose) is concerning.

Direction-changing nystagmus in eccentric gaze (Nystagmus)

- Assess for a presence of nystagmus.
- Central Finding: Any vertical nystagmus or horizontal nystagmus that changes direction with lateral gaze ("bidirectional nystagmus") is concerning.

Vertical skew (Test of Skew)

- Cover one eye for several seconds and then uncove g a short time period.

HINTS in the ED

The 2009 HINTS exam study was part of a 9-year data collection period on stroke in AVS at a regional stroke referral center for 25 community hospitals. It included a high-risk cohort, with inclusion criteria of AVS and at least one stroke risk factor, with most patients having ≥ 2 risk factors. While the overall rate of stroke as a cause of AVS is approximately 6%, it was > 50% in these studies.

Although the HINTS exam was originally developed and performed by neuro-ophthalmologists, its use by ED physicians has been on the rise.[4,5] When performed and interpreted correctly, the HINTS exam is a useful bedside tool for identification of a posterior stroke in patients with AVS. Thus, when examining patients with dizziness, it becomes paramount to perform an objective study such as HINTS to delineate central vs. peripheral. ✶

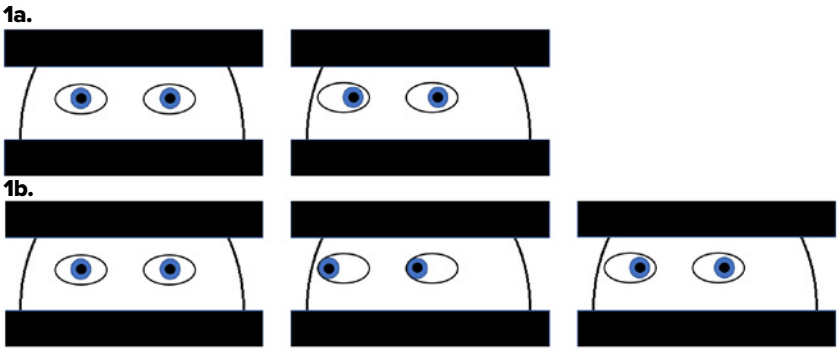

FIGURE 1. A head impulse test where the eyes remain fixed on target (1a), compared to one where the eyes lose fixation, with a corrective saccade (1b).

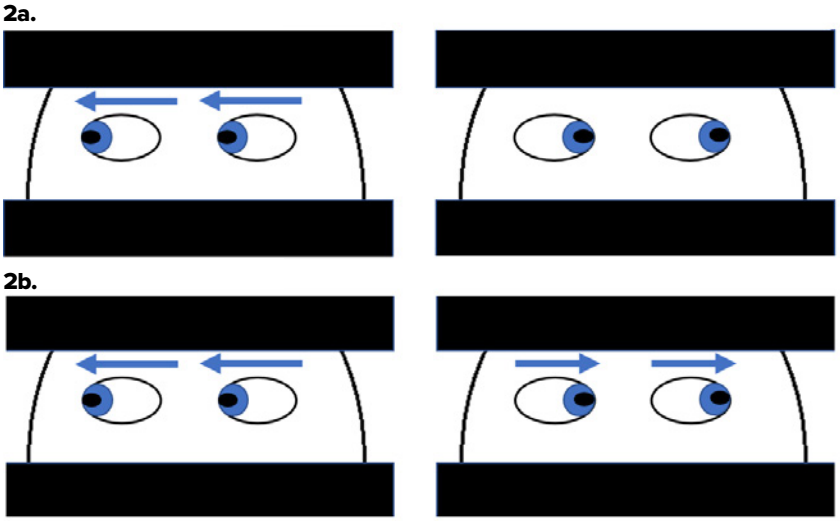

FIGURE 2. Nystagmus that occurs only when looking to the right (2a), compared to nystagmus that changes direction on lateral gaze, a.k.a. bidirectional nystagmus (2b).

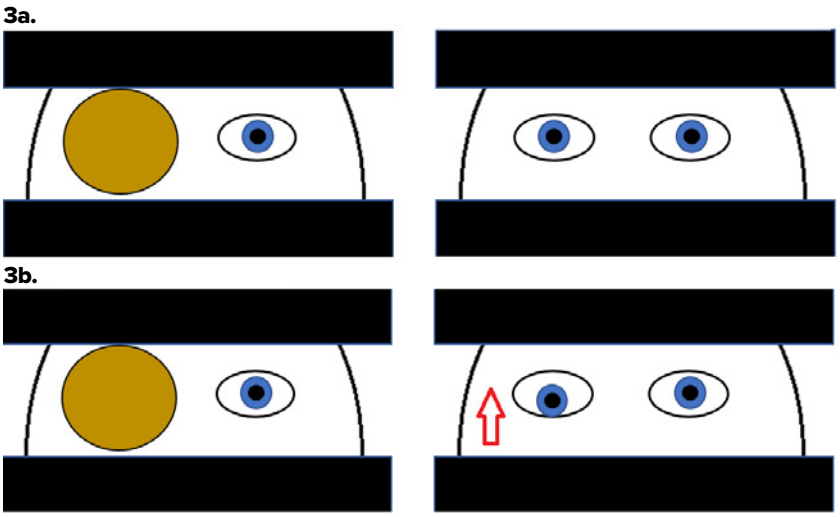

FIGURE 3. A test of skew with no verticla skew (3a) compared to one with a vertical skew that begins to correct when the eye is uncovered (3b).

	Peripheral	Central
Head Impulse	Saccade	No saccade
Nystagmus	Unidirectional	Bidirectional
Test of Skew	No skew	Vertical skew

References available online

Alcoholic Ketoacidosis

Mind the Gap, Give Patients What They Need

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Patients with alcohol use disorder commonly present to the ED critically ill due to a myriad of underlying pathologies. Alcoholic ketoacidosis (AKA) should be considered in anyone with prolonged and/or binge consumption of alcohol. AKA is a diagnosis of exclusion, and many other life-threatening alternative or concomitant diagnoses present similarly, and must be ruled out. Failure to make the diagnosis can result in severe metabolic abnormalities, acidosis, and shock.

Case

A 32-year-old male presented to the ED with 24 hours of consistent non-bloody, non-bilious emesis after a 10-day alcohol binge. He reported nausea, vomiting, abdominal pain, chills, and sweats. His past medical history included alcoholic cardiomyopathy (left ventricular ejection fraction of 20%), renal artery embolism, and upper GI bleed of unknown etiology. The patient's home medications include digoxin, sacubitril/valsartan, eplerenone, metoprolol, pantoprazole, and warfarin; however he reported medication noncompliance for 11 days prior to presentation. On exam, he was toxic appearing and in moderate distress secondary to pain. Vital signs were significant for a heart rate of 118 beats per minute and a blood pressure of 113/72. He was tremulous and had epigastric tenderness to palpation. The remainder of his physical exam was unremarkable. The differential diagnosis on initial evaluation was broad, and included pancreatitis, alcohol-induced gastritis, alcohol withdrawal, alcoholic hepatitis, sepsis, toxic alcohol ingestion, metabolic derangement (AKA, DKA, lactic acidosis, upper GI bleed), and Boerhaave Syndrome.

TABLE 1. Pertinent Laboratory Findings

Lab	Result
Na	130 mmol/L
K	5 mmol/L
Cl	83 mmol/L
CO_2	11 mmol/L
BUN	27 mg/dL
Creatinine	1.9 mg/dL
Glucose	167 mg/dL
Anion gap	36
Lactate	1.9 mmol/L
WBC	17,200 per µL (90.4% neutrophils)
Urinalysis	3+ ketonuria
INR	11

The remainder of the patient's laboratory evaluation—including liver enzymes, amylase, and lipase—were within normal limits, and methanol, ethylene glycol, salicylate, and digoxin levels were negative. Of note in the table above, the patient's INR was greater than 11, above the upper limit of the assay, and this was confirmed by repeating the test. The patient showed no signs of bleeding.

The presumptive diagnosis of AKA was made, and the patient was treated with two, 250 ml boluses of 5% dextrose in normal saline spaced out over 1.5 hours given his history of heart failure, and then 5% dextrose in half normal saline at 75 mL/hour, 100 mg intravenous thiamine, and 10 mg of oral vitamin K. He was also placed on CIWA protocol while in the ED and received 1 mg of oral lorazepam. He was admitted to the internal medicine service for continued management. On hospital day one, after continued fluid resuscitation with 5% dextrose in half-normal saline, the patient's anion gap closed, his INR decreased to 5.9, and he did not require lorazepam for treatment of alcohol withdrawal. By hospital day two, the patient's INR normalized to therapeutic range and his warfarin was restarted. On hospital day three, the patient was discharged home with outpatient services for his alcohol use disorder.

Discussion

This case highlights the importance of diagnosing patients with AKA and providing the appropriate treatment. With early diagnosis and appropriate treatment, patients improve rapidly and serious complications are prevented.

Pathophysiology

Alcoholic ketoacidosis occurs through 3 metabolic pathways:

1. Ethanol metabolism depletes NAD resulting in an elevated NADH/NAD ratio
2. Volume depletion results in increased catecholamines and cortisol levels
3. Starvation results in decreased glycogen stores

These three physiologic derangements create a state of catabolism with decreased insulin levels and increased glucagon levels resulting in inhibition of aerobic metabolism, ketone production (primarily β-hydroxybutyrate), and subsequent lipolysis.[1,3]

Diagnosis

Patients typically develop AKA after cessation of a prolonged episode of alcohol use. Clinically, patients experience nausea, vomiting, decreased PO intake, and abdominal pain but maintain a normal mental status. Lab values demonstrate a wide anion gap metabolic acidosis, with positive serum and/or urine ketones. Patient's blood glucose levels can be low, normal, or mildly elevated.[2] In AKA, high NADH/NAD causes pyruvate to be metabolized to lactate and volume depletion causes peripheral tissue hypoperfusion, resulting in a mild lactic acidosis.[3] In the setting of altered mental status or markedly elevated blood glucose levels, clinicians should consider DKA rather than AKA as the diagnosis. In both AKA and DKA, the

β-hydroxybutyrate level is expected to be in elevated, however it is expected to be more significantly elevated in AKA in comparison with DKA.[3]

Treatment

Treatment for AKA requires glucose administration, thiamine supplementation, and volume repletion. Patients should be treated with 5% dextrose in normal saline until rehydrated, and then given 5% dextrose in half normal saline for maintenance. Importantly, volume repletion alone does not correct AKA as quickly as co-administration with dextrose, as dextrose administration stimulates insulin production, which halts lipolysis, and stops ketone production.[4,5,6] One-hundred milligrams of thiamine should be given intravenously prior to dextrose administration to reduce the risk of Wernicke's Encephalopathy and also to prevent further shunting of pyruvate to lactate. If the patient's anion gap does not close with dextrose containing fluid administration, consider other causes of the patient's anion gap including toxic alcohol ingestion. If patients are severely acidotic with a pH <7.0, clinicians can consider administering sodium bicarbonate. Patients with AKA often have electrolyte abnormalities which should be corrected accordingly. Cardiac arrhythmias associated with markedly abnormal electrolytes, including hypokalemia and hypomagnesemia, are a serious complication seen in patients with AKA.[6] In patients with severe hypokalemia, it is important to replete potassium before administering any dextrose containing fluids, as this stimulates insulin production, causing an intracellular shift of potassium thus worsening their hypokalemia. It is important to recognize the difference between AKA and DKA, because patients with AKA treated with insulin develop severe hypoglycemia.

