University of CINCINNATI Emergency Medicine

ANNALS OF B POD

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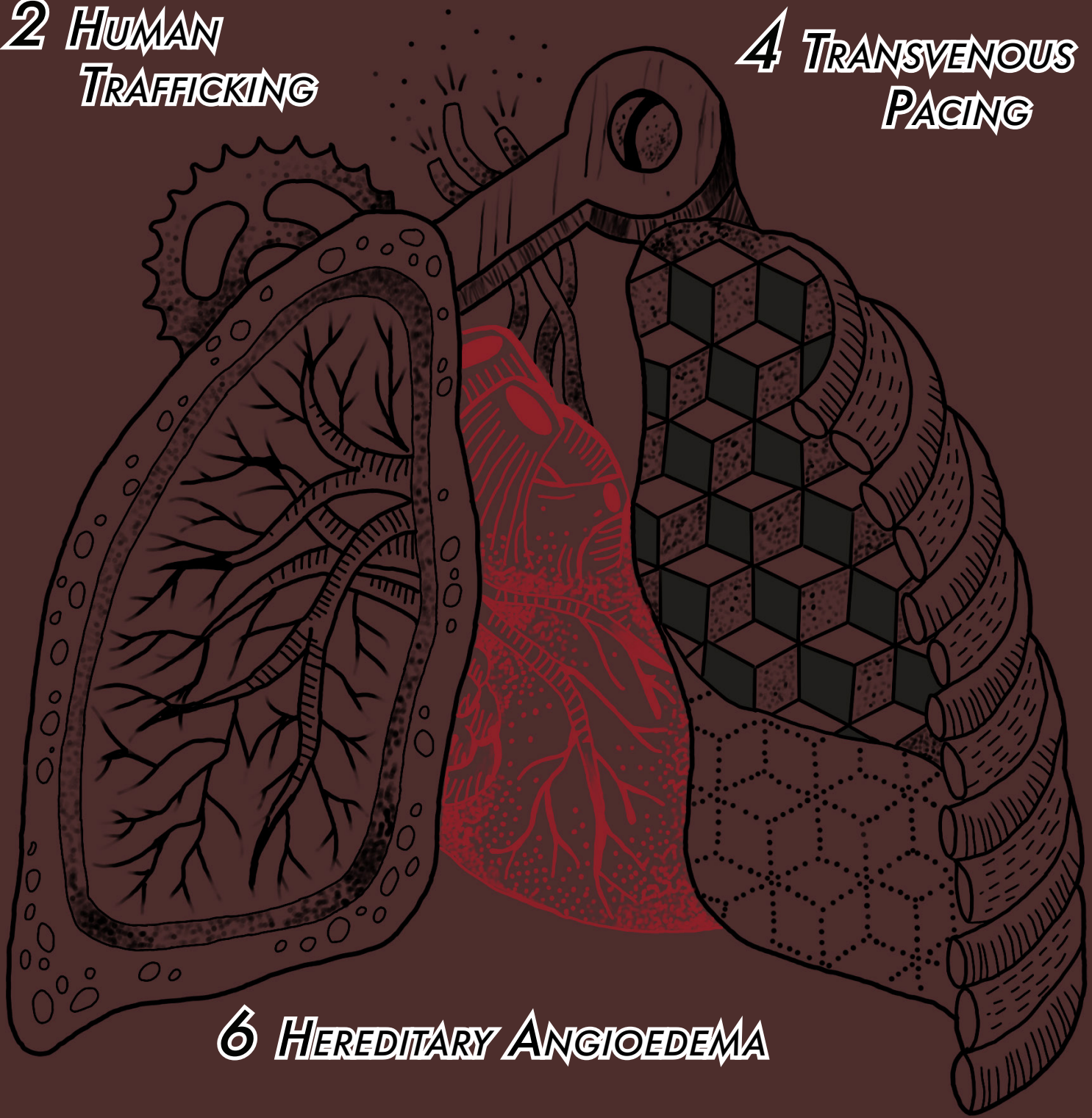
B POD: THE NUTS AND BOLTS



ANNALS OF B POD
FALL 2018

B pod starts to fall into a routine in the fall as interns embrace their roles as doctors and R4s as teachers. In the Fall 2018 issue of Annals of B Pod, we look to present cases that in a few months to years will become bread and butter to our interns with the AirCare Burns and STEMI articles. We also seek to offer cases that could prove difficult for even the most seasoned R4 with the Human Trafficking and Transvenous Pacing articles. Learn and enjoy.

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Human Trafficking

Kelli Jarrell, MD
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History of Present Illness

The patient is a female in her early thirties presenting via EMS after an unintentional opiate overdose. She received 2 mg of intranasal narcan approximately 10 minutes prior to presentation in the emergency department (ED). On arrival, she endorses nausea and dizziness but denies any other complaints.

Vital Signs

T: 98.0 HR: 88 RR: 14 BP: 106/70 SpO2: 98%

Physical Exam

She is generally well-appearing, non-toxic, and in no acute distress. Cardiovascular, pulmonary, and abdominal exams are within normal limits. Musculoskeletal and neurologic exams are also within normal limits. Skin exam is notable for ecchymosis of the bilateral lower extremities and multiple tattoos.

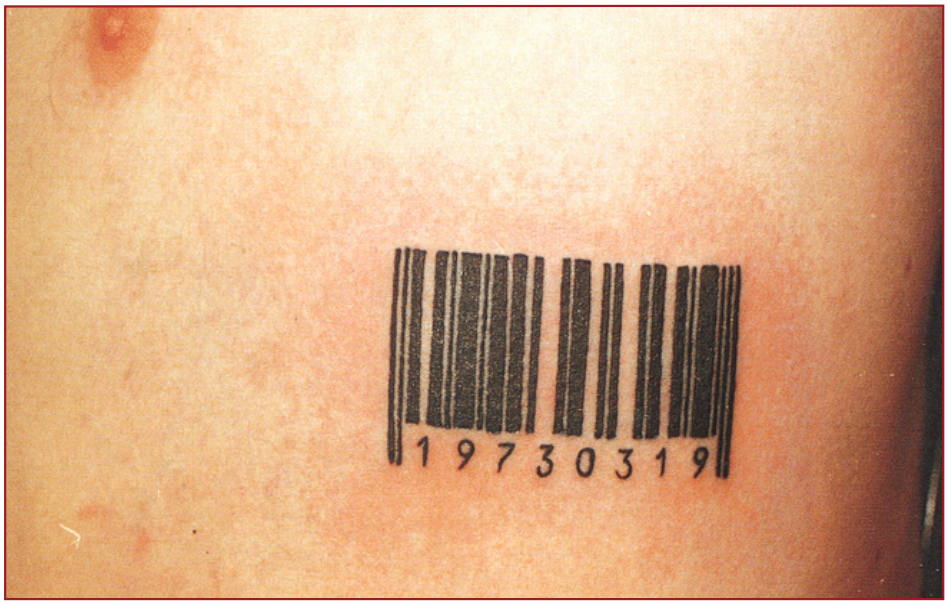

Image 1: Representative image of barcode tattoo, similar to the patient's tattoo[21]

Hospital Course

The patient is given oral ondansetron with improvement of her nausea. She is monitored in the ED and maintains adequate oxygen saturations without any respiratory distress. An HIV test is performed and negative. The social worker is involved to assist in disposition, and her assessment reveals that she is both jobless and homeless. The patient is noted to have bruises on her bilateral lower extremities from an unknown source, and she is unwilling to disclose how she received them. She states that she is from the Southwest U.S. and is involved with a younger man she met through a family friend. She recently moved in with this younger man, and reports that her family friend has videotaped her without her permission during intimacy with her significant other. The patient reports that she paid for a vehicle that this family friend is now withholding from her because she pressed charges against him. She also reports that this family friend secretly listens to her private conversa-

tions and will not allow the patient to leave her home independently. Based on this discussion, the patient is ultimately dispositioned to the Salvation Army's End Slavery Cincinnati program.

Discussion

Human trafficking (HT) is simultaneously complex and simple. While there is no "typical" human trafficking victim, repeated abuse, physical and sexual trauma, and addiction are the rule, not the exception, in the lives of victims of trafficking. Traffickers routinely and systematically destroy their victims' identities so that the men and women they traffic feel devoid of protection and can therefore be maximally exploited. For many victims, the process of de-identification starts long before they cross the threshold of any emergency department.

Simply put, trafficking is slavery. There are more slaves today than any other time in human history, with an estimated 40.3 million victims of human trafficking globally and 24.9 million people trapped in forced labor.[1] HT is also a very complex legal, ethical, social, and economic issue. It is the second largest and fastest growing organized crime trade in the world, recently surpassing the illegal arms trade, and anticipated to surpass the illegal sale of drugs in the next few years.[2,3] This growth surge is likely explained by the fact that HT is an unfortunately profitable industry, earning $150 billion in profit annually for traffickers, tripling the 2016 yearly profit of the first ranking Fortune 500 company.[4,5]

Physical	*Behavioral*
• Appears malnourished or shows signs of repeated exposure to harmful chemicals • Shows signs of physical and/or sexual abuse, physical restraint, confinement, or torture • Underdressed for the weather particularly during winter season • Has few or no personal possessions	• Fearful, anxious, depressed, submissive, tense, or nervous/paranoid • Exhibits unusually fearful or anxious behavior after bringing up law enforcement • Avoids eye contact • Loss of sense of time • Numerous inconsistencies in his or her story • Presents with an older man

Table 1: Red flags for human trafficking

The United Nations defines trafficking as "recruitment, transportation, transfer, harboring, or receipt of persons by improper means (such as force, abduction, fraud, or coercion) for an improper purpose including forced labor or sexual exploitation."[6,7] The U.S. government differentiates between those trafficked for sex and those trafficked for labor or services, dividing victims of HT into three groups.[7] The first group includes minors under the age of 18 years that have been persuaded into commercial sex. The second group is adult sex workers who are forced into commercial sex work via threat of bodily harm, coercion, or fraud. The third group includes those who are forced to perform labor against their will. Recent data shows that forced labor exploitation (64% of the total) and commercial sexual trafficking (19%) are the most common forms of human trafficking. However, the majority of trafficking profits (estimated at $99 billion) come from commercial sex work. The average annual profit generated by each woman in sexual servitude is $100,000, yielding profit margins between 100% to 1,000%. Additional forms of human trafficking include the illegal sale of human organs as well as the trafficking of children, primarily boys, for armed combat.[4,8]

An estimated one million persons are trafficked across international borders on an annual basis with 14,500 to 17,500 persons trafficked within and across the US.[9] Human trafficking has been reported in all 50 U.S. states and the District of Columbia.[3] Women and girls are disproportionately affected, as three quarters of trafficking victims are female. The average age of entrance into the commercial sex trade is 12 to 14.[11] Despite an estimated 2 million children entering the global human trafficking market per year, only a fraction of traffickers are prosecuted with just over 9,000 convictions for trafficking globally in 2016.[4]

Although there is no "typical" victim, marginalized populations such as homeless youth and those in extreme poverty are at especially high risk.[6,7] Traffickers prey on vulnerable groups such as adolescents and young adults with a history of child abuse, involvement in child protection and welfare systems, and those who identify as lesbian, gay, bisexual, transgender, or queer. Runaway youths are particularly vulnerable, and some experts suggest that adolescents are likely to be approached to participate in the commercial sex industry within 48 hours of being on the street.[11] Other at-risk populations are persons with disabilities, immigrants, migrant workers, ethnic minorities, and financially insecure persons with limited education or prospects for formal employment.[7] In the United States, American Indian and Alaskan Native women are frequently trafficked and disproportionately represented in prostitution arrests.

Although there has been much discussion of HT within national and international law enforcement, it is only recently that the health care community has joined the discussion. As many as 87.7% of victims of human trafficking have come into contact with the health care community, and the ED was identified as the most frequent setting where victims seek medical care.[12,13] Emergency providers have the unique opportunity to identify victims and intervene on their behalf.

Identifying those at risk can prove to be difficult since victims of human trafficking often have a wide array of physical, reproductive, and mental health problems. Acute traumatic injuries are common chief complaints. These injuries are often secondary to physical abuse, lack of protective equipment, or hazardous work conditions. Workplace injuries, exposure to chemicals, environmental exposure, and communicable diseases from poor living conditions are common in victims of labor trafficking. Common complaints in victims of sex traffick-

Continued on page 13

procedure

Transvenous Pacing

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Introduction

In clinical scenarios where a bradyarrhythmia is compromising a patient's hemodynamics or causing clinical symptoms, transvenous pacing is a crucial temporizing measure that emergency physicians need to have in their armamentarium. Tachyarrythmias, albeit more often treated with cardioversion or pharmacotherapy, can occasionally be treated with overdrive pacing to suppress atrial fibrillation, atrial flutter, or torsades de pointes. However, bradyarrhythmias will be the primary indication for pacing in the emergency department. The following will discuss why, when and how to successfully transvenously pace a patient.

Indications

Transvenous pacing is a temporizing measure until underlying etiologies can be addressed. Transvenous pacing can also serve as a bridge to permanent pacemaker placement. Indications for transvenous pacing include: failure to create an electrical impulse at the sinoatrial node, cardiac stunning after blunt cardiac trauma , acute myocardial infarction, recent cardiac surgery, and sick sinus syndrome. Failure to conduct an electrical impulse through the atrioventricular (AV) node can lead to fatal bradycardias requiring pacing, as is seen in conditions such as second degree Mobitz II, third degree block, and bundle branch blocks. Finally, metabolic and electrolyte derangements such as hyperkalemia and drug toxicities (e.g., calcium channel blocker overdose) may require transvenous pacing. It is important to note that achieving capture is often quite difficult in these cases, and definitive care requires correction of the patient's metabolic derangements. There are certain situations in which transvenous pacing has been proven to not be beneficial, including asystolic arrest, traumatic arrest and hypothermia-induced bradycardia.

Studies have shown that 3-15% of acute ST-elevation myocardial infarctions (STEMIs) are associated with high degree AV block in the immediate post-infarct period. The most common bradyarrhythmias encountered are second-degree Mobitz II or third-degree heart blocks. Patients with inferior STEMIs are 2-4 times more likely to develop a conduction abnormality when compared to anterior STEMIs. Inferior STEMIs are often associated with heart block due to increased parasympathetic activity; these arrhythmias are often transient. However, anterior STEMIs associated with heart block are often due to AV node destruction from decreased septal perfusion and portend a worse prognosis.2 According to the 2013 ACCF/AHA guidelines on managing STEMIs, bradycardia associated with inferior and posterior infarcts can largely be temporized with atropine and pacing if needed. However, anterior and lateral infarcts usually require pacing as first line therapy, and may require permanent pacemaker placement.3 There are certain situations in which transvenous pacing has been proven to not be beneficial, including asystolic arrest, traumatic arrest and hypothermia-induced bradycardia.

While initiating temporary pacing is associated with a doubling of in-hospital mortality rates, this is likely due to the poor prognosis associated with high degree blocks in STEMI patients and not with the intervention itself.4 Thus, temporary pacing is warranted to facilitate more definitive therapy and increase survival rates. Given the risk of morbidity and mortality associated with ischemia-induced bradycardia, it is vital that all emergency medicine providers are able to perform this potentially life-saving procedure.

Alternatives

The most common methods of pacing used in the ED are transcutaneous and transvenous pacing. Although transcutaneous pacing can be started quickly and easily, it tends to be less pragmatic long term. The amount of chest wall musculature and structures between the transcutaneous electrodes and the targeted myocardium generally require more energy than transvenous pacing to ensure reliable capture. Transcutaneous pacing is more uncomfortable for the patient and frequently requires analgesia or sedation. Hypotension secondary to sedation may make transcutaneous pacing an even less viable option. Thus, it is reasonable to prepare for a transvenous cardiac pacer after initiating transcutaneous pacing.

Supplies

1. Central venous access:
2. Triple lumen catheter kit
3. Sterile gown, gloves, cap, mask
4. Sterile drape and towels
5. Ultrasound, probe cover, sterile gel
6. Cleaning solution (Chlorhexidine or betadine)
7. Sheath introducer – either Cordis or MAC
8. Pacing apparatus:
   - Pacer sheath
   - 5F bipolar pacing catheter
   - External Medtronic pacing generator – contains connector and red cable connector pins in bag

Procedure

The procedure itself can be divided into two objectives. The first step is to establish central venous access with a large bore sheath-introducer. After this is achieved, the next step involves introducing the pacing apparatus through the sheath and "floating" the pacer into the right ventricle. Appropriately placed, the electrode reaches the right ventricle and paces the ventricular endocardium in a VVI mode (ventricular-sensing, ventricular-pacing, inhibited by intrinsic activity). Once the pacer has been appropriately placed, one must understand and be able to troubleshoot common issues with the pacer box. The two main settings where providers may

encounter difficulty are with output and sensitivity settings. The output should be increased until capture is obtained, while the sensitivity should initially be on a high setting and decreased until the pacemaker fires at the appropriate time.

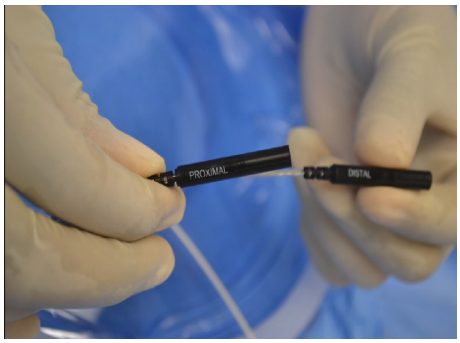
In most cases, the right internal jugular or left subclavian approaches are best suited for transvenous pacing, as these sites allow for a more direct line of passage into the right ventricle. While the left internal jugular and right subclavian can be utilized if other sites are unavailable, they provide a more tortuous pathway that may make the procedure more challenging. If both right internal jugular and left subclavian are accessible, the right internal jugular site is preferred, leaving the left subclavian available for permanent pacemaker placement if necessary. Brachial sites are generally less suitable due to increased risk of infection and thrombotic complications. Femoral access usually requires a rigid introducer catheter to traverse the long trajectory into the right ventricle and is most safely accessed under fluoroscopy.