Why was this patient's INR so high?

There was initial concern for acute liver failure until the patient's hepatic function panel returned and argued against this diagnosis. Warfarin overdose was also considered, although the patient repeatedly denied this and reports he did not have access to his medications. Vitamin K deficiency was also considered. Vitamin K absorption is highly dependent on bile reabsorption, so patients with cholestatic liver disease, cirrhosis, or small bowel malabsorption are at risk for vitamin K deficiency.[7,8] Additionally, adults with prolonged fasting or excessive vomiting can have vitamin K deficiency.[9] Vitamin K deficiency can cause an elevation of INR due to poor function of vitamin K dependent coagulation factors, but will not affect PTT, platelet count, or fibrinogen level.[9] One study found that patients with chronic alcohol use had abnormal carboxylation of prothrombin, resulting in abnormal prothrombin function. Subsequent vitamin K supplementation decreased levels of abnormal prothrombin in those same patients.[10] Taken together, our patient's INR may be elevated due to a vitamin K deficiency as a result of chronic alcohol intake and poor nutritional status, causing altered prothrombin function, resulting in a supratherapeutic INR. Further, vitamin K administration in our patient resulted in normalization of his INR.

Conclusion

Signs and symptoms of AKA can often be non-specific and should be considered in patients with recent cessation of heavy alcohol use with vomiting and metabolic derangements. It can be treated promptly with fluids, dextrose, and thiamine. An elevated INR in a patient with chronic alcoholism may be due to vitamin K deficiency, which has not been previously reported. ✶

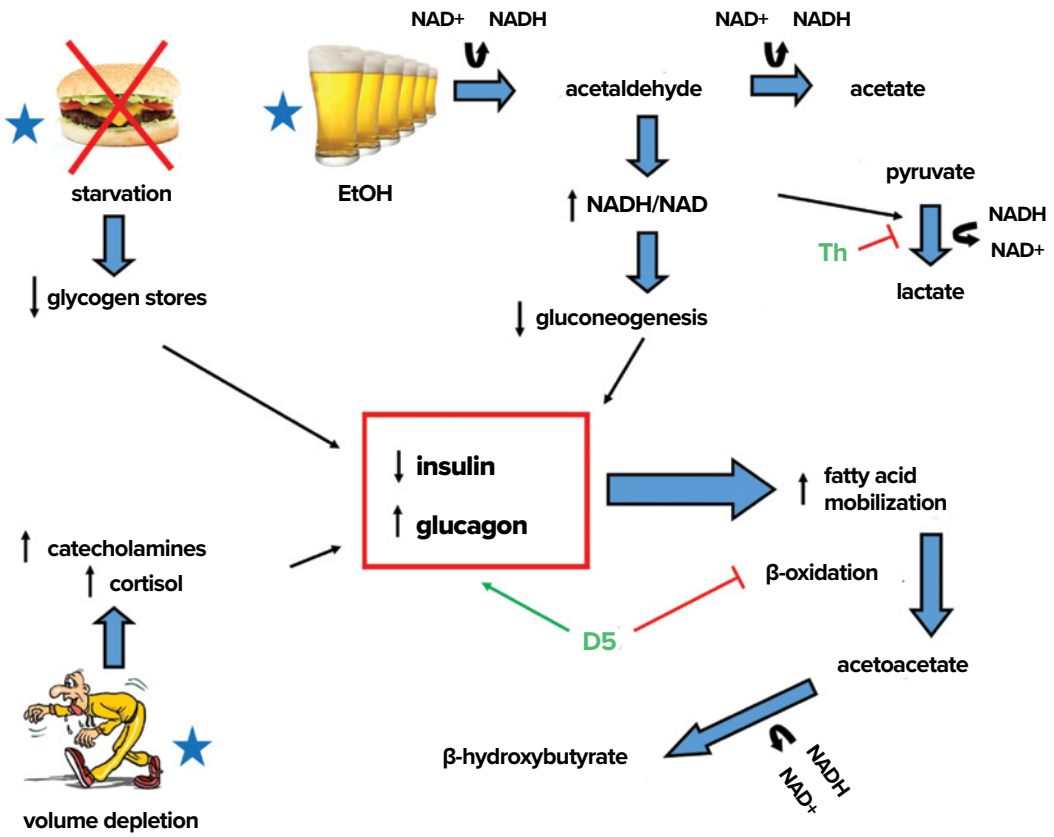
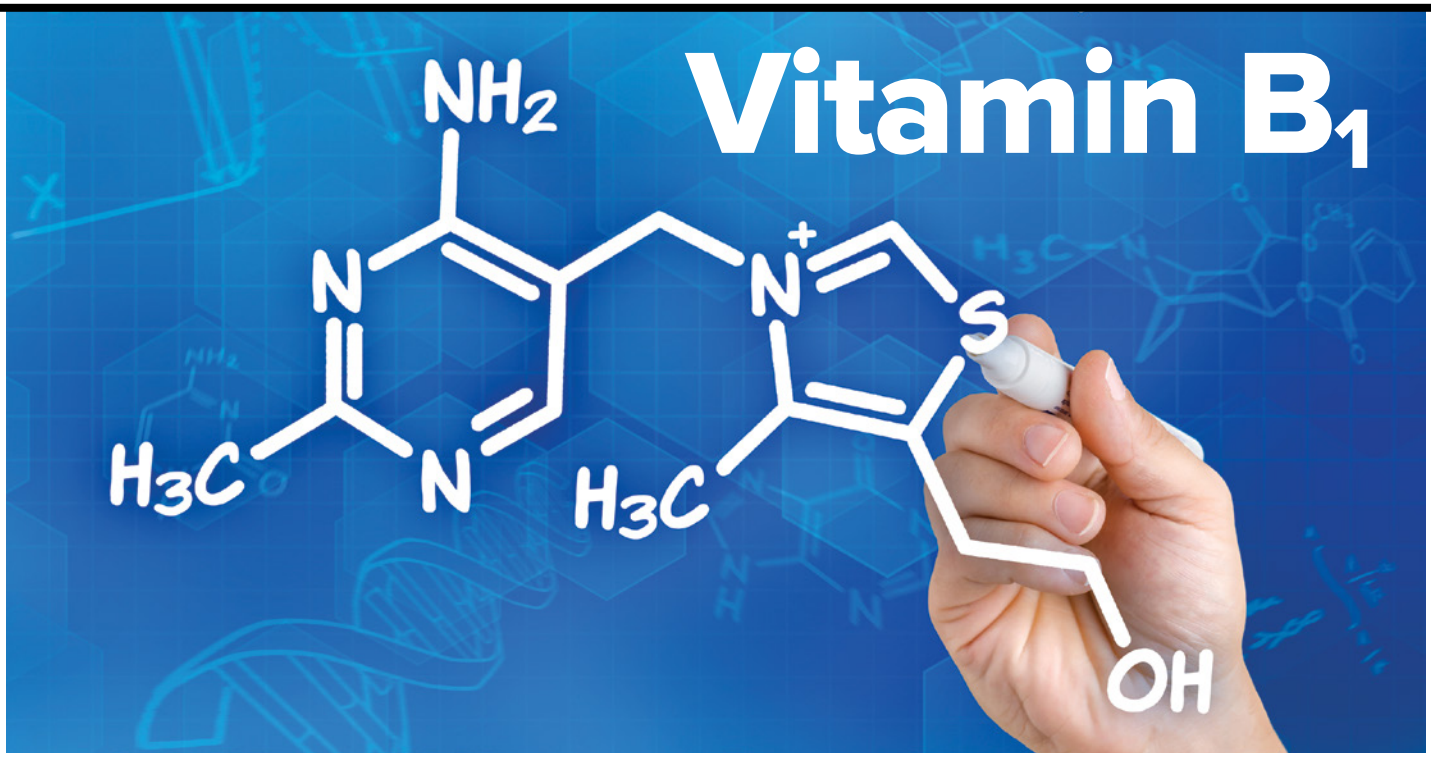


FIGURE 1. Pathophysiology of Alcoholic Ketoacidosis (adaptation of Figure 2 from Wrenn et al. The Syndrome of Alcoholic Ketoacidosis)

References available online



Detecting Wernicke-Korsakoff Syndrome

A 34-year-old female is brought to the ED with a chief complaint of "altered mental status." A neighbor called EMS after she found the patient unable to walk and noted she was "acting strangely." Chart review reveals that the patient underwent a sleeve gastrectomy seven months ago complicated by two months of post-operative hyperemesis as well as a remote history of polysubstance use disorder. She has missed most of her follow-up appointments with bariatric surgery. The patient is alert to self, but not to place, time. She answers questions in complete sentences; although, her answers are tangential and nonsensical. Physical exam reveals horizontal nystagmus, bilateral lower extremity weakness, and significant dysmetria.

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Wernicke encephalopathy (WE) is an acute neurological complication of thiamine deficiency characterized by altered mental status, oculomotor dysfunction, and ataxia. Korsakoff syndrome is a late, chronic manifestation of WE that typically presents as anterograde and retrograde amnesia with intact long-term memory.

Autopsy studies estimate a prevalence of approximately 1.3% in the general population and as high as 9% in chronic alcohol users. It is significantly underdiagnosed. Only 70% of patients with alcohol use disorder and post-mortem evidence of WE were diagnosed antemortem. 90% of WE cases in industrialized nations are associated with alcohol use disorder. After alcohol use disorder, the most common causes of WE are cancer (1.8%), gastrointestinal surgery (1.7%), hyperemesis gravidarum (1.2%), and starvation/fasting (1%).[1]

The exact incidence of Korsakoff syndrome is unclear. Untreated WE advance to Korsakoff syndrome in 56-85% of patients and leads to death in approximately 20%.[2]

Pathophysiology

Thiamine is a cofactor for pyruvate dehydrogenase and alpha-ketoglutarate dehydrogenase. It is needed for pyruvate to enter the citric acid cycle to allow for aerobic metabolism of glucose to adenosine triphosphate (ATP). Lack of

ATP production in areas of the brain susceptible to thiamine depletion leads to neuronal death. WE lesions are characterized by petechial hemorrhages and are most often found in a symmetrical distribution around the third ventricle, cerebral aqueduct, and fourth ventricle.

Thiamine deficiency is most often multifactorial. In patients with alcohol use disorder, the etiology is threefold: inadequate nutritional intake, reduced hepatic storage, and inhibition of intestinal thiamine transporters by ethanol. Genetic variations in the affinity between thiamine and thiamine transporters may contribute to variable individual susceptibility to WE.[3-6]

Korsakoff syndrome is much less frequent in cases of WE that are not associated with alcohol use, suggesting that ethanol may have a direct neurotoxic role in its development.[2,7]

Clinical Features

The classic triad of WE is encephalopathy, oculomotor dysfunction, and gait ataxia. However, only 8.2% of patients present with all three symptoms. 53% of patients present with altered mental status, 25% with cerebellar signs, 24% with ocular signs, and 23% with amnesia. The encephalopathy is characterized by disorientation and inattention. Nystagmus and ophthalmoplegia are the most common ocular findings.[1]

Other symptoms seen include vestibular dysfunction, peripheral neuropathy, hypothermia, and tachycardia. Given the nonspecific presentation, it is essential to maintain a high index of suspicion of WE in any patient with a potential source of malnutrition.