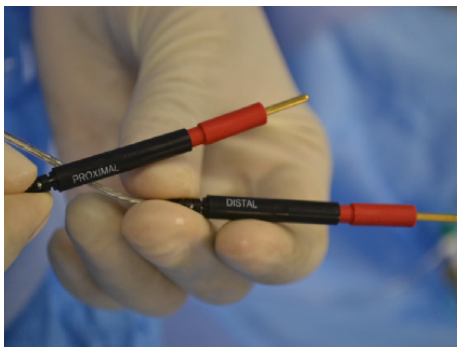
Central venous access:

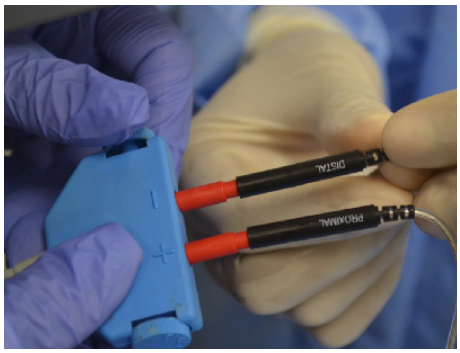
1. Prepare the site using sterile technique by cleansing the area, draping the patient, applying a sterile ultrasound probe cover, and donning a gown, cap, mask, and sterile gloves
2. Prepare the equipment by testing the wire, assembling the introducer-dilator, and flushing the introducer
3. Anesthetize the region
4. Using the ultrasound, gain access to the central vein of choice with needle and syringe
5. Remove the syringe and feed the wire through the needle
6. Remove the needle while ensuring stabilization of the wire, and use the scalpel to make a wider skin opening
7. Advance the dilator-introducer complex along the wire and into the vessel
8. Remove the wire and dilator, leaving the introducer in place

Pacer introduction:

1. Ensure the pacer catheter is one size smaller than the introducer apparatus
2. Ensure integrity of the pacer balloon by inflating with approximately 1.5mL of air and deflate
3. Feed the pacer sheath onto the distal end of the pacer catheter – do not extend
4. Prepare the pacer apparatus:-Red cable connector pins should be placed on the ends of the black electrodes
   - Connect red cable connector pins to the hub of the connector wire
   - Proximal cable inserts into the positive port
   - Distal cable inserts into the negative port
   - Non-sterile assistant tightens thumb screws on connector hub to ensure that connector pins are securely in place
   - Place the connector wire into the pacer generator in the "V" slot, which stands for "ventricular"







Images 2-4: Assembling the pacer apparatus

5. Advance the tip of catheter into the introducer approximately 15cm to ensure the balloon has cleared the end of the introducer
6. Inflate the balloon with 1.5ml of air and lock the catheter
7. Have the non-sterile assistant turn on the generator
8. Set the generator to 80bpm, current to 10-20mA and sensitivity to 2mV so the pacer will fire asynchronously
9. Start advancing the pacer catheter while monitoring the generator screen
10. Once capture is achieved , stop advancing and carefully deflate balloon
11. Suture in the catheter, noting the distance of catheter
12. Stretch out the pacer sheath along the remaining pacer catheter to allow for sterile advancement or retraction of the catheter
13. Use ultrasound to visualize the pacing catheter tip in the right ventricle

Post-Procedure

Post-procedure management includes obtaining a chest X-ray to ensure appropriate placement of the transvenous pacer, as well as obtaining an EKG to ensure appropriate capture. In terms of settings for the pacing generator, there are three controls that need to be adjusted. Voltage control should be set at 2-3 times the minimum value of ventricular capture. The rate should be set to whatever ensures hemodynamic stability. Sensitivity control should be set to the range that paces only when intrinsic cardiac activity is not enough to improve hemodynamics.

Complications

This procedure is not without its complications, associated with either central venous access or pacing catheter advancement. Complications from central venous access include arterial puncture and dilation, hematoma formation, and pneumothorax. Complications from catheter advancement are primarily cardiac in nature and include myocardial perforation, tricuspid valve injury, arrhythmia, and lead dislodgement.

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bpodcase

Hereditary Angioedema

Chris Shaw, MD
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History of Present Illness

The patient is a male in his early twenties with a past medical history notable for hereditary angioedema (HAE) and opioid abuse who presents to the emergency department (ED) complaining of tongue swelling. He was found by EMS providers on the morning of presentation after reportedly using heroin. He received naloxone, became alert and oriented, and was taken to an outside hospital for evaluation. After a reassuring examination and a normal head CT were performed, he was discharged to a correctional facility in police custody. Soon after discharge, the patient began complaining of tongue swelling and dysphagia. The swelling then progressed to include his left eye, so he was taken to the ED for further evaluation. On arrival, the patient reports significant swelling of his tongue and left eye as well as vision changes in that eye. He denies shortness of breath, voice changes, nausea, vomiting, abdominal pain, and rash.

Past Medical History

Hereditary angioedema
Heroin use disorder

Past Surgical History

None

Medications

None

Allergies

None

Vitals

T 37 HR 57 BP 122/82 RR 14 SpO2 100% room air

Physical Exam

The patient is a young adult male who appears his stated age and is in no apparent respiratory distress. There is a small abrasion to the left forehead. The tongue is moderately edematous, both anteriorly and posteriorly, occluding the majority of the posterior oropharynx from view. The left upper and lower eyelids both exhibit significant edema extending beyond the orbit, which is tender to palpation, but not warm to touch. There is no erythema. Cardiovascular, pulmonary, abdominal, and neurologic exams are within normal limits.

Labs & Imaging

14.1		
6.2		177
41.6		

137	104	8	81
4.0	27	0.79	

CK: 464

Hospital Course

Upon presentation to the ED, the primary concern was for an acute exacerbation of the patient's known hereditary angioedema. Initially he was protecting his airway and handling his secretions appropriately. He was given oral diphenhydramine, famotidine, and intravenous dexamethasone. An infusion of a C1 esterase inhibitor was administered. After a short period of observation, he began complaining of dyspnea at rest and difficulty swallowing. For this reason, he was electively intubated due to concerns for impending airway occlusion. The patient was fiberoptically intubated and no glottic or laryngeal edema was noted during the intubation. He was subsequently admitted to the medical intensive care unit (MICU) for further monitoring and management of his angioedema.

The patient had a prolonged MICU stay after developing ventilator associated pneumonia, and he was ultimately extubated on hospital day 12. At his outpatient appointment with allergy and immunology he was provided with a prescription for icatibant to self-administer in the event of future acute attacks.

Discussion

Angioedema is a physical exam sign defined as non-pitting edema that is transient and confined to a specific anatomic region.[1] Hereditary angioedema (HAE) is a diverse group of rare inherited disorders resulting from a mutation in the C1-inhibitor gene. This disease classically presents with intermittent eruptions of deep dermal swelling.

The pathophysiology underlying HAE is complex, as is seen in Figure 1.[2] The problem stems from a mutation in the sequence of C1 esterase inhibitor. This leads to a dysfunctional endogenous enzyme, which in turn results in a surge of bradykinin. Bradykinin increases endothelial cell activation, resulting in an efflux of fluid into the interstitium.3 Factor XII is also involved in this process by activating kallikrein and prompting release of bradykinin from the HK-B complex.

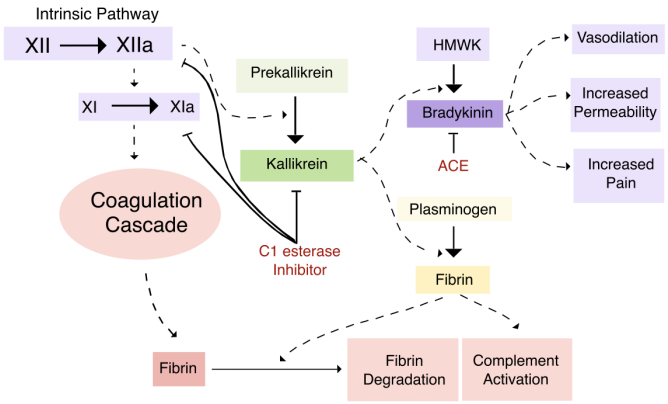

Figure 1: Kallikrein-Bradykinin Pathway

It is unclear why HAE attacks occur, but they often occur following "triggers" specific to the affected individual. The most common trigger of an HAE attack is mental stress, followed by menstruation, physical exertion, infection, and occasionally narcotic abuse. Patients will usually be cognizant of their specific

triggers and try to avoid them if possible. Patients with HAE usually have a care plan in place when they are exposed to a trigger and will usually present to the nearest ED for treatment. Emergency physicians should therefore be familiar and comfortable managing these patients when they present following an acute HAE attack.

In the patient described above, the primary concern was for airway compromise due to laryngeal edema. However, gastrointestinal and cutaneous symptoms are much more common than laryngeal involvement in HAE.[2] Gastrointestinal attacks generally produce crampy, severe abdominal pain accompanied by nausea, vomiting, and diarrhea. Management focuses on analgesia and investigation of other emergent causes of the abdominal pain. Unfortunately, many unnecessary procedures are performed on undiagnosed HAE patients with abdominal pain due to the severity of their symptoms.[3] These attacks may be accompanied by hypotension due to fluid efflux into the bowel wall that can mimic intra-abdominal sepsis. Cutaneous attacks may lead to edema in many parts of the body including the face, distal extremities, or genitalia. Management in these cases focuses on early administration of the therapeutic agents discussed below.

On initial presentation, HAE may be easily confused with anaphylaxis, particularly if there is no personal or family history. Anaphylaxis is due to the massive release of pre-made granules from mast cells and basophils. The biologically active components of these granules are histamine and tryptase. These compounds cause urticaria, bronchoconstriction, nausea, vomiting, and circulatory collapse.[4] Morbidity and mortality is reduced with early administration of intramuscular epinephrine. Adjunctive treatment with antihistamines and glucocorticoids reduces symptom burden and prevent theoretical late-phase effects of anaphylaxis. The symptoms of HAE are caused by binding of the bradykinin receptor, which is unaffected by antihistamines or epinephrine. There is no published data examining the effect of epinephrine in acute attacks of HAE, although anecdotally some authors report transient benefit.[1,6] Despite the lack of evidence for epinephrine in HAE, clinicians should strongly consider early epinephrine for patients in distress presenting with angioedema. Both conditions present with significant overlap and anaphylaxis is much more common than HAE. Epinephrine and antihistamines are readily available in the ED. Therefore, it is generally recommended to administer epinephrine and antihistamines to patients with undifferentiated angioedema in the ED.

Diagnostics

The primary focus in most cases of HAE will be on airway management. However, obtaining C4 and tryptase levels during an acute event can be very helpful for the admitting team or for outpatient follow up with an allergist.[7] C4 levels help inpatient teams confirm the diagnosis of HAE when the clinical picture is not entirely clear. Tryptase is useful to differentiate anaphylaxis from HAE.

Management

Airway protection is a common concern in HAE patients. It is difficult to separate which patients may experience simple facial swelling from those who may go on to develop laryngeal edema and airway compromise. In a retrospective review of 123 patients with HAE, laryngeal edema was preceded by facial swelling in only a small minority of patients.[8] Only six patients in this cohort required definitive airway management, with four patients requiring cricothyrotomy. In a second retrospective review examining 58 patients with known HAE, 23 deaths were attributed to asphyxiation secondary to airway edema.[9] Long term follow up of known HAE families in Hungary suggested that edema of the face and lips preceded 15-30% of clinical events of upper airway compromise.[10]

There are currently no published guidelines for definitive airway management of the patient with HAE and this decision is primarily based on the provider's clinical judgment. While physical exam is unreliable at determining who is at high risk of developing airway obstruction, adjunctive techniques can provide greater clarity. Nasopharyngoscopy is an essential tool for evaluating patients presenting with HAE. A consensus statement published by both emergency and allergy physicians recommends visualization of the supraglottic structures in patients with voice changes, hoarseness, angioedema of intraoral structures, or stridor on examination.[1] Intubation is recommended if the swelling extends to any airway structures or the base of the tongue. It is important to remember that intubation will not address the patient's underlying pathophysiology. Adjunctive pharmacotherapy is still required in order to counteract ongoing edema.

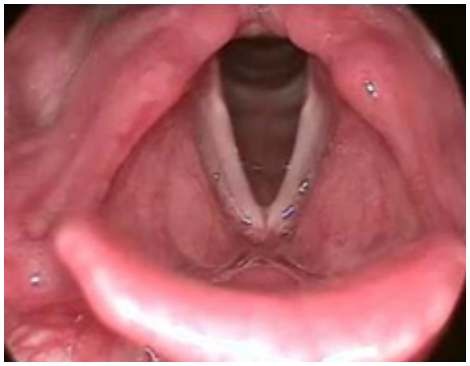

Image 5: Representative image of normal larynx[22]

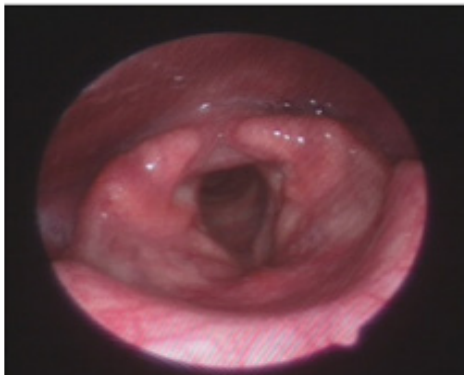

Image 6: Representative image showing laryngeal edema[23]

Acute Management of Hereditary Angioedema
First Line Therapies
C1 Esterase Inhibitor (C1INH): 20 units/kg infusion over 10 minutes
Icatibant (bradykinin receptor antagonist): 30 mg subcutaneous injection
Ecallintide (kallikrein inhibitor): 30 mg intramuscular injection
Second Line Therapy - Fresh Frozen Plasma: 3 units every 4 hours

Table 2: Medications for the management of hereditary angioedema

C1 esterase inhibitor

Molecular therapies for HAE were first described in 1980, when C1 esterase inhibitor (C1INH) was used to treat acute attacks.11 C1INH is administered as an infusion of 20 units per kilogram over 10 minutes. C1INH has

Continued on page 12

aircarecase

Air Care Annals: Burn Management

Shaun Harty, MD
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History of Present Illness

Air Care 1 was dispatched for a scene flight involving a 20-year-old male patient with extensive thermal burn injuries from self-immolation. In addition to his severe burns, the patient was reportedly hypoxic on a non-rebreather mask and tachycardic. Emergency Medical Services (EMS) at the scene reported that the self-immolation occurred outside of an apartment building and there were no known additional traumatic injuries. The patient was only able to vocalize his name, age, and that he had no known drug allergies. EMS had established an 18-gauge IV and administered one liter of normal saline.

Past Medical History

Unkonwn

Past Surgical History

Unknown

Family History

Unknown

Medications

Unknown

Allergies

NKDA

Vitals

HR 116 BP 118/60 RR 24 SpO2 97% on non-rebreather

Physical Exam

The patient was an obese male who appeared his stated age, was ill-appearing, and smelled of gasoline. He had full thickness burns to his face, head, neck, chest, abdomen, upper back, and upper and lower extremities. He had partial thickness burns of the remaining back and his genitals. He was awake with a Glasgow Coma Scale (GCS) of 15. Pupils were 3 mm bilaterally and reactive. He had carbonaceous material in his oral cavity. He was tachypneic with clear breath sounds bilaterally. He was tachycardic with a normal S1 and S2 without appreciable murmur. His central pulses were bounding. His abdomen was soft and not distended.

Prehospital Interventions

Given the erythema, edema, and carbonaceous material in the patient's airway, the air medical crew team was concerned about airway compromise and impending obstruction, and elected to proceed with endotracheal intubation in anticipation of complete airway obstruction. Ketamine and succinylcholine were used to facilitate rapid sequene intubation (RSI). The patient's airway was secured via direct laryngoscopy on the first attempt sans hypoxia, and was confirmed with end-tidal capnography. The Air Care crew continued IV fluid resuscitation and rapidly transported the patient to the level 1 trauma center..

Hospital Course

Upon arrival at the level 1 trauma center, the patient lost his only point of IV access during transport. Additional peripheral access was obtained and a femoral central venous line was placed. The patient's total body surface area burned was estimated at 91%. Fluid resuscitation was initiated per the Parkland formula and the patient was admitted to the burns specialty care unit (BSCU). Escharotomies were performed at the bedside on the anterior torso, and the upper and lower extremities. Burn debridement and allografting was performed several times during the patient's admission. The patient's hospital course was complicated by septic shock and high dose vasopressor requirement. Unfortunately, the patient's clinical status continued to deteriorate and care was withdrawn one month after admission.

Discussion

Thermal burn injuries are encountered frequently in the pre-hospital setting. According to the American Burn Association, there are approximately 486,000 patients treated annually for burn injuries. 40,000 patients are admitted annually for inpatient burn management, and 3,275 patients die from exposure to fire, flames, or smoke.[1] Pre-hospital critical care providers must quickly gather information and begin appropriate therapeutic interventions while transporting these patients to definitive care.

Obtaining a history from a patient with severe burns is often difficult due to airway compromise or profound shock. Critical care transport providers should obtain collateral information and a pertinent history from ground EMS providers once the patient has been stabilized. Important historical elements include whether the patient was in an enclosed space, if there was any associated traumatic injury, or if there was any chemical component to the burn requiring decontamination. Any patients with burns to the face, hands, soles of the feet, genitals, or greater than 15 percent of total body surface area should be transported to a burn center.

The initial approach to the severely burned patient follows the ATLS algorithm and begins with the primary survey. Providers should place the patient on a continuous cardiac monitor, obtain large-bore IV access, and provide supplemental oxygen if necesary. IV access should be placed in healthy, non-injured skin.[2] If IV access can only be obtained through burned skin, it must be well secured. Subcutaneous edema and weeping fluid from burned skin can easily displace the IV cannula. Sutures or staples can be used to prevent loss of access during transport.

A thorough airway assessment is critical in burned patients. In addition to determining patency, providers must investigate for the following:

- Erythema, edema, or blistering of the neck, face, lips, and oropharynx
- Carbonaceous material (soot) in the oropharynx
- Singeing of facial or nostril hairs
- Voice changes[3]

Patients with evidence of airway involvement often have associated inhalation injury. Inhalation injury includes supraglottic thermal injury, subglottic chemical injury, and systemic toxicity from products of combustion.[4] Laryngeal edema may progress to complete airway obstruction within minutes of onset.[5] Early endotracheal intubation should be performed if supraglottic structures are injured before laryngeal edema develops.[4]

It can be difficult for critical care transport providers to determine when endotracheal intubation is indicated. Isolated singeing of facial hair and partial thickness facial burns with no oropharyngeal signs are not definitive indications for endotracheal intubation. It is reasonable to defer definitive airway intervention in these patients if the expected trasport time is short. However, providers must remember that these physical exam findings are associated with occult inhalational injuries and these patients should be closely monitored during transit.[6] Patients with oropharyngeal edema, carbonaceous material in the airway, vocal changes, severe face or circumferential neck burns should be intubated. In general, critical care transport providers should have a very low threshold to secure the patient's airway with endotracheal intubation prior to the development of more significant edema and obstruction.