The diagnosis of WE is clinical. Normal serum thiamine levels do not rule out WE as they may not accurately represent brain thiamine levels. Imaging is not necessary to diagnosis WE but may be useful to rule out other etiologies. Mammillary body atrophy is the most specific radiological finding in patients with WE, though it is not diagnostic.[8,9]

Presence of 2 of the 4 Caine criteria can assist in making the diagnosis and have a specificity of 87%.[10]

Caine Criteria

1. Dietary deficiency
2. Oculomotor abnormalities
3. Cerebellar dysfunction
4. Altered mental status or mild memory impairment

Korsakoff syndrome typically presents as anterograde and retrograde amnesia with intact long-term memory. Some patients also display confabulation: the unconscious production of fabricated memories. Other areas of higher cognitive functioning are generally preserved; although, some patients may develop global dementia. The onset of Korsakoff syndrome usually presents as a progression of clinical WE syndrome. However, some patients may have subclinical WE, and initially present with Korsakoff symptoms only. Patients with Korsakoff syndrome seldom recover memory function.11,12

Management

Patients with signs and symptoms concerning for WE require high dose parenteral thiamine. The exact dose and duration are debated. Standard practice is to start thiamine 500 mg IV infused over 30 minutes, given 3 times daily. Patients should be admitted, as they will need at least 5 days of IV thiamine. These patients frequently have mixed vitamin deficiency. Thus, they should also be started on a B-complex multivitamin as well.

Malnourished patients should receive thiamine before, or in tandem with, glucose loading as the glucose load may induce thiamine deficiency. However, the evidence for this is limited, and glucose administration should not be delayed if needed.[13]

In WE, ocular palsies should resolve over hours to days, and confusion should resolve within days to weeks. Almost all patients exhibit some residual deficits.[14]

Almost no patients recover memory function after the onset of Korsakoff syndrome. Small case series have evaluated the use of memantine for patients with Korsakoff with some success. Although there is limited evidence to support it, the devastating effects of Korsakoff syndrome may justify its use in some cases. Patients with Korsakoff syndrome often require long-term care.[15]

Prevention

Current practice guidelines recommend that chronic alcohol users should receive parenteral thiamine supplementation to prevent the onset of WE. There is insufficient evidence to determine the optimal dose or route of prophylactic thiamine or to establish its efficacy. The risks and cost of thiamine are extremely low and are significantly outweighed by the potential benefits.[1,16,17]

Case Resolution

When you return to the patient's room after several minutes, she states that she met you last week. A non-contrast head CT shows no intracranial pathology. The patient is started on high dose thiamine and admitted to neurology for further workup and management. Her motor and cerebellar symptoms improve over the next month, however, the amnesia persists and she is eventually discharged to a long-term care facility. ✶

TAKE-HOME POINTS

- Consider WE in any patient with a potential nutritional deficiency or chronic alcohol use disorder
- If a patient has 2 or more of the following, start thiamine 500 mg IV TID and admit for further parenteral thiamine repletion.
  a. Dietary deficiency
  b. Oculomotor abnormalities
  c. Cerebellar dysfunction
  d. Amnesia or altered mental status
- Prevent, diagnose, and treat WE aggressively as Korsakoff syndrome is permanent and devastating.

References available online

Everest Trek

High-Altitude Medicine

David J. Miranda, MD
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In April 2018, during my third year of residency, I completed a wilderness medicine educational trek to Mt. Everest base camp. The trip provided a glimpse of remote medical care in the Himalayan region of Nepal, training on high altitude and wilderness ailments, and fulfilled my lifelong goal of visiting Everest (all during a residency elective!).

This 2-week, high-altitude hike alongside yaks and well-conditioned Nepalese porters provided a glimpse into how medical care is provided in remote and austere environments, ranging from arid mountain passes to snowy glaciers. At 12,600 feet, the 15-bed Khunde Hospital in the village of Khumjung is staffed by local doctors. It is equipped to provide maternal ultrasounds, EKGs, slit lamp exams, defibrillation, oxygen, X-ray, and preventative services. The hospital is remote and accessible only by foot or helicopter. The thousands of residents served by the hospital often walk for hours, sometimes days, to receive care. It's a sobering perspective on the relative abundance of resources available to EM residents in the U.S.

At 17,600 feet, base camp is situated atop a constantly moving glacier and takes about an hour to traverse end-to-end. The emergency medical tent is about a 40-minute walk from the camp's entrance, over hostile, unstable terrain. Inside, however, the medical tent feels like a safe, spacious landing. Three physicians staff the tent for the 2- or 3-month climbing season each year. When I visited, there was an emergency physician, an orthopedic surgeon, and a general practitioner.

These 3 providers spend a significant amount of their time caring for the climbing support staff who live at base camp during the season, both for their acute and chronic conditions. They are also called upon to treat climbing injuries and medical conditions that occur on the mountain, including orthopedic injuries, high-altitude pulmonary edema (HAPE), high-altitude cerebral edema (HACE), and frostbite. The tent has no advanced imaging capabilities, so much of the physicians' decision-making relies heavily on their history and physical exam.

Some climbers have additional options for medical care on Everest. For example, climbing teams might employ their own doctors to help care for members. As helicopter technology improves, it is possible for climbers injured on Everest to be evacuated from higher altitudes directly to larger hospitals, rather than descending to base camp for preliminary treatment.

Around 14,000 feet, during our descent from Everest base camp, we stopped at the Himalayan Rescue Association's clinic in Pheriche. The 2a-room clinic is staffed by local and

volunteer doctors who assist both the local Nepalese population and trekkers. It is open during the main trekking seasons and, like the other clinics in the region, lacks advanced imaging capabilities. During my visit, however, the clinic had use of a portable ultrasound brought by one of its Wilderness Medicine Fellows. The clinic's 4 beds are used to treat patients suffering from conditions including acute mountain sickness, diarrheal illness, hypertension, diabetes, and more severe conditions such as HAPE and HACE.

The clinic's patients can also be evacuated by helicopter to Kathmandu, Nepal's capital, if weather permits and if medically warranted. Although the clinic's resources are limited, it provides essential care in the region. For example, the clinic helps coordinate rescue and evacuation during devastating natural disasters, including during the 2015 earthquake that killed thousands of Nepalese people and triggered an avalanche that killed 18 people on Everest base camp.

In addition to visiting medical facilities in the region, the trek's program included near-daily lectures on various aspects of wilderness medicine by two experts who happened to be married. The lectures covered topics ranging from acute mountain sickness spectrum, exposure and frostbite, diarrhea, water purification, bacterial/parasitic infections, and weather and animal related injuries, to name a few. We also had hands-on demonstrations using a portable hyperbaric chamber called the Gamow bag, portable oxygen, and makeshift patient transport, as well as lessons on designing an appropriate first aid kit.

Another enriching aspect of this wilderness medicine trip was getting to spend time with a diverse group of health care professionals from across the U.S. Doctors of all types attended the trek (emergency medicine, cardiothoracic surgery, OB/GYN, radiology, ophthalmology, family medicine, neonatology), nurses, physical therapists, and search and rescue EMS crew members. After spending nearly 2 weeks on the trail together, our group of strangers developed strong friendships and formed an extended professional network that I am lucky to have on call.

For emergency physicians, an inherent degree of resourcefulness is ingrained during training, a skill that makes this specialty particularly capable of providing care in resource-poor environments. Visiting these remote Himalayan villages and facilities gave me a new appreciation for how valuable my emergency medicine training has been and how it can open the door to opportunities I hadn't previously imagined. I encourage everyone to also take a step out of their comfort zone and seek their own adventure. ✶

Climate Change is Killing Patients.

Let's Not Make it Worse.

Laura Haselden, MD
University of Texas at Austin Dell Medical School

Give us your tired, your poor, your huddled masses yearning to breathe free…

These are the words inscribed on the Statue of Liberty, penned by Emma Lazarus in 1883, yes—but are they also the emblem of the emergency physician?

In our careers, we care for the tired, the poor. The homeless, the mass casualties. The asthmatics and COPD flares. The indigenous, the wealthy, the healthy and unhealthy, the traumas and the sick.

In our careers, in caring for these groups, we advocate for preventative medicine, for safety belts and helmets and vaccines. We recognize the dangers of driving drunk, of injection drugs, of untreated HIV. We counsel on smoking cessation and blood pressure control. We do these things in the name of making our future jobs easier—of preventing health catastrophes. While we love our work, we would all love never to see another victim of a drunk driver, or another CHF exacerbation suffocating, or another hemorrhagic stroke. We advocate for preventative medicine because it's better for our patients and our communities.

Our Climate is Changing, Our Patients Are Suffering

The Fourth National Climate Assessment (NCA4), released last November, looked comprehensively at the current and projected state of climate change in the world, particularly in the US, and its effects on human and population health. It found what we should already suspect—those communities most at risk are precisely those communities we, as emergency physicians, are tasked with caring for.

The very young and old are more severely affected by changing temperatures, particularly by increased heat related illnesses. Those with cardiopulmonary illnesses are disproportionately affected by draughts and decreasing air quality. Low income and coastal communities are devastated by flooding and food insecurity. We are all affected by decreasing phytonutrients and increasing vector-borne illness, contaminated food and water, increased mental health burdens of extreme weather events.[4]

We're Making it Worse

The NCA4 also found what we, as physicians, might not expect—how drastically the healthcare industry contributes to climate change. On a national level, healthcare contributes about 10% of US greenhouse gas emissions each year.[4] Hospitals generate thousands of tons of landfill waste a day. On every level, we, our employers, and our industry take thousands of actions a day that cumulatively create an incredible impact on the health of our communities and our world. Think of the landfill waste you generate in a day of caring for patients—the gowns and gloves, disposable lac trays, the IV bags and tubing. Think of the energy required to clean and power an OR for even the most routine case. Our careers, our

livelihoods, have environmental impacts beyond even what we as individuals have in our daily lives.

What Do We Do?

We, as doctors, know prevention is the best medicine. We know a changing global climate is destroying the health of already vulnerable populations. We know our careers, our industry, contributes to global climate change in a substantial way. Why, then, are we, the defenders of the defenseless, the last and only line of healthcare for so many of our fellow men and women, not more alarmed?

We Can Make it Better

We, as emergency medicine residents, have a unique position to create change in our facilities and communities. Let's use that position to shift the delivery of healthcare to a more sustainable model. By reducing our industry's environmental impact, we can help protect our patients' health, our community's health, and our world's health.

In a greening world, our industry is beginning to see that sustainability is key. Our hospitals are starting to go green as administrators realize not just the ethical and environmental advantages, but the financial advantages, of creating energy-efficient infrastructure.

But from our vantage point in the trenches, we can do even more.

How Can We Help?

We can advocate for reusable supplies—the reusable, sterilizable surgical instruments for your lac repair are not only a higher quality, but more sustainable, and potentially even more cost effective.

We can advocate for recycling in our facilities—while reducing our single use plastics will always be the most effective way of reducing landfill waste, a great deal of what we use in the emergency department is recyclable — notably, empty IV bags and tubing that are not contaminated with bodily fluids, pill bottles and containers, and plastic containers for tools and equipment are easily recyclable at almost any facility.