A burned airway should always be considered a difficult airway.[6] Providers should have their backup plans and rescue devices immediately available. An endotracheal introducer such as a bougie should be considered early to maximize first past success.[7] Video laryngoscopy can help navigate edematous and distorted airway anatomy. A supraglottic device can be used as a rescue device if an endotracheal tube cannot be secured. An "awake look" with topical anesthesia and procedural sedation with ketamine would be an excellent approach to this difficult airway. Unfortunately, this may not be possible given the limited resources in the pre-hospital setting. Surgical cricothyrotomy should be performed if orotracheal intubation is not possible and the patient cannot be oxygenated or ventilated by other means, such as an LMA.

Once the patient's airway has been assessed and intervened upon as necessary, providers should move on to the next portion of the primary survey. Two specific findings should be considered during assessment of the patient's respiration. First, patients that have sustained circumferential full thickness burns of the chest can develop an eschar. An eschar is comprised of leathery, inelastic burned skin.[3] Edema forms beneath the eschar due to both the burn itself and crystalloid resuscitation. This will restrict ventilation and manifests clinically as difficult bag-mask ventilation or high peak pressures on the ventilator. When ventilation is inhibited by eschar, an escharotomy is indicated to improve respiratory biomechanics. Escharotomy is a surgical procedure where incisions are made through the eschar to the depth of the subcutaneous fat. Most escharotomies are performed two to six hours after a burn injury. There are multiple methods to perform chest wall escharotomies. The most common method for critical care transport providers begins with incisions from the clavicle to the costal margin in the anterior axillary line bilaterally. These incisions can then be connected by a transverse incision across the costal margin as shown in Figure 1. Full thickness burns are insensate, so no anesthesia is needed. Silver nitrate can be used for hemostasis of the incisions, and the wounds should be loosely packed with saline-soaked sterile gauze.[8]

The second important component of respiratory management is to utilize a lung protective ventilation strategy. Approximately half of intubated burned patients admitted to burn centers develop acute respiratory distress syndrome (ARDS).[9] Lung protective ventilation has a proven mortality benefit in ARDS.[10] This strategy avoids barotrauma by setting the tidal volume at 6-8 mL/kg of ideal body weight and keeping plateau pressures less than 30 cmH2O. Providers should use a PEEP scale to rapidly titrate PEEP and FiO2 to a goal oxygen saturation of 88-95%.[11] The exception to this rule is if there is a suspected carbon monoxide inhalation, in which case the FiO2 should be kept at 100%.

Burned patients have an overwhelming systemic inflammatory response. There is increased secretion of catecholamines, cortisol, glucagon, renin-angiotensin, antidiuretic hormone, and aldosterone. Fluid and proteins are lost in the burned tissue. All of these changes can lead to profound intravascular hypovolemia and vasodilation resulting in hypotension.

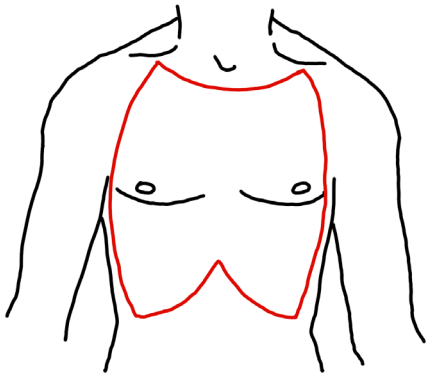

Figure 1: Chest Escharotomy

Once IV access has been obtained and the patient's hemodynamics have been addressed, providers should complete a thorough exam for circumferential extremity burns, which can cause compartment syndrome. A significant amount of pressure can develop posterior to a circumferential burn from edema below the inelastic, burned skin. This will result in irreversible nerve and muscle injury if not addressed quickly. Compartment syndrome from a burn is treated with escharotomy as opposed to fasciotomy in traditional compartment syndrome. Limb escharotomy is not routinely performed by critical care transport providers because it is not immediately life-threatening. Compartment syndrome typically develops over the course of hours once the patient has arrived at the burn center.

Continued on page 14

FiO_2 and PEEP scale from ARDSnet ARMA trial																
FiO_2	0.3	0.4	0.4	0.5	0.5	0.6	0.7	0.7	0.7	0.8	0.9	0.9	0.9	1	1	1
PEEP	5	5	8	8	10	10	10	12	14	**14**	14	16	18	20	22	24

quickhit

Complete Heart Block

Jessica Koehler, MD
University of Cincinnati R2

History of Present Illness

The patient is a 42-year-old male who presented to the Emergency Department (ED) after a syncopal event. This event occurred while the patient was walking down a hallway at work. He denied prodromal symptoms and recalled later waking up on the floor. He is unsure how long he was unconscious and his co-workers woke him up. He was back to his normal neurologic baseline immediately. His initial presentation did not reveal any social history of drug abuse. His workup during this visit included an EKG that demonstrated normal sinus rhythm with normal intervals, and no ST segment or T wave changes. His finger stick blood glucose was 120. Cardiology was consultedand the patient was discharged home with a Holter monitor. The patient was called two days later and asked to return to the ED after arrhythmias were recorded on the Holter monitor. During this visit, the patient's only complaint was generalized fatigue. He denied chest pain, shortness of breath, or any additional syncopal or pre-syncopal events.

Past Medical History	Medications	Past Surgical History
Hypertension Hyperlipidemia	Atenolol Bupropion Cyclobenzaprine Fluoxetine Omeprazole Simvastatin	None
		Allergies
		None

Vitals

T 37 HR 57 BP 122/82 RR 14 SpO2 100% RA

Physical Exam

The patient was in no acute distress. His head was normocephalic and atraumatic. He had full range of motion of the neck without tenderness. Cardiopulmonary and abdominal exams were normal. Distal pulses were 2+ and there was no peripheral edema. The patient was awake and alert, with normal mental status and a non-focal, non-lateralizing neurologic examination.

Labs & Imaging

138	104	11	120
3.6	24	0.90	

Hemoglobin: 12.3
LFTs: ALP 55, AST 13, ALT 21
Troponin <0.04 x2
TSH 1.69 T4 0.72
Serum Lyme: Negative

Hospital Course

Cardiology was consulted to assist with interrogation of the patient's Holter monitor, which was remarkable for 6 to 7 seconds of complete heart block as seen below. The patient was admitted to the cardiology servicewith continuous telemetry. During his hospital stay there were no runs of heart block observed.

Cardiology was consulted to assist with interrogation of the patient's Holter monitor, which was remarkable for 6 to 7 seconds of complete heart block as seen above. The patient was admitted to the cardiology servicewith continuous telemetry. During his hospital stay there were no runs of heart block observed.

The patient was offered multiple treatment options by the cardiology team, including a permanent pacemaker, implantable loop recorder, or external event monitor. The patient declined all interventions. The patient's atenolol was discontinued, and he was subsequently discharged home on losartan for hypertension. The patient has not followed up since discharge.

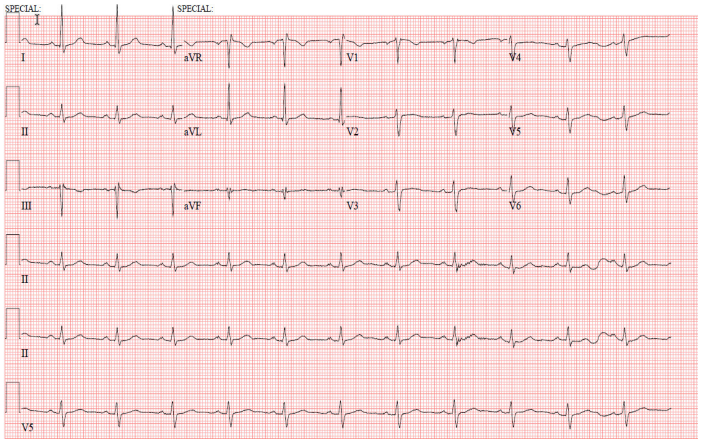

Image 7: The patient's initial normal EKG

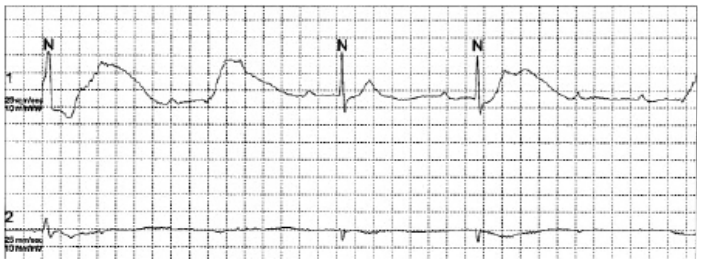

Image 8: The patient's Holter monitor demonstrating complete AV dissociation

Discussion

Heart block occurs when the electrical conduction from the atria to the ventricles is slowed or eliminated, resulting in abnormal conduction or bradycardia. Heart block is further stratified into first, second, or third degree blocks

First degree heart block results from slowed AV node conduction and is defined as a PR interval that is greater than 0.20 seconds. Second degree heart block results from inconsistent AV node conduction. Second degree heart block is further classified as type I (Wenckebach) or type II. In type I second degree block the PR interval progressively lengthens and is followed by a non-conducting P wave with a dropped QRS. Type II second degree block occurs when any number of atrial depolarizations fail to conduct through the AV node. This often occurs in a ratio (e.g. if every fourth P wave fails to conduct, the result would be four P waves for every three QRS complexes, or a 4:3 AV block). Unlike type I second degree heart block, the PR interval is constant in type II second degree heart block. Third degree heart block, also known as complete heart block, occurs when there is complete electrical dissociation between the atria and the ventricles at the level of the AV node.[1]

Heart block occurs after an insult to the cardiac conduction system from ischemia, infiltrative disease, infection, or drug toxicity. Myocardial infarction is a common cause of heart block in the emergency department. Emergency physicians should consider acute coronary syndrome (ACS) and coronary artery disease for any patient presenting with heart block. The majority of the cardiac conduction system derives its blood supply from the right coronary artery, and it naturally follows that heart block is classically associated with inferior and right-sided myocardial infarction.[2]

Heart block may also occur as a result of physical stress on the conduction system secondary to cardiomyopathies. Patients with dilated cardiomyopathy are prone to heart block as the conduction system is stretched and deteriorates. Infiltrative processes such as cardiac amyloidosis and sarcoidosis cause direct injury to the electrical conduction system and can lead to heart block and other arrhythmias.[3] Electrolyte abnormalities such as hypomagnesemia and hypokalemia affect action potential generation in the sinus node and are a common reversible cause of conduction abnormalities.

Infection may lead to heart block either by direct inflammation of the heart and conduction system or by provoking an immune response targeting cardiac conduction tissue. Viruses such as parvovirus B19, enterovirus, and HIV are the most common infectious agents responsible for acute myocarditis. Bacterial causes include Staphylococcus species, Streptococcus species, diphtheria, Lyme disease, and Rickettsia. Any of these infectious agents can damage the cardiac conduction system and lead to heart block. Chagas Disease is a notable cause of heart block, especially in patients with a native or travel history to Central and South America.

Multiple drugs have been associated with heart block, both at therapeutic doses and in overdose, and the mechanism of action is typically through AV nodal block. Common agents include beta blockers, calcium channel blockers, digoxin, clonidine, dexmedetomidine, pentamidine, and fingolimod. Inhalants contain hydrocarbons which confer both abusive potential and cardiotoxicity, and have been associated with heart block.[4] Products such as paint thinner, solvents, glue, refrigerants, and propellants are commonly abused.[5]

The treatment of third degree heart block depends on the patient's hemodynamics. Patients who are hemodynamically stable do not require emergent intervention, but are at risk for decompensation and need to be admitted for a broad diagnostic workup. At minimum, these patients should be admitted to the telemetry unit. It is also not unreasonable to discuss admission to an intensive care unit if the patient has significant cardiac comorbidities (e.g. severe cardiomyopathy, multi vessel coronary artery disease, or heart failure with significantly reduced ejection fraction).

The American Heart Associated recommends that providers manage unstable patients according to standard ACLS protocols. Atropine is first line therapy and should be dosed at 0.5 mg every 3-5 minutes up to a maximum dose of 3 mg.[6] Unfortunately, most patients in complete heart block will not respond to atropine because the ventricles lack the parasympathetic innervation that occurs higher in the conduction system. If the patient's heart rate does not respond to atropine, transcutaneous pacing should be initiated quickly. If transcutaneous pacing fails to obtain capture, transvenous pacing should be initiated in the ED.[1] Emergency physicians can also use medications with cardiac sympathetic activity, such as epinephrine, in refractory cases.

Adjunctive therapies may be of benefit in cases where reversible causes of heart block are thought to be the underlying cause. High-dose insulin therapy can be used in beta-blocker and calcium-channel overdose, in addition to vasopressors with beta agonist activity. Digoxin-specific antibody should be administered to all patients with digoxin toxicity and subsequent heart block. Broad spectrum antibiotics are indicated in cases of suspected infectious myocarditis. If reversible causes have been excluded, most patients will require definitive treatment with an implantable cardiac pacemaker.[7] Patients with heart block secondary to ischemic heart disease are the most likely to benefit from pacemaker placement.[6]

Third degree heart block is an uncommon but life threatening condition. Emergency physicians must be able to quickly identify this rhythm and aggressively manage these patients. Most patients who are hemodynamically stable can be closely monitored and treated with urgent placement of a permanent pacemaker. In patients with hemodynamic compromise, emergent transcutaneous or transvenous pacing is indicated and these patients should be admitted to the cardiovascular intensive care unit.

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been found to be superior to placebo for treating acute attacks in a number of studies. The largest of these was a randomized, double-blinded, placebo-controlled study of 125 patients. The primary outcome was time from administration to onset of symptom relief. Symptom relief occurred within 30 minutes in the treatment group versus 1.5 hours in the placebo group.[12] In a 2001 case series of 95 patients with known HAE, 193 episodes of laryngeal edema were treated with C1INH. Clinical response was documented within 30-60 minutes of receiving the treatment in 192 of these cases.[13]

When C1INH was originally approved in the US, the inhibitor was pooled from donated plasma, coalesced, and administered as a collective dose. Recombinant C1INH became available in the US in 2014 and has demonstrated similar efficacy when compared to plasma-derived C1INH. The recombinant form minimizes potential transmission of blood borne infectious vectors.14 One minor drawback is that the half-life of recombinant C1INH is only 3 hours compared to over 20 hours in plasma-derived C1INH. Despite this, there have been no associated relapses of symptoms with recombinant treatment compared to plasma-derived C1INH. No head-to-head trials exist to suggest that one option outperforms the other, although the recombinant drug avoids the issues associated with administration of plasma-based products.

Bradykinin receptor antagonist

The bradykinin receptor can also be targeted by pharmacotherapy in acute HAE attacks. Icatibant is a synthetic peptide that blocks bradykinin from binding to its receptor. This medication is administered as a subcutaneous injection of 30 milligrams. Several multi-center, randomized, placebo-controlled trials have examined icatibant. These studies were published in succession known as the FAST (For Angioedema Subcutaneous Treatment) series. FAST-1 compared icatibant to placebo and showed no difference between the two groups. FAST-2 compared icatibant and tranexamicacid, and showed that icatibant significantly reduced time to initial onset of symptom relief.15 FAST-3 attempted to settle the score by randomizing patients with moderate to severe cutaneous or abdominal symptoms to icatibant or placebo. Patients who received icatibant had quicker onset of symptom relief and reached 50% symptom reduction sooner than the placebo group.[16] Adverse effects were minimal and included injection site pain, nausea, dizziness, and headache. It is important to note that patients with known ischemic heart disease were excluded from these trials because icatibant has been observed to reduce coronary blood flow in animal models. Based on the most recent evidence, icatibant should be administered if available to help reduce symptoms, in addition to C1INH.

Kallikrein inhibitor

Ecallantide is a recombinant protein approved by the FDA in 2009 for treatment of acute HAE exacerbations. This molecule interferes with production of bradykinin via inhibition of kallikrein. Decreased bradykinin decreases edema by preventing migration of fluid into surrounding tissue. Ecallantide is administered intramuscularly in a single dose of 30 milligrams. In the 2010 EDEMA3 trial, patients receiving ecallantide reported significant improvement of their symptoms at a higher rate than patients in the placebo arm after 4 hours.[17] Ecallantide can be re-dosed within one hour of the initial injection in the case of suboptimal symptom reduction.

Plasma

The therapies discussed above are expensive and may not be immediately available in all emergency departments. Plasma has long been utilized to manage acute presentations of HAE. Assuming the donors are not affected by HAE, plasma will contain normal C1INH and improve symptoms. Fresh frozen plasma (FFP) is most commonly available in the ED. No controlled trials have demonstrated FFP's efficacy despite the fact that FFP had been the only treatment for acute HAE for years in the United States. A review of 12 case reports found that patients who received plasma all experienced some relief, although the response was highly variable.[18] Providers can initially transfuse one to three units of FFP and repeat this every four hours if improvement is not observed. With the advent of targeted therapies, the risks of blood product transfusion such as transfusion related acute lung injury (TRALI), transfusion associated cardiac overload (TACO), and infection, coupled with the lack of quality data demonstrating efficacy make FFP a second line agent in management of acute attacks.[1]

Angioedema Stage	Structures Affected	% Requiring Airway Intervention
I	Face, Lip	0
II	Soft Palate	0
III	Tongue	7
IV	Larynx	24

Table 3: Stages of hereditary angioedema[21]

While there are several agents available to treat acute HAE, no single treatment has been proven to be clearly superior. One review article attempted to quantify differences between treatment options specifically in laryngeal attacks.[19] Of the 12 eligible studies included in the review, plasma-derived C1INH conferred the shortest time to onset of symptom relief, followed by icatibant, ecallantide, and recombinant C1INH. The authors did note that the heterogeneity of the studies included limited their conclusions and no recommendation could be given based on this data. The latest guidelines from the World Allergy Organization strongly recommend administration of plasma-derived C1INH, ecallantide, or icatibant for any acute HAE attack.[20] If these therapies are not readily available, then plasma can be considered.