While you are advocating for recycling capacity at your facility—educate! Recycling is only effective when done correctly—avoid food, trash, and bodily fluid contamination.

The possibilities for improvement are endless, and beyond the scope of this brief letter—you need only to look. The most important thing, to start, is to recognize the problem.

Our careers save lives, every day. And our careers are damaging the earth. We must recognize our ability and our responsibility to maximize our impacts, change our careers and our practices, to best benefit our communities and the world.

Get More Involved!

This article brought to you on behalf of the EMRA Social EM Committee. Want to learn more about SEM? You can connect with others interested in SEM topics by joining the Social EM Committee Basecamp on the EMRA website. ✶

References available online

A CHANCE TO PROVE OUR VALUE

The Acute Unscheduled Care Model

Editor's Note: This article, written by the 2019 recipients of EMRA's EDPMA travel scholarship, is being simultaneously shared with EDPMA.

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"I'm sorry we didn't find the reason for your abdominal pain today, but your work-up in the ED did not reveal any acute life-threatening illness. I'm glad you feel better; please follow up with your primary care doctor or return for any worsening symptoms."

Raise your hand if that sounds familiar. Go ahead—we'll wait. Now, what if we told you that only 17% of those patients are able to follow up with their PCP within 7 days, and it takes 30 days for 83% of patients to get into their PCP's office? That doesn't even include the nearly 20%, according to some government surveys, of Americans who don't have a PCP to begin with. What do you do with that information? More important, who is responsible for the care of these patients after they leave the ED? Well, if the ACEP proposal to CMS is approved, it just might be you.

The proposal, entitled *Acute Unscheduled Care Model (AUCM): Enhancing Appropriate Admissions* is meant to allow emergency physicians to participate in Alternative Payment Models (APMs) by accepting financial risk directly attributable to their discharge disposition decisions. While that may sound unnerving, it may actually be a blessing in disguise. Current value-based (aka cost-saving) payment models attempt to remove the ED physician from patient care by curbing visits to the ED that insurance providers, cough-cough, deem "unnecessary." This is done via coordinated outpatient care, which sounds great, but the outpatient setting is cracking and patients are continuously falling through these cracks. Pair that with the hot topic of balanced billing, and it seems that lately emergency physicians are being scapegoated for the rising costs of health care in America. This is where the AUCM (or "awesome"") proposal comes into play, attempting to place emergency physicians back in the driver's seat and providing us with the chance to prove our value within the health care delivery system.

At its core, the proposal is elegantly simple. It focuses on rewarding emergency medicine providers while simultaneously decreasing cost of health care delivery in 3 ways.

- First, it aims to reduce hospital inpatient admissions and observation stays by providing a safer discharge option.
- Second, it empowers providers to coordinate, manage and avoid unnecessary post-discharge services on a case-by-case basis.
- Third, it attempts to avoid post-ED visit patient adverse events (repeat ED visits, inpatient events, observation stays and death) and their inherent costs.

By doing so, it increases the "value" of care provided by ED physicians. Essentially, the AUCM proposal makes ED providers and their facility responsible (for lack of a better word) for a discharged patient 30 days from their initial encounter.

We know what you're thinking… sounds good in theory, but HOW?

Keeping in mind that some details may change, the current proposal focuses on 4 chief complaints most ED physicians can approach in their sleep: chest pain, abdominal pain, syncope and altered mental status. Any patient checking into the ED with one of these 4 chief complaints will be eligible to enroll in the AUCM payment model upfront. The final diagnosis in theory does not matter, although if your chest pain ends up being a STEMI, they would be pulled from AUCM. In fact, no patients admitted to inpatient or observation status would qualify under the current proposal. But the nearly 60% of chest pain patients discharged with a diagnosis of anxiety, costochondritis, GERD, or — our personal favorite — "non-cardiac chest pain"(thanks for being specific, doc), would fall into the model.

What does that actually mean?

Well, it means that instead of sending your patient out into the abyss, you would now have a tool set at your disposal to bubble wrap patients until they are back to their baseline. These tools, as proposed, include next-day telemedicine appointments, home visits by nurse practitioners/RNs, and other forms of transitional care; as well as care coordinators in the ED to help facilitate everything. Now this clearly comes with an upfront financial investment. However, when this new cost is compared to the hospital's

current benchmarks for the same episodes of care, the upside is projected through the reduction of admission, readmission and/or complication costs. The bottom line is if emergency physicians appropriately reduce unnecessary admissions, there will be a cost-savings, which by voluntarily participating in this awesome AUCM program, will be shared with ED providers.

Now, you may be reading this and saying, "but wait, I went into emergency medicine so I wouldn't have to provide longitudinal care, and now you're saying I may have to?" Recall that your purpose as a professional is to provide **quality** care. The proposed model does not, by any means, require you to establish lifelong relationships with your patients. Neither does it mandate that you adjust a patient's insulin regimen 1 unit at a time until their HbA1c is just right (we got palpitations just writing that). What it does require, however, is for you to step up, be accountable, and ensure that patients do not fall through the cracks when they leave the walls of the department. The beautiful thing about emergency physicians is that we provide care to whoever, whenever, and wherever; not to whoever we prefer, whenever we see fit, and wherever it's convenient. Traditionally, that care has been in the ED where we've served as a safety net for all, but now the safety net needs to expand beyond the ED. While that may seem like a tremendous burden, it actually provides our profession the opportunity to vastly increase our sphere of influence.

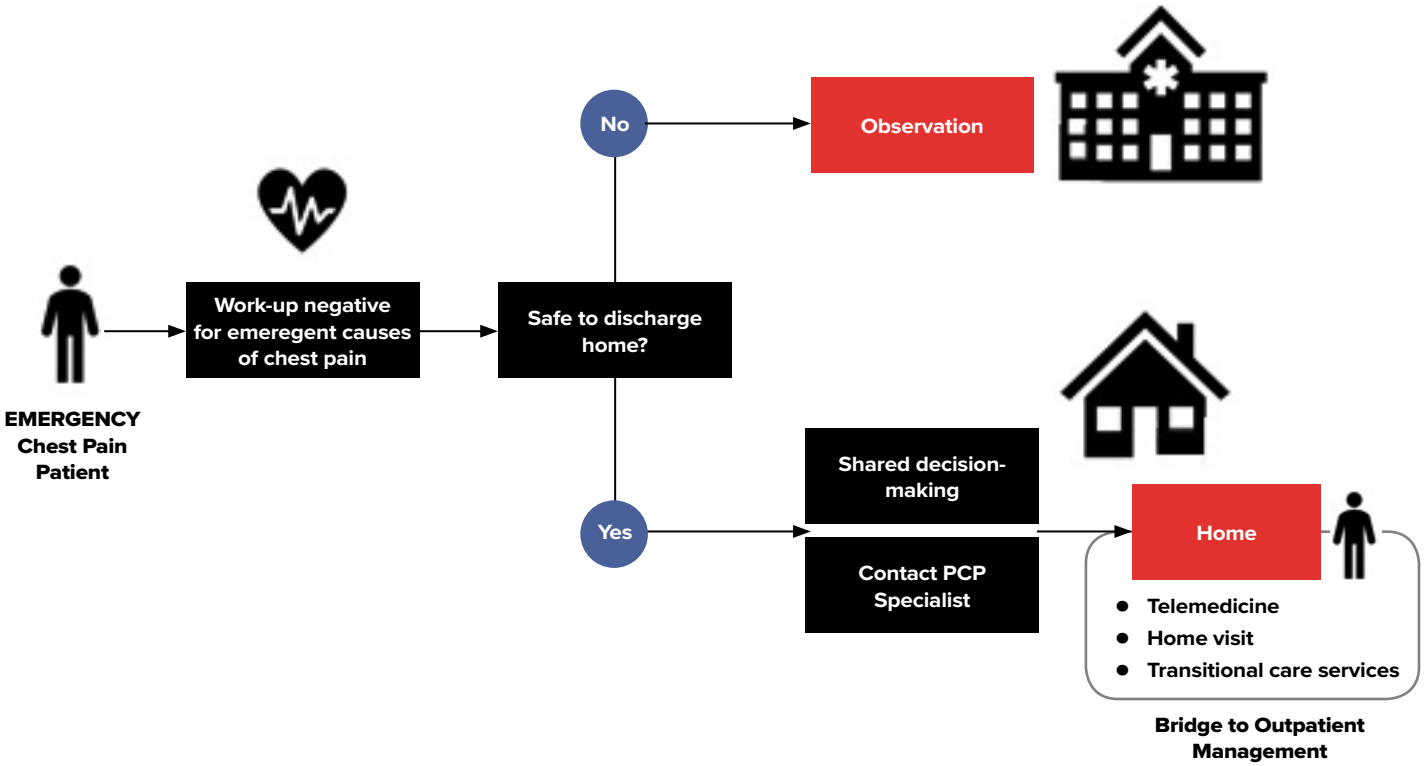
We pride ourselves on being go-getters, community leaders, problem-solvers, and innovators, and the AUCM proposal offers us a chance to once again prove our value. Sounds, well...awesome, doesn't it?

About the Authors

Dr. Polavarapu and Dr. Bolduc are the current EMRA EDPMA scholars. The EDPMA (Emergency Department Practice Management Association) is a trade association for emergency physician groups and their practice partners, such as billing and coding companies. EDPMA's members deliver (or directly support) health care for about half of the 146 million patients that visit U.S. emergency departments each year. EDPMA advocates and educates on reimbursement that can sustain the delivery of high-quality, cost-effective care in the ED to an underserved patient population who often has nowhere else to turn.

To learn more about the organization, visit their website at www.EDPMA.org. ✶

The proposal, entitled *Acute Unscheduled Care Model (AUCM): Enhancing Appropriate Admissions* is meant to allow emergency physicians to participate in Alternative Payment Models (APMs) by accepting financial risk directly attributable to their discharge disposition decisions.



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Should I Stay or Should I Go?

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The job hunt approaches. You are probably receiving advice from all over: Practice at a community site. Practice at an academic center. Go far away and create your own identity and practice patterns. Stay safe your first year out where plenty of consultants are available. Go where there aren't any consultants to solidify your procedural knowledge and learn when to transfer.

But what will be best for you? It becomes a very personal decision, and one that only you can ultimately make.

When it comes to staying put, it seems like an easy decision for many. You have had 3 or 4 years to develop relationships with the ED staff, consultants, the patient population, and your electronic medical record. You are comfortable. You are confident. Remember how stressful it was to start each new rotation as an MS3? Leaving the comfort of 3 or 4 years would exacerbate that feeling a hundredfold! It would be ideal to continue on a path you started a few years ago. Your significant other may have a job they love, your family may be settled—all the perks of never having to move again!

While it seems like an easy decision, there is more to consider. While you are comfortable in your role right now, the nurses, consultants, and the junior residents behind you are also comfortable with you in that position. They may not immediately see you as the attending, which can be stressful. Leaving may be a time to help you create an identity as an attending. Those in education leadership can tell many stories about the middle of the pack residents from their programs who have left and been some of the most successful docs at their new institutions. They can also tell stories about their star residents who go out and hate their new roles and come back.

Beyond comfort, staying at your home institution may limit your exposure to practice variation and diverse patient populations—many new grads will have spent the majority of their training explicitly at an academic center, while the majority of jobs are in the community. Were you at a tertiary care center that was on the receiving end of so many transfers? It may be a great opportunity to be on the shipping end to see how things are done with less resources.

Full disclosure. I am someone who stayed on where I trained with and have zero regrets. Only after 4+ years post residency have I moved on to a new institution. I attribute much of my success in my first year out to my former chair. He thought about what I would need as a new attending before he agreed to hire me. I was required to work the majority of my shifts away from residents at our 2 community sites where we had had little exposure in residency. I was taken out of my comfort zone the majority of the first year out to learn new systems, establish new relationships, and develop confidence in my practice patterns. I was also not allowed to take on "side jobs" (hospital committees, residency lectures, etc.) until I had taken my written boards. At the time I hated it, but looking back I can see that the shifts I worked with my former co-residents in the place where I had felt most comfortable were some of the hardest and darkest days of my first year out. My teaching evaluations said that I was "mean, angry all the time, not the same chief resident I remember, etc." and looking back it was likely all related to the stressors I felt at being the ultimate decision-maker for so many complex and critically ill patients at a very busy trauma center.

So what do I recommend? I recommend doing what will make you happy!

When in doubt, remember most EM contracts are only for a year, and many of your future colleagues out there practicing have had more than one job out of residency! I conducted a very academic Twitter poll while working on this, and the majority of folks were happy and would not have changed their first job out of residency in both sides—those who stayed and those who left. So, be happy at home or take the leap and find happiness elsewhere! Either way, be kind to yourself in your first few weeks and months. Becoming an attending came with more emotional responsibility than I was expecting. Do not be afraid to ask for help. You will never truly be on your own in the practice of emergency medicine. All of your co-residents, those who trained you, and those who are your senior colleagues at your new institution will be there to help.

No matter where you end up—just remember: the medicine will be your constant! If you ever feel lost, just remember that your home residency institution will always be that, home. And you can always go home. ✶

TAKE-HOME POINTS

- Do what makes you happy!
- There are perks and pitfalls to staying or leaving.
- The medicine will be your constant.
- You can always come home.

The Next Generation Medical Student

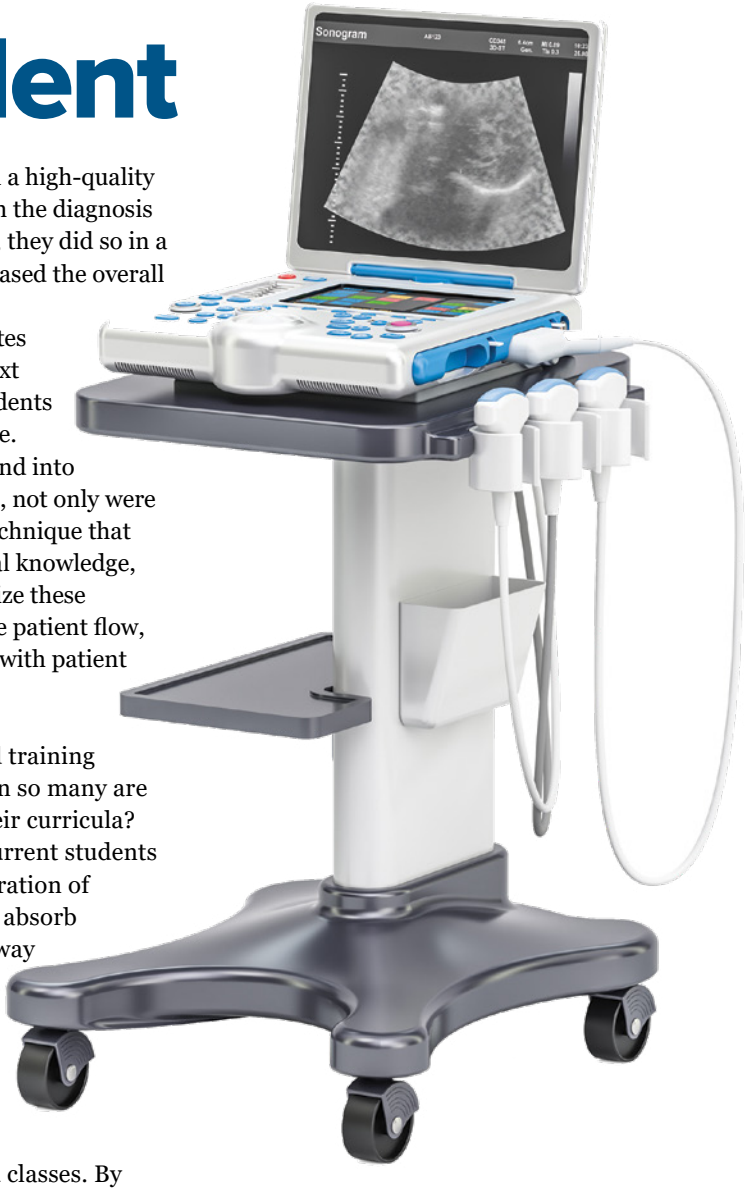
Jonathan Brewer, MD
UT Southwestern Emergency Medicine Residency
@jbrew452

Medical education has been evolving since the days of Sir William Osler, a founder of Johns Hopkins Hospital and initiator of the residency training concept in medicine. While the overall goal of developing quality physicians has stayed the same since then, medical students have become increasingly focused on board exam scores and clinical evaluations rather than learning the "practice of medicine." At the same time, burnout among students has increased substantially while clinical hours have decreased. This phenomenon is also seen among surgical residents, for whom a decrease in working hours does not seem to lower levels of burnout.[1] What is the reason for this? I believe that the answer is due to a lack of purpose felt by medical students. This is something we need to change. The question is: **How do we incorporate the modern medical student as an essential member of the treatment team in an era of increased liability while bolstering that student's education?**

One answer can be found in the field of ultrasound. A landmark article on student-performed point-of-care ultrasound (SP-POCUS) published in 2017 found that students who had received integrated ultrasound training during a single year of their medical school curriculum altered attending physician management of a patient in the emergency department in 17.3% of scans performed.[2] The prospective observational study by Daniel Udrea and colleagues at Loma Linda University School of Medicine also found an average of 94.7% physician agreement with the SP-POCUS diagnosis. In addition, attending physicians avoided ordering additional imaging for more than half of the SP-POCUS scans performed. So not only did students perform a high-quality ultrasound that assisted in the diagnosis and treatment of patients, they did so in a way that potentially decreased the overall cost of care.

This study demonstrates the profound effect the next generation of medical students can have upon patient care. By incorporating ultrasound into their first year curriculum, not only were students able to learn a technique that augments their anatomical knowledge, they were also able to utilize these skills in the ED to increase patient flow, decrease costs, and assist with patient management.

So how do we justify implementing ultrasound training into medical schools when so many are already "accelerating" their curricula? The answer lies in how current students learn best. The next generation of medical students will not absorb information in the same way as prior generations; the modern medical student tends to rely on technology and learns well through tactile and interactive experiences, rather than lecture-based classes. By teaching ultrasound in a module-based format that can be spread over the course of a year and offering clinical experiences that will carry forward if the student chooses, medical schools will encourage student participation. While the didactics of medical school are extremely important, providing ample and early clinical experience has shown immense benefits.[3] By giving students the opportunity to practice medicine as first-year students, we allow them to discover increased motivation for their studies while combating the monotonous grind that can characterize the pre-clinical years.



POCUS has been growing since the 1970s due to the advent of newer ultrasound modalities and technologies. It is now a staple of many medical specialties but it has yet to make a significant impact at the student level. I firmly believe that by introducing this skill into first-year curricula across the nation, we can revolutionize medical student education. This will allow students to play more important roles in the treatment team and will give them greater purpose in medical school. It will also allow them to combat personal burnout while changing the landscape, and cost, of care. ✶

References available online

Finding My Voice

Katherine Dowdell, MD
Baylor College of Medicine

As emergency physicians we are extensively trained in the art of decision-making. I was once naïve enough to believe that the only stakeholders involved in medical decision-making were patients and their treating physicians. However, I can no longer ignore the power of external influences. Behind each decision is a complex web of resources, funding, cultural beliefs, social determinants, and policy that often pulls my patients in directions I do not feel is best for their health. Determined to understand these extensive barriers, and the numerous ways in which they impact care.

The Texas College of Emergency Physicians hosts a Leadership and Advocacy Fellowship to introduce young physicians to advocacy efforts. As part of the fellowship I attended numerous state and national meetings, including ACEP LAC in Washington, D.C. Just as in the ED, my educational experience at LAC unfolded as I was tossed in to the action — preparing to discuss one of the specific issues highlighted at the meeting: securing appropriate access to mental health services in emergency departments. I am convinced that every emergency physician has experienced the frustration of a patient presenting with a mental health emergency, only for that patient to be stuck in a noisy, cramped ED for hours while a disposition is pending. Work on this crucial issue has already begun. In fact, a bill by Rep. Raul Ruiz, MD, of California titled "Improving Mental Health Access from the Emergency Department Act of 2019" was introduced to the House in May. This example is one of many that illustrates the impact of policy on not only the emergency care of patients, but also on the educational experience of EM residents.

There were many excellent lectures at LAC, but I was particularly taken by a talk from Bradford Fitch, president and CEO of the Congressional Management Foundation. He explained how to successfully initiate and cultivate relationships with your elected representative. **As a budding advocate I would liken this skill to calling consultants in the ED**—know the facts, be concise, be flexible, and always be polite!

1. **Make contact**: While hundreds of emergency physicians arrive at Capitol Hill each year, it may not always be feasible to travel to D.C. Each representative has a local office and may hold town hall meetings as well. Explore other forms of communication, including phone calls, letters, and email.
2. **Prepare:** The biggest aspect of preparation is knowing your audience. Your representative will want to know how the issue affects their constituents, as well as any economic impact. Are many of your patients in this representative's district?
3. **Refine your expectations:** Your representatives are quite busy. It is possible that you will be meeting with their staff. They are knowledgeable about current legislation, viewpoints, and needs within their districts.
4. **Put your best foot forward:** Show your passion for your patients, but try not to come across as demanding or accusatory. As a doctor, you have an incredibly unique and privileged perspective of society. Channel your emotions and actively recruit your legislators as allies.
5. **Follow up:** Because numerous bills are introduced each session, advocating for a cause you believe in is rarely a singular effort! Choose a form of communication you are comfortable with and remind your representative why action should be taken. It can be particularly crucial to do this in a timely manner if a bill has already been introduced.

Traveling to D.C. for LAC and participating in meaningful conversations with congressional staff has opened my eyes to a whole new way to care for my patients. But what's next? How do I keep the momentum going?

Get involved in state government. Beyond being more accessible geographically, it may be more feasible to make headway at a state level and focus on issues that are uniquely important to your area. Many issues critical to us as emergency physicians are primarily dealt with on a state level including Medicaid programs, insurance regulations and regulations involving opioid use and abuse. Sharing another example from my home state, the Texas Medical Association holds monthly "First Tuesdays" in Austin when the Texas Legislature is in session. Doctors come together to advocate for optimal patient care and appropriate work conditions. Consider searching for similar programs in your own state!