These medications are unlikely to be readily available in most EDs in the United States. Most of these medications require reconstitution and often need to be transported from a central pharmacy. As such, coordination with a dedicated emergency pharmacist or with the central pharmacy early in the patient's course is essential.

Disposition

Many emergency physicians may feel uncomfortable determining

a safe disposition for non-intubated HAE patients, and with good reason. Patients who do not initially require intubation still need high level monitoring in an airway capable unit.1 One otolaryngology study sought to create a risk stratification tool based on airway assessment that can help determine appropriate disposition for patients with angioedema.[21] These recommendations are based on a retrospective review of HAE patients over a ten-year period. Patients are classified in different stages depending on their physical exam findings. Stage I patients have a facial rash, lip swelling, or other facial edema. Soft palate edema placed patients in stage II while lingual swelling constitutes stage III. Finally, stage IV patients demonstrated laryngeal edema. Based on their findings, patients in stage I or II can likely be managed in an outpatient or floor setting. Stage III and IV patients should strongly be considered for ICU admission as airway intervention was required in 7% of stage III and 24% of stage IV patients.[21]

Early consultation with an allergist or immunologist can help facilitate administration of the appropriate treatments, establish a follow-up plan, and allow physicians to feel comfortable discharging otherwise well patients. In summary, HAE is a rare hereditary disease that can present with a wide variety of patient complaints. While uncommon, HAE can be life-threatening, and emergency providers should recognize when airway intervention may be required and be familiar with the available treatment options.

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Human Trafficking Continued from page 3

ing include vaginal or perineal injury, recurrent sexually transmitted infections, unintended pregnancy, lack of prenatal care, and unsafe or forced abortion. Untreated chronic disease and mental health issues are also common. HT victims have exceedingly high rates of substance abuse, suicidal ideation, suicide attempts, and self-injurious behavior. Patients may present with sequelae of these behaviors rather than from injuries or illnesses directly related to trafficking. Another common finding, particularly for victims of sex trafficking, are tattoos or branding. The most common tattoos are barcodes and words like "property of..." or "daddy."[8,14,15]

Exam findings include those typical of the injury patterns noted above. Additionally, victims may be malnourished, disheveled, dressed inappropriately, or present in the end stages of chronic disease processes such as HIV or other sexually transmitted diseases.[14]

Given the wide array of chief complaints and physical exam findings that may accompany HT victims, and the difficulty in correctly identifying those being abused, the American College of Emergency Physicians (ACEP) has issued a policy guideline on human trafficking. ACEP recommends that emergency clinicians be familiar with potential signs, symptoms, and indicators of human trafficking. Providers must maintain a high index of suspicion when evaluating patients who appear to be at risk for abuse and violence. Providers should assess for indicators of trafficking with a culturally sensitive and patient-centered approach. The policy encourages the creation of protocols to assure the medical, psychological, safety, and legal needs of these patients are met, and that providers familiarize themselves and receive regular training on these protocols.[16]

Additional Resources and Reading

- National Human Trafficking Resource Center Hotline (1-888-373-7888 or text "HELP" to 233-733)
- The Polaris Project - https://polarisproject.org
- Safe Horizon Anti-Trafficking Program and Hotline (1-800-621-HOPE)
- US Immigration and Customs Enforcement (1-866-872-4973)
- Hope for Justice - http://hopeforjustice.org
- ACEP Human Trafficking Policy - https://www.acep.org/Clinical---Practice-Management/Human-Trafficking
- UNODC: What is Human Trafficking? - https://www.unodc.org

Local Resources - Ohio and Cincinnati

- End Slavery Cincinnati - http://www.endslaverycincinnati.org
- Survivors Ink - http://www.survivorsink.org

With the help of the entire ED staff, a patient-centered approach can be implemented to better treat victims of HT. Providers should actively seek to minimize retraumatization and foster physical, psychological, and emotional safety. Examples of this include ensuring appropriate verbal consent, the presence of chaperones before examinations of sensitive areas, and assisting patients to disrobe in a sensitive way to minimize retraumatization. Other important considerations include providing certified interpreters and/or interpreting services for non-English speaking patients and not relying on family members or friends to interpret.[8,14]

There is currently no externally validated screening tool for identifying persons at risk for HT to be specifically used in the emergency medical setting.[17] The Vera Institute of Justice developed an evidence-based screening tool that was validated in 2014. However, this tool is cumbersome, recommended for providers to use after rapport is established, and is not appropriate for use in the ED.[8] However, multiple proposed screening tools and lists of "red flag" indicators exist. One particular study implemented a HT screening tool and treatment algorithm at a level 2 trauma center in southwestern Pennsylvania. This screening tool included

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Human Trafficking
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social screening by registration, medical screening by ED clinical staff, and a silent notification tool. Signs in the restroom instructed potential victims to identify their urine specimen cup using a blue dot sticker thus triggering additional investigation by the ED nurse. 38 patients were identified in five months: 20 via medical red flags and 18 via the silent notification system. All were offered intervention and five received help, including four adults who accepted assistance and one minor who received mandatory intervention as per state child abuse laws. After the intervention, staff members were surveyed. 97% of participants were committed to change their practice and most responded that receiving this education improved their perception of the problem.[18]

Currently in Cincinnati, a local task force comprised of social workers, forensic nurses, law enforcement, and a local hospital are working to develop and validate protocols both for EMS and emergency nurses. They have developed a tool for the electronic medical record used by nurses during the registration process that includes evaluation of visual and behavioral cues. A certain score on these cues prompts a second list of red flag indicators, similar to those provided (Table 1, page 3).[15]

Determining an appropriate disposition for these patients is often quite challenging. It is important to become familiar with available local resources and to involve social work, if available, early in the care of these patients. Reporting is mandatory for patients under the age of 18 and should be completed via the state's child protective service agency.[11] With adult patients who do not appear to be in imminent danger, providers should inform the patient about their concerns before reporting a crime. Providers often lack the resources to protect patients outside of their clinical venue and reporting the patient's trafficker may place the patient in imminent danger.[11,19] In some instances the appropriate intervention may be to disposition the patient to a shelter or protected setting. For others, the safest response may be to offer the patient educational and self-help resources prior to discharge.[14]

Human trafficking is a global and domestic problem that is vastly underestimated and often goes unrecognized in the health care setting. Emergency physicians are in a unique position to recognize and intervene early in these cases. The key to making a difference in this epidemic is having a high index of suspicion. Evidence-based protocols are currently being created, and screening tools are currently available, but the key to recognizing HT is to consider this as part of the differential. As part of a patient-centered approach to providing care, concerns about HT by any ED staff member should be addressed prior to disposition. By heightening awareness among staff and having protocols available to help victims of HT, emergency physicians can provide compassionate and life-changing care to this extremely vulnerable and under-represented patient population.

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Providers should then begin a focused neurologic assessment. This is especially important with patients that have sustained additional traumatic injuries from motor vehicle accidents or blast injuries. Confusion and altered mental status may also be secondary to carbon monoxide (CO) or hydrogen cyanide (CN) inhalation.

Carbon monoxide poisoning is a well-known sequelae of smoke inhalation injury. Adult hemoglobin has a much higher affinity for CO than oxygen, causing a relative hypoxemia. Symptoms of CO poisoning include flushing of the skin, headache, vomiting, and altered

Burns Managment:
Continued from page 9

mental status. All patients with suspected CO exposure should receive 100% oxygen and should be evaluated for hyperbaric oxygen therapy. Indications for hyperbaric oxygen therapy include carboxyhemoglobin levels >25% for adults (>15% in pregnant women), altered mental status, coma, seizure, or other focal neurological deficits.[3]

Hydrogen cyanide is formed by the combustion of nitrogen-containing polymers found in upholstery and insulation such as wool, silk, polyurethane, and vinyl. CN uncouples mitochondrial oxidative phosphorylation and causes profound tissue hypoxia. CN toxicity presents with dyspnea, loss of consciousness, seizures, altered mental status, and hemodynamic instability.[3] Hydroxocobalamin (Cyanokit) is carried by Air Care and is the antidote for cyanide toxicity. It is a natural form of Vitamin B12 and binds CN, forming cyanocobalamin which is excreted renally. Providers should administer this to any patient with an enclosed space inhalational injury and altered mental status,

Relative percentages of areas affected by growth (age in years)

	0	1	5	10	15	Adult
A: half of head	9.5	8.5	6.5	5.5	4.5	3.5
B: half of thigh	2.75	3.25	4	4.25	4.5	4.75
C: half of calf	2.5	2.5	2.75	3	3.25	3.5