Regardless of where you are in your medical training or your level of experience with advocacy, know that you have a unique insight to some of the most fundamental issues in your community. **I challenge you to apply the skills you have learned in the ED to a new arena and be a megaphone for your patients and colleagues.** If this article has piqued your interest, be sure to check out EMRA's *Advocacy Handbook* for a deeper dive! Hope to see you at LAC 2020! ✶

Shout-out to ACEP Board of Directors member Alison Haddock, MD, FACEP, for editorial assistance.


Monday, Oct. 28
10 pm - 2 am
Club Vinyl
EMRA
PARTY
Envision
PHYSICIAN SERVICES
Visit Envision Physician Services' Booth #811
First 500 residents receive a wristband for a free drink at the party.
acep
Scientific Assembly
DENVER 19

Plan Now for EMRA Events at ACEP19

EMRA stays busy during ACEP Scientific Assembly — join us! Get up-to-date event information at **emra.org/acep#emraschedule**.

Oct. 24: EMRA Resident Councilor Mixer
Oct. 25: Medical Student Meet-Up
Oct. 26: Fall Medical Student Forum and Residency Program Fair
Oct. 27: EMRA Resolution Review and Public Hearing
Oct. 27: EMRA Committee Workshops and Programs
Oct. 27: Case-Con Poster Competition
Oct. 27: EMRA Job & Fellowship Fair
Oct. 28: Fall Representative Council Meeting & Elections
Oct. 28: EMRA Resident SIMWars
Oct. 28: EMRA Party, sponsored by Envision Physician Services
Oct. 29: 20 in 6 Resident Lecture Competition, sponsored by Hippo Education
Oct. 29: EMRA Airway Stories, sponsored by Vituity
Oct. 30: EMRA MedWAR, sponsored by BTG (Volunteers needed! Visit emra.org/medwar.)

These events are free for EMRA members and do not require ACEP19 registration (but we do encourage it — using promo code EXPLORE). Start building your network by attending these events, volunteering, and competing. ✶

EMRA Committees to Host Expert Panels, Skills Labs at ACEP19

Get ready for a whole new approach to networking with your committee peers! EMRA's 18 committees have reimagined the traditional business meeting/planning session.

Join EMRA on Sunday, Oct. 27, at the Embassy Suites in Denver to participate in the skills workshops, expert panels, and competitions that will help you gain knowledge and form valuable connections.

- ✓ **TEE Lecture & Lab**, presented by #TheResusTEEWorkshop team and hosted by the Critical Care Committee (registration required; hands-on slots will be awarded via lottery).
- ✓ **Combined PEM-Ultrasound-Simulation Workshop** featuring multiple hands-on skills stations.
- ✓ **Case-Con Competition** hosted by the Research Committee
- ✓ Plus, benefit from EMRA committees making connections with ACEP Sections to offer career insight in areas such as Government Services, sports medicine, international medicine, and more.

Updated details will be posted to **emra.org/acep#emraschedule** as available. ✶

Elect the EMRA Board of Directors

In addition to resolutions, the RepCo elects members of the EMRA Board of Directors. We will fill the following vacancies in October, including:

1. President-elect
2. Secretary/EM Resident editor
3. Vice Speaker of the Council
4. Director of Education
5. Director of Technology

If you want to run for the EMRA Board, **submit your nomination** by Sept. 13. Then attend the Fall Representative Council meeting on Oct. 28 to make your bid for the position you want. Members will browse your candidacy packet online ahead of the vote.

Get details and submission forms at emra.org; search "*Become a Board Member*." ✶

EMRA RepCo Resolutions Due Sept. 13

Do you think we need to take a stand on an issue? EMRA members can submit resolutions on any topic pertaining to EM residency or the practice of emergency medicine. For **step-by-step instructions and a sample resolution form**, search "Representative Council" at emra.org. **Resolutions are due Sept. 13.**

SPECIAL NOTE: We want every program to vote! Get your program representative ready for the meeting on Oct. 28. (Vote in-person or online.) ✶

Apply for Writing and Visual Arts Awards

The ACEP Section of Medical Humanities is soliciting submissions for awards, and **nominations are due Sept. 4**.

- **13th Annual Writing Award:** Eligible pieces are creative works published in print or online between November 2018 and August 2019. Word limit is 2500. Blog entries will only be eligible if reconfigured and submitted as an independently publishable piece of creative writing. Nominate yourself or others. Poetry and prose are separate categories. Limit 2 pieces per person.
- **7th Annual Visual Arts Award:** Show off paintings, photography, etc. Submit a digital image or file of the visual art (photograph, sculpture, textile, pottery, painting, etc). Limit 2 pieces per person.

Nominees must be ACEP members (all EMRA members are ACEP members). Email submissions to Tracy Napper (tnapper@acep.org) before **Sept. 4**. The winning pieces will be announced at ACEP19 in Denver. ✶

Servant Leaders in Our Communities

During September, EMRA will recognize **EM Day of Service** activities. We encourage all EM colleagues to undertake community-driven initiatives that uplift, support, and answer the needs of neighborhoods and fellow citizens. From helping with park cleanup to food banks to nursing home visits – or whatever volunteer opportunity fits your program — get out there and improve your own wellness by helping others. Make good happen! Show us what you're doing by using #EMDayofService on social media channels! ✶

ECG Challenge

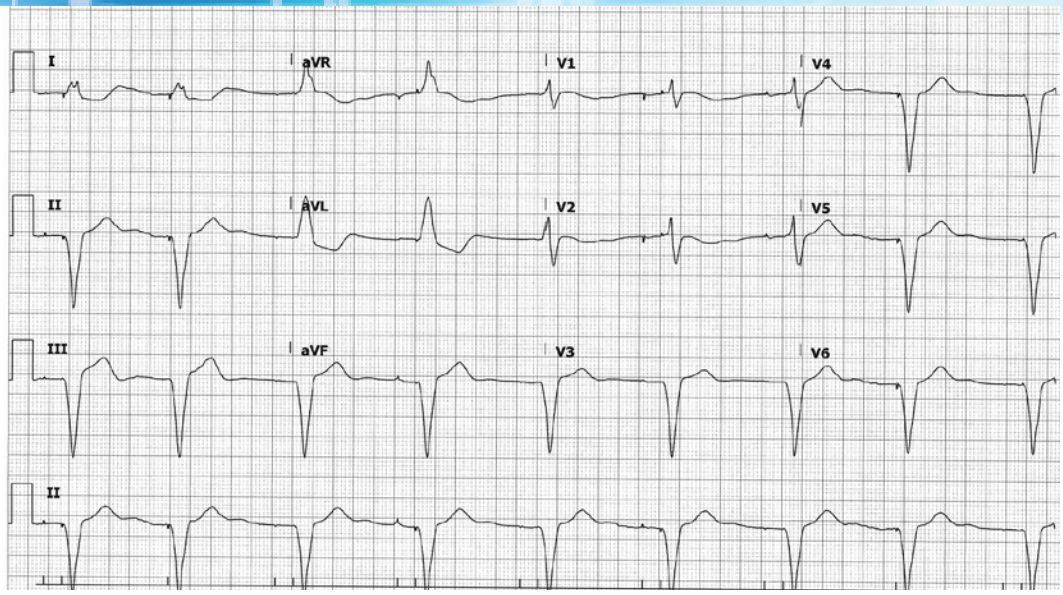
Sarah Frantz, MD
Christiana Care Health System

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

CASE. An 87-year-old female presents with generalized weakness for the past several days after a recent increase in her diuretic medication by her cardiologist.

What is your interpretation of the following ECG?
(Hint: there are 3 significant abnormalities.)

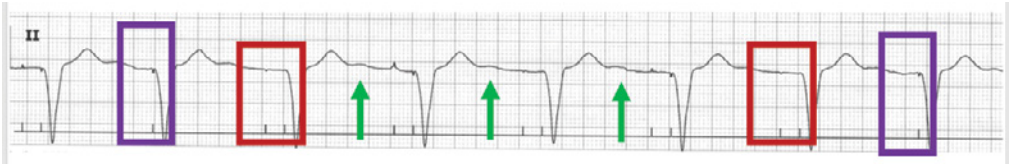
See the ANSWER on page 42



ECG Challenge

Answer: Pacemaker Malfunctions

This EKG shows an atrioventricular paced rhythm at 50 bpm. The 3 abnormalities are U-waves (green arrows), failure to pace (purple box), and failure to capture (red box).



1. U-waves (green arrows) are low-amplitude deflections that occurs after the T-wave, typically most visible in V2-V3 with bradycardic rates. U-waves can be seen with hypokalemia, and this patient's potassium was 2.2 mEq/L likely due to a recent increase in her furosemide dose.
2. Failure to Pace (purple boxes) describes the absence of pacer spikes when expected. Note that there are 2 pacers spikes for all the beats except for the 2 highlighted in blue boxes. The 2 pacer spikes represent the expected pacing seen with a dual chamber pacemaker: atrial pacing followed by ventricular pacing. The absence of the atrial pacing spikes in the purple boxes is likely due to the U-waves, which the pacer is mistaking for intrinsic P-waves. This was confirmed when there were no more episodes of failure to pace after the patient's hypokalemia was treated and the U-waves disappeared.
3. Failure to Capture (red boxes) describes the absence of myocardial depolarization after a pacing stimulus. Note that there is no P-wave after the initial (atrial) pacer spike in most of the beats. This also resolved after the patient's hypokalemia was corrected, and electrolyte abnormalities are a common cause along with ischemia and structural problems (eg, wire fracture)

LEARNING POINTS

Failure to Pace

General Features
- Paced stimulus is not generated when expected

EKG Features
- Decreased or absent pacemaker function

Clinical Significance
- Causes: oversensing, lead fracture, or insulation defect
  - — Oversensing: pacing inhibited by non-cardiac activity (eg, skeletal muscle activity) inappropriately recognized as native cardiac activity

Failure to Capture

General Features
- Delivery of pacing stimulus without subsequent myocardial depolarization

EKG Features
- Absence of depolarization after pacing spikes

Clinical Significance
- Causes: functional (electrode displacement, wire fracture) and pathologic (electrolyte disturbances, MI)

U-waves

EKG Features
- Low-amplitude deflection that occurs after the T-wave
- Most evident in V2 and/or V3
- Amplitude is rate dependent
  - — Increases with bradycardia
  - — Rarely seen with tachycardic heart rates

Clinical Significance
- Clinical significance not fully understood
- Can see U-wave larger than T-wave with moderate to severe hypokalemia
- Inverted U-waves in leads V2-V5 is abnormal (seen in ischemia and hypertension)
- When measuring QT, exclude U-waves or measure in a lead without U-waves (usually aVR or aVL)

Hypokalemia

General Features
- EKG triad of decreased T-wave amplitude, ST depression, and U-waves

EKG Features
- Decrease in T-wave amplitude seen first
- ST depressions ≥ 0.5 mm
- U-wave > T-wave amplitude in same lead

Clinical Significance
- Hypokalemia changes can be masked by tachycardia
- Concurrent hypomagnesemia can precipitate ventricular dysrhythmias
- Hypokalemia in MI is associated with increased risk of ventricular dysrhythmias

Case Conclusion

The patient was admitted to the Cardiac Short Stay Unit for electrolyte repletion, rehydration, and adjustment of her diuretic dose. Both her symptoms and her EKG abnormalities resolved with treatment, and she was discharged the next day. ✶

Board Review Questions

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

1. A 56-year-old man presents with pain and "cloudy vision" in his right eye since he woke up. He has had three similar episodes over the past several years that resolved over several days. He has a history of poorly controlled diabetes and occasional cold sores; he does not use contact lenses. Examination reveals decreased visual acuity, no photophobia, and no cell or flare. Slit lamp examination is difficult due to mild diffuse opacification of the cornea with small, white keratoprecipitates over the central inner surface of the cornea. No abrasions or dendritic ulcers are seen. What is the cause of the lesion?
   A. Consistently elevated blood sugar levels
   B. Exposure to high-intensity ultraviolet light
   C. Herpes simplex virus
   D. Systemic autoimmune disease

2. Which of the following therapeutic agents could worsen the hemodynamic status of an infant during a cyanotic tet spell?
   A. Bicarbonate
   B. Isoproterenol
   C. Morphine
   D. Phenylephrine

3. Which characteristic is more commonly seen with an atypical pneumonia than with other causes of pneumonia?
   A. Chest pain
   B. Dry cough
   C. Dyspnea
   D. Fever

4. For which ingested agent is it most appropriate to administer activated charcoal?
   A. Aspirin
   B. Ethylene glycol
   C. Lithium
   D. Sodium hydroxide

5. Which statement regarding pelvic fractures is correct?
   A. AP compression most commonly leads to fracture of bony prominences such as the iliac spines
   B. Fractures caused by lateral compression forces are associated with bladder injuries and hematuria
   C. Injury vectors that decrease pelvic volume are more likely to cause hemodynamic compromise
   D. Upper sacral fractures can injure nerve roots and result in loss of perineal sensation and rectal tone ✶

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

Get out of your inbox!

EMRA gets you in front of the decision makers!

EMRA Residency Program Fair

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

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

EMRA Job & Fellowship Fair

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

Looking for the next opportunity after you graduate? Don't miss the largest recruiting event in emergency medicine with nearly 220 organizations from all over the United States. This is your chance to network and find your dream position.

Exhibitor Registration Now Open!

emra.org/exhibitors

Need more information?

James Bryant
jbryant@emra.org
469.499.0187

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EMRA

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INDIANA

South Bend — Memorial Hospital of South Bend. Stable, Democratic, 23 member group seeks additional BC/BE Emergency Physicians. 60K visits, Level II Trauma Center, double, triple and quad physician coverage, also micro-hospital opening fall 2019. Equal pay, schedule and vote from day one. Over 400K total package with qualified retirement plan; group health and disability insurance; CME reimbursement, etc. Favorable Indiana malpractice environment. University town, low cost of living, good schools, 90 minutes to Chicago, 40 minutes to Lake Michigan. Teaching opportunities at four year medical school and with FP residency program. Contact Joseph D'Haenens at southbendemergency@gmail.com.

TEXAS

LEMA
LEADING EDGE MEDICAL ASSOCIATES
Clinical Excellence – Innovative Solutions

Leading Edge Medical Associates is a one-of-a-kind, private, independent group of all board-certified EM physicians in northeast Texas, offering a full range of clinical opportunities in EM. Our physicians enjoy shifts in a tertiary care trauma center as well as in nearby, lower volume clinical settings, all with high compensation and excellent full benefits. We are known for innovation in the industry and for developing strong EM leaders through LEMA's Leadership Development Institute. Almost half our physicians are former chief residents. LEMA is unique in its ability to offer physicians the best of both worlds, hospital-based and freestanding, academic and community medicine. LEMA is a group of exemplary physicians who work together as a team, value each member's input, and have a level of integrity, honesty, and trust that makes this innovative group truly one-of-a-kind. Interested in joining Texas's premier private group? Contact: SUZY MEEK, MD, CAREERS@LEMA-EM.COM.

WEST VIRGINIA

Huntington – Saint Mary's and VEP Healthcare are committed to high-quality care and patient satisfaction while providing physician support and high compensation. Saint Mary's Hospital in Huntington is a 393-bed acute care hospital. The emergency department cares for 57,000 patients annually and is equipped with 46 exam rooms with 3 trauma bays, and 4 psych beds. Offering 10-hour shifts using Sorian EMR system. VEP offers independent contractor status, paid malpractice +tail, stock ownership, flexible schedule, leadership training and development, and productivity-based compensation! Cindy Keller, (925) 482-8419, mkeller@vephealthcare.com, http://www.vephealthcare.com.

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?

Come join our team today!

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THE AREA

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

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.

RUTGERS
New Jersey Medical School

Academic Emergency Physician

Rutgers New Jersey Medical School, Newark, NJ

The Department of Emergency Medicine at Rutgers New Jersey Medical School in Newark, NJ, is recruiting highly qualified, full-time BC/BE Emergency Medicine Faculty at the Assistant or Associate Professor level.

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Optimal candidates will have a desire for clinical, academic, or administrative excellence. Subspecialty of other training desired, but anyone with clinical and academic aspirations is strongly encouraged to begin or enhance your career at Rutgers NJMS. The salaries are competitive, the institutions and leadership are very supportive, and the patient population is highly in need of quality healthcare.

Live nearby in beautiful suburban or urban New Jersey or within a short commute from New York City. The medical school is blocks from the New Jersey Institute of Technology and the Rutgers Newark Campus, as well as the rejuvenating downtown Newark area, and is close to Newark Liberty Airport and Newark Penn Station Amtrak.

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

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

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NSMC NORTH SHORE MEDICAL CENTER

EMERGENCY MEDICINE OPPORTUNITY NEAR BOSTON, MA

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ONE FOCUS.

One thing sets North Shore Medical Center apart—our team based model of care which is founded on the principle that physicians, nurses, and other care providers working together will provide higher quality and a better patient experience. Today, that team focus drives our providers to be leaders of quality of care, patient safety and process improvement initiatives throughout NSMC.

While practicing as an Emergency Medicine physician at NSMC you will enjoy:

- working at one of the top hospitals in Boston in a brand new Emergency Department that opened in October 2019
- the benefits of NSMC's membership in the Partners Healthcare System, founded by Massachusetts General Hospital and Brigham and Women's Hospital
- our annual adult ED volume of 80,000 visits provides an array of pathology with a fast track and PA program in place and excellent multispecialty back up
- a culture focused on communication, growth, and work/life balance
- excellent compensation and comprehensive fringe benefits
- being an active contributor to quality of care, patient safety and process improvement initiatives

Imagine the great things we can achieve together.

Interested candidates should send their CV to: Louis Caligiuri, Director of Physician Services at lcaligiuri@partners.org

24/7 EMERGENCY CARE, P.C.

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POSITIONS:
Full-time
Part-time
Per diem

Start a healthcare career on the beach in The Hamptons New York.

Full time partnership tracks include:

- Paid vacation/CME time
- $10,000/year CME budget
- Profit sharing
- 401(k) with match
- Major medical insurance
- Occurrence malpractice insurance
- Performance bonuses

Per diem positions include:

- All travel expenses and housing
- Potential participation in 401(k), profit sharing and bonus programs

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 25 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

Yale University School of Medicine Department of Emergency Medicine Fellowship Programs

For specific information including deadlines and requirements, visit http://medicine.yale.edu/emergencymed

The **Implementation Science** fellowship is a 2-3 year program that will train investigators in the principles and practice of dissemination and implementation science. Supported by a K12 grant from NIH's National Heart, Lung, and Blood Institute, fellows will receive training at the new Yale Center for Implementation Science (YCIS), the Yale Center for Clinical Investigation, and the National Clinician Scholars Program. Eligible candidates may receive a Masters in Health Sciences degree. Mentors come from the Yale Schools of Medicine, Nursing, and Public Health, as well as many community-based organizations in New Haven. For further information, contact **Steven L. Bernstein, MD, steven.bernstein@yale.edu**.

The **Research** fellowship is a 2-3 year program focused on training clinician scholars as independent researchers in Emergency Medicine. Scholars will earn a Master of Health Sciences degree from Yale combining clinical experience with extensive training in research methods, statistics, and research design. With the guidance of research content experts and professional coach mentors, the scholar will develop a research program, complete a publishable project and submit a grant application prior to completion of the program. The program is credentialed by the Society for Academic Emergency Medicine. For further information, contact **Steven L. Bernstein, MD, steven.bernstein@yale.edu**.

The **Yale Drug use, Addiction, and HIV Research Scholars (DAHRS)** Mentored Career Development Program (NIDA K12) provides a 3 year post-doctoral interdisciplinary, research training experience preparing investigators for careers focusing on drug use, addiction, and HIV prevention and treatment in general medical settings. Scholars earn the Master in Health Sciences degree that combines vigorous research methodology, statistics and design didactics in small group sessions and seminars covering topics related to drug use, addiction and HIV, leadership, grant writing and responsible conduct of research. Candidates complete mentored research project(s), multiple manuscripts, and apply for independent funding. For further information, visit **www.medicine.yale.edu/dahrs** or contact **Gail D'Onofrio, MD, MS, dahrs@yale.edu**.

The fellowship in **Emergency Ultrasound** is a 1 or 2 year program that will prepare graduates to lead an academic/community emergency ultrasound program. The 2-year option includes a Master of Health Sciences or Master of Public Health with a focus on emergency ultrasound research. This fellowship satisfies recommendations of all major societies for the interpretation of emergency ultrasound, and will include exposure to aspects of program development, quality assurance, properties of coding and billing, and research. The program consists of structured time in the ED performing bedside examinations, examination QA and review, research into new applications, and education in the academic/community arenas. We have a particular focus on emergency echo and utilize state of the art equipment, as well as wireless image review. Information about our Section can be found at **http://eus.yale.edu** . For further information, contact **Rachel Liu MD, rachel.liu@yale.edu, or apply online at www.eusfellowships.com**.

The **Administration** fellowship is a 2-year program that will prepare graduates to assume administrative leadership positions in private or academic emergency medicine as well as hospitals and health systems. The fellow will acquire experience in all facets of emergency department clinical operations, with close mentorship from department and hospital administrative leaders. Fellows will complete the recently #1 ranked Executive MBA program at the Yale School of Management. In addition, the candidate will assume graduated leadership roles on one or more projects supporting departmental activities usually culminating as Assistant Medical Director in the second year of the fellowship. For further information, contact **Arjun Venkatesh, MD, MBA, MHS, arjun.venkatesh@yale.edu**.

The **Global Health and International Emergency Medicine** fellowship is a 2-year program offered by Yale in partnership with the London School of Hygiene & Tropical Medicine (LSHTM). Fellows will develop a strong foundation in global public health, tropical medicine, humanitarian assistance and research. They will receive an MSc from LSHTM, a diploma in Tropical Medicine (DTM&H) and complete the Health Emergencies in Large Populations (HELP) course offered by the ICRC in Geneva. In addition, fellows spend 6 months in the field working with on-going Yale global health projects or on an independent project they develop. For further information, contact the fellowship director, **Hani Mowafi, MD, MPH, hani.mowafi@yale.edu**.

The fellowship in **EMS** is a 1-year program that provides training in all aspects of EMS, including academics, administration, medical oversight, research, teaching, and clinical components. The ACGME-accredited program focuses on operational EMS, with the fellow actively participating in the system's physician response team, and all fellows offered training to the Firefighter I or II level. A 1-year MPH program is available for fellows choosing additional research training. The fellowship graduate will be prepared for a career in academic EMS and/or medical direction of a local or regional EMS system, and for the ABEM subspecialty examination. For further information, contact **David Cone, MD, david.cone@yale.edu**.