Table 4: Comparison of TBSA burned in pediatric and adult patients

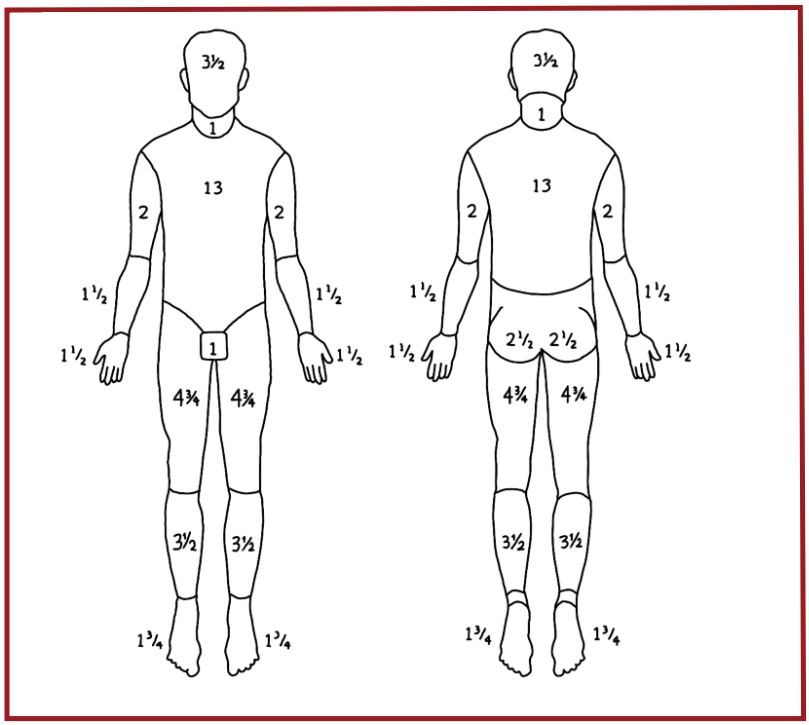

Figure 2: Lund Brower Diagram for estimation of burn size

hemodynamic instability, or unexplained lactic acidosis.

Before packaging the patient for transport, providers should remove all burning, hot, or singed clothing. Rings, watches, jewelry, and belts should also be removed to prevent a tourniquet-like effect.[3] The extent and degree of the burned skin should also be assessed prior to transport.

Burns are classified based on their depth. Superficial burns affect the epidermis and cause erythema that is painful without blistering, similar to a sunburn. Partial thickness burns affect the dermis. They are split into superficial partial thickness and deep partial thickness burns. Deep partial thickness burns do not blanch as opposed to superficial partial thickness burns. Second degree burns are very painful and cause blistering. Full-thickness burns affect the entire dermis; the skin is insensate and appears charred and leathery. Full thickness burns can affect deeper structures including fat, muscle, and bone.[3]

Providers should attempt to estimate the total body surface area affected by second and third degree burns. Various methods exist to help with this calculation. The "rule-of-nines" is the most common in the prehospital environment. This calculation needs to be adjusted in infants and children due to their proportionally larger heads and smaller legs. It is important to remember that the "rule-of-nines" often overestimates the total area of burned skin. Another method is to use the dorsal surface of the patient's hand as 1% to estimate the total extent of burns on the body. Finally, a Lund-Browder diagram may be filled out and is what is typically used by burn surgeons. This has been shown to be very precise and adjusts for age, and the figure above shows how to calculate this.[3]

Burned patients have very high insensible fluid losses and often require significant crystalloid resuscitation. The Parkland formula is the most common method used for calculating fluid requirements in burned patients. The amount of crystalloid required in the first 24 hours can be calculated using the formula below:

TBSA x Kg x 4 = Volume (mL)

Half of this volume is given over the first 8 hours and the second half is given over the subsequent 16 hours. Balanced isotonic solutions, such as lactated ringers or normosol, should be used.

While these patients often require very large volumes of crystalloid, they can easily be over-resuscitated. In the year 2000, Pruitt first described "fluid creep," which was coined to demonstrate the harmful side effects of over-resuscitation with crystalloid.[12] Burned patients who received too much crystalloid can develop abdominal compartment syndrome and pulmonary edema.[12] Over resuscitation and "fluid creep" continue to occur as demonstrated by several published retrospective cohort studies.[13]

The Parkland formula should be used in prehospital burn management to estimate fluid requirements and serve as a starting point. Providers should then use hemodynamic response and urine output as resuscitation guides, and hourly fluid rates can be adjusted as needed. The goal urine output should be 0.5 - 1.0 mL/kg/hr in adults with normal baseline renal function. Critical care transport providers should initiate fluid resuscitation but should be judicious with fluid boluses to prevent complications from over-resuscitation.

Severely burned patients can be intimidating for even the most seasoned critical care transport providers. These patients often require aggressive resuscitation and multiple procedures in a relatively short period of time. It is often easy for providers to become overwhelmed, necessitating an algorithmic approach to the patient, similar to traumatically injured patients. By advancing through the primary survey and stabilizing the patient while starting aggressive but goal directed crystalloid resuscitation, critical care transport providers can bring ICU level care to one of the sickest pre-hospital patient populations.

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EKG focus

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History of Present Illness

A 55 year old man presented to the emergency department with a chief complaint of chest pain. The patient had an extensive history of cardiovascular disease, including prior myocardial infarction necessitating percutaneous coronary intervention. The emergency physician initiated an evaluation for possible acute coronary syndrome. The patient's electrocardiogram demonstrated what appears to be a 1 mm ST-elevation in lead aVR with diffuse ST depression in the lateral and inferior leads that appeared new from prior studies. When asked if she would like to activate the interventional cardiology team, the physician hesitated - does ST-elevation in aVR indicate true acute myocardial infarction?

ST-Elevation in aVR

Lead aVR is one of the augmented (hence the "a" in its title) unipolar leads. Lead aVR's axis evaluates the heart cavity from the right shoulder, intersecting with the right ventricular outflow tract and basal aspect of the septum. Prior studies have shown that ST-elevations in aVR, with or without concomitant changes in other anatomically contiguous leads, may be considered a form of STEMI equivalent suggestive of large vessel occlusion. One such study found that ST segemen elevation in lead aVR was 81% sensitive and 80% specific for a left main coronary artery (LMCA) occlusion. A separate study in 2005 identified that ST-elevation in aVR ≥ 0.5 mm might serve as a usual predictor for significant three vessel disease.

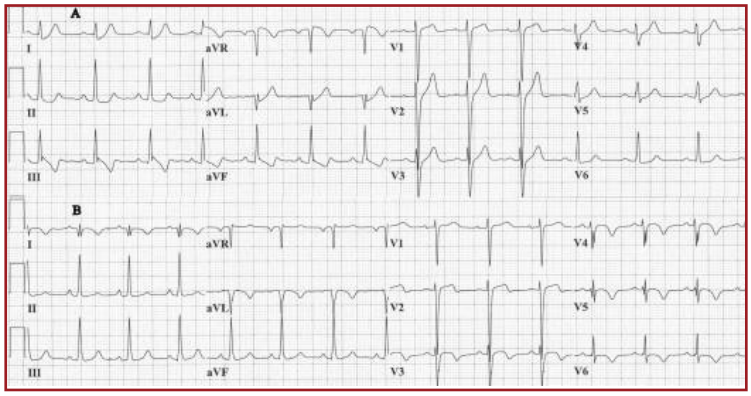

Figure 1. EKG demonstrating ST-elevation in lead aVR and ST-depressions in the inferior and lateral leads

ST-Elevation Criteria

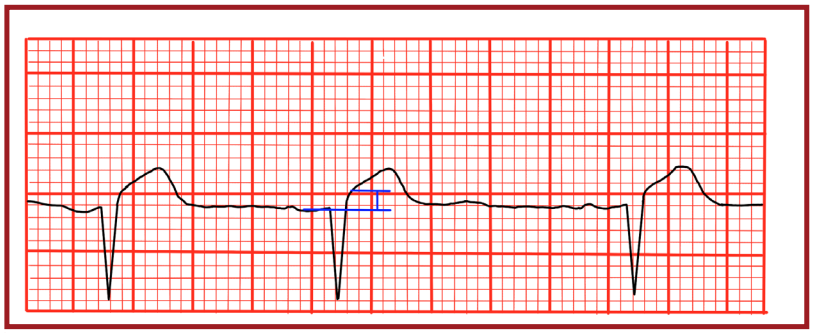

Figure 2. Lead aVR demonstrating > 1 mm ST-elevation

Subsequent studies and expert consensus, however, suggest that aVR does not possess adequate discriminatory power in isolation to determine LMCA/LAD occlusion. Rather, evidence-based practice propones for the use of aVR in conjunction with the patient's clinical context, serum biomarkers, and dynamic EKG changes to discern concerning cardiovascular pathology. Furthermore, ST changes in aVR should prompt emergency providers in an appropriate clinical scenario to consider other alternative diagnoses, such as pulmonary embolism or tricyclic antidepressant overdose, which may present with similar morphologic abnormalities.

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Please submit cases via EPIC In Basket message to Dr. David Habib. Make sure to include the R1/R4 involved in the case.

Submitted B Pod Cases

Case	Providers
3rd Degree Heart Block	Shewakramani
Bladder Rupture	Banning/Palmer
Wolff-Parkinson-White	Burkart/Leenellett
2nd Degree Heart Block	Burkart/Benoit
Ocular Syphilis	Barengo/Ronan
Blepharoconjunctivitis with Trichiasis	Bainbridge/Toth
Testicular Torsion	Harris/Fermann
Subarachnoid Hemorrhage	Harty/Moellman
Neurogenic Shock	Harty/Lafollette
Diverticulitis with Incarcerated Hernia	Kies/Paulsen