The **Medical Simulation** fellowship is a 1-year program that provides training in all aspects of healthcare simulation, including high fidelity mannequin simulation with computer program training, acquisition of debriefing and teaching skills, use of novel wearable technologies, and procedural simulation. The fellow will participate in all educational programs for medical students, nurses, residents and faculty at the Yale Center for Medical Simulation. The program includes options to train in research methodology through the Research Division of the Department of Emergency Medicine and participate in medical education coursework through Yale School of Medicine. The fellowship will include attendance of the one-week Comprehensive Instructor Workshop at the Institute for Medical Simulation in Boston. For further information, contact **Leigh Evans, MD, leigh.evans@yale.edu**.

The **Educational Leadership** fellowship is a 1 or 2-year program that provides the training and education to develop academic emergency physicians to have the skills, knowledge and experience to be strong educators and leaders in Emergency Medicine education with the focus on developing leaders in EM residencies or in Undergraduate Medical Education. The fellow will be an Assistant Residency Program Director and an integral member of the education faculty. They will be supported to attend leadership training as well as using other internal resources, CORD and ACEP to further their education. For further information, contact **David Della-Giustina, MD, FACEP, FAWM, david.della-giustina@yale.edu**.

The **Wilderness Medicine** fellowship is a 1-year program that provides the core content of medical knowledge and skills in being able to plan for and to provide care in an environment that is limited by resources and geographically separated from definitive medical care in all types of weather and evacuation situations. The fellow will be supported to obtain the Diploma in Mountain Medicine and other Wilderness Medical education. The fellow will become a leader and national educator in the growing specialty of wilderness medicine. For further information, contact **David Della-Giustina, MD, FACEP, FAWM, david.della-giustina@yale.edu**.

The **Clinical Informatics** fellowship is a 2-year program that provides ACGME-approved training in all aspects of clinical informatics. The program is administered through the Yale Department of Emergency Medicine. In the first year, the fellow will rotate between the Yale-New Haven Health and Veterans Affairs. Major blocks will be devoted to electronic health records, clinical decision support, databases and data analysis, and quality and safety. Experiential learning will be combined with didactic classes and conferences. The second year is dedicated to advanced learning and project leadership. The fellow will attend the American Medical Informatics Association annual meeting. The program prepares fellows for Clinical Informatics Board examination. For further information, contact **Ted Melnick, MD, MHS, edward.melnick@yale.edu**.

All require the applicant to be BP/BC emergency physicians and offer an appointment as an Instructor to the faculty of the Department of Emergency Medicine at Yale University School of Medicine. Applications are available at the Yale Emergency Medicine web page http://medicine.yale.edu/emergencymed/ and are due by November 15, 2019 with the exception of the Clinical Informatics Fellowship, the Wilderness Fellowship, and the Educational Leadership Fellowship, which are due by October 1, 2019.

Yale University and Yale-New Haven Hospital are affirmative action, equal opportunity employers and women, persons with disabilities, protected veterans, and members of minority groups are encouraged to apply.

UT HEALTH
THE UNIVERSITY OF TOLEDO

The Department of Emergency Medicine at The University of Toledo Medical Center is seeking an energetic, highly motivated, and talented physician to join The Emergency Medicine Department.

Join a well-balanced Department with a welcoming environment from colleagues and dedicated support staff. The Level l Trauma Center provides the full spectrum of care with all specialties and subspecialties. The department of Emergency medicine has approximately 36,000 visits a year with high acuity of patients. In this practice, the EM Physician will be scheduled a total of 1,440 hours / year.

We are looking for candidates with strong interpersonal skills and ability to work in a dynamic academic and clinical environment. Applicants must have graduated from an accredited emergency medicine resident program and be board certified by ABEM/AOBEM.

We offer a competitive salary, academic stipend and excellent benefits. The University of Toledo is an equal access, equal opportunity, affirmative action employer, and educator.

Our mission is "to serve the Northwest Ohio community and Toledo Area by providing quality health care services in a courteous, compassionate and respectful manner to all people regardless of ability to pay."

For further information call Department of Emergency Medicine Administrator Hesham Youssef, MBA at 419-383-4439 or e-mail hesham.youssef@utoledo.edu.

WESTERN PENNSYLVANIA

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UPMC has a long history of emergency medicine excellence, with a deep and diverse EM faculty also a part of the University of Pittsburgh. We are internationally recognized for superiority in research, teaching and clinical care. With a large integrated insurance division and over 25 hospitals in Pennsylvania and growing, UPMC is one of the nation's leading health care systems. We do what others dream - cutting edge emergency care inside a thriving top-tier academic health system.

We can match opportunities with growth in pure clinical or mixed careers with teaching, research, and administration/ leadership in all settings - urban, suburban and rural, with both community and teaching hospitals. Our outstanding compensation and benefits package includes malpractice without the need for tail coverage, and employer-funded retirement plan, generous CME allowance and more.

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EOE Minority/Female/Vet/Disabled 72714C HRI&E 02/17

Emergency Medicine Opportunities

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When you join Geisinger, you'll be part of an organization that's leading healthcare change. Opportunities include **Emergency Medicine** and **Pediatric Emergency Medicine** full-time positions for experienced and new graduate physicians.

To learn about our competitive compensation and benefits packages which provide professional development and support for a healthy work/life balance, visit **geisingerjobs.org/emergency-medicine.**

Interested candidates, please reach out to **Miranda Grace** at **717-242-7109** or **mlgrace@geisinger.edu**

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AA/EOE: disability/vet.

EMERGENCY MEDICINE OPPORTUNITIES IN SOUTHEASTERN PA

Phoenixville Hospital, a 139 bed tertiary care facility located in Phoenixville, Pennsylvania, is growing its Emergency Medicine team with the following opportunities:

- **Emergency Medicine Physicians**
  - Day/Evening positions available
  - Nocturnal positions available
- **Advanced Practice Providers**
  - Both Physician Assistants and Nurse Practitioners will be considered
  - Emergency Medicine experience post-training is preferred
  - Opportunities to expand skill set in a challenging and exciting environment. Providers will not be limited to fast-track patients only. This position will include care for acute and critically ill patient panels.

Phoenixville Hospital:

- 24,000 patient visits annually
- 24 ED beds
- Triple coverage at peak times
- 24/7 OB/GYN, Pediatrics, Interventional Cardiology, and Anesthesia coverage
- Vibrant, diverse community provides a picturesque setting with a downtown that is the center of commerce and community living

What We Offer:

- Competitive compensation
- Comprehensive benefits including health, dental, vision, life, and disability insurance
- Retirement savings plan with employer automatic contribution and match
- Claims-made malpractice insurance with tail coverage
- Generous paid time-off allowance
- CME time and stipend
- Relocation assistance
- Supportive health system to advance goals

For more information, please contact:

Carrie Moore, MBA, *Physician Recruiter, Emergency Medicine*
484-628-8153 • Carrie.Moore@towerhealth.org
Careers.TowerHealth.org

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EOE

{ Job Opportunities }

Division Chief, Pediatric Emergency Medicine
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
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- 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

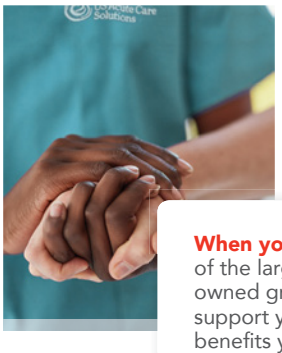
What the Area Offers:

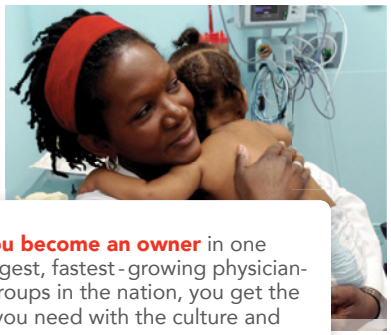
We welcome you to a community that emulates the values Milton Hershey instilled in a town that holds his name. Located in a safe family-friendly setting, Hershey, PA, our local neighborhoods boast a reasonable cost of living whether you prefer a more suburban setting or thriving city rich in theater, 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:

PennState Health

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





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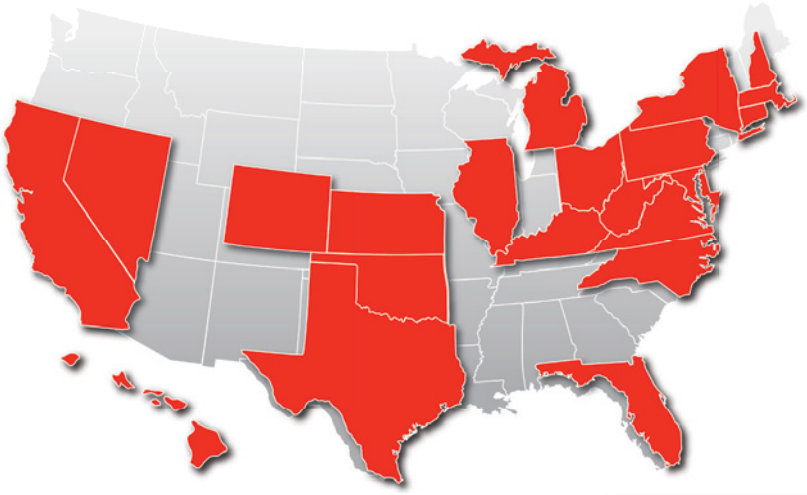

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Allegheny Health Network Emergency Medicine Management
Western, PA | 12-53,000 pts./yr.

CarlolinaEast Medical Center
New Bern, NC | 68,000 pts./yr.

Carolinas HealthCare System
Charlotte, NC | 15-77,000 pts./yr.

Carteret Health Care
Morehead City, NC | 49,000 pts/yr.

Catholic Medical Center,
Manchester, NH | 33,000 pts/yr.

⊕ **Doctors Hospital**
Columbus, OH | 72,000 pts/yr.

Fairfield Medical Center
Lancaster, OH | 37,000 pts/yr.

Lake Health System
Cleveland, OH | 10-34,000 pts./yr.

Mercy Health System
Cincinnati, OH region | 9-53,000 pts./yr.

MedStar St. Mary's Hospital
Leonardtown, MD | 49,000 pts./yr.

Meritus Medical Center
Hagerstown, MD | 68,000 pts./yr.

⊕ **Ohio Valley Medical Center**
Wheeling, WV | 25,000 pts./yr.

Saint Francis Hospital
Tulsa, OK | 107,000 pts./yr.

Sentara RMH Medical Center
Harrisonburg, VA | 68,000 pts./yr.

Sharon Regional Medical Center
Sharon, PA | 33,000 pts./yr.

Springfield Regional Medical Center
Springfield, OH | 62,000 pts./yr.

Stamford Health
Stamford, CT | 56,000 pts./yr.

Summa Health System
Akron, OH | 7-81,000 pts./yr.

Valley Children's Hospital (PEM)
Madera, CA | 113,000 pts./yr.

⊕ **University Medical Center**
Las Vegas, NV | 79,000 pts./yr.

⊕ Denotes an academic leadership position

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