TXA in Pre-Hospital Care
Howard Mell MD

▶ International Trauma Life Support (ITLS) recently came out with a position statement on the use of tranexamic acid (TXA) in the management of traumatic hemorrhage in the field. They concluded that, “ITLS believes that there is sufficient evidence to support the use of TXA in the management of traumatic hemorrhage in the adult patient, pursuant to system medical control approval. Following initial resuscitation including control of external bleeding and stabilization of airway, consideration should be given to administration of TXA during early stages of transport.” [https://www.itrauma.org/wp-content/uploads/2014/05/TXA-Resource-Document-FINAL.pdf]

▶ The use of TXA in the prehospital setting is an emerging trend in the United States. Mell is the medical director for the Fire Department in Newark, Ohio, which is the first department to place TXA on their ground ambulances. They have accumulated a year and half of experience, and the initial impression is that it helps to control hemorrhage in severely injured patients. Average transport time to their receiving hospital is around 45 minutes.

▶ There have been three major studies.


• There are multiple papers stemming from this study. It was a randomized, placebo-controlled, double blinded study with over 20,000 patients. It was carried out in Western Europe, sub-Saharan Africa and western India, which has led to some questions regarding its generalizability. Some critics point out that there were no defined inclusion criteria. The gestalt of the treating physician led to enrollment in the study. If the physician felt that the patient had a significant trauma, the patient was enrolled.

• While the two study arms were randomized appropriately and looked similar statistically, would the same care be given in the United States?

▶ Some research looking at subgroups indicates that the drug is most effective if it is given within one hour of injury. If the drug is given more than 3 hours after injury, it may lead to harm.

▶ Realistically, if you are involved in a serious injury in most of the United States, you won’t have reached the trauma center and will be under the care of EMS or an outlying hospital that will arrange transfer to a trauma center. Some of the centers in developing nations are similar to the EMS setting, with minimal diagnostic equipment.

▶ The study showed a 1.5% reduction in all-cause mortality at 28 days without significant adverse effects.

▶ The number needed to treat was 67. The cost to administer a single dose is $107. The cost to save a life is about $7000.

▶ The dosing scheme was 1 gram over 10 minutes, followed by a second gram over eight hours.


• This was an observational trial but suggested that the number needed to treat fell significantly in more severely injured patients. The group receiving TXA was more severely injured but had improved mortality rates. In the most severely injured patients, the number needed to treat fell as low as 7.


• This was a randomized, placebo-controlled trial that looked at cryoprecipitate, tranexamic acid, both, or placebo. The best outcomes were with cryoprecipitate and tranexamic acid. However, tranexamic acid alone was independently associated with markedly decreased mortality.

▶ The CRASH3 trial is in protocol phase with an anticipated publication date of 2018.

▶ The PATCH trial is happening in Australia and looks at the prehospital administration of tranexamic acid for control of hemorrhage.
How does tranexamic acid work? When we have an injury, we make a fibrin clot right away. However, as soon as we make the fibrin clot, we begin to make the factors to break it down. We release tPA, which binds with plasminogen to make plasmin, which breaks down fibrin clot. This presents two problems in trauma: 1) the clot is gone, allowing bleeding; and 2) if enough clots are broken down after massive trauma, the resulting products can lead to a coagulopathy that can worsen bleeding. The ideal drug is one that stabilizes the fibrin clot.

- TXA binds to a lysine-binding site on the plasminogen so that the plasmin created upon binding with tPA is inactive. This keeps the initial clot in place. It doesn’t cause a DVT or PE. It isn’t thrombogenic; it just stabilizes the fibrin clot.

The data shows it is most effective if it is given within the first hour after injury, placing it in the realm of EMS and outlying hospitals. The data shows that if the drug is given more than 3 hours after injury, it is not effective and can cause harm. It is also more effective when it is coupled with a blood component-based resuscitation effort. It has been included in a lot of massive blood transfusion protocols.

EMS medical directors may consider using tranexamic acid in the prehospital setting in patients with significant trauma and an anticipated time of over an hour before hospital evaluation. It may be worth adding it to the formulary in outlying hospitals and Level 3 trauma centers, who anticipate transport of patients for higher level of care, so that patients may receive TXA in a timely manner.

Dosing. If you are administering a drug over ten minutes, do you need pumps? The Newark Fire Department assembled kits, which contain tranexamic acid (which comes in 1 gram per 10mL of liquid), a 10mL syringe, a filter needle, an injection needle, a 50 cc bag of normal saline and some 10cc/mL drip tubing, and a bracelet that says “TXA given.” To give the drug over ten minutes, draw up the drug into the 10mL syringe and inject into the 50mL bag of normal saline. This gives you a bag containing 1 gram of tranexamic acid in 60mL of fluid. Hang the 10cc/mL tubing attached to the bag. Set the drip at 1 drip per second. There are 600 drips in 10 minutes. There is no need for a pump and you will administer it within close to ten minutes.

This is not to say that tranexamic acid is more effective than the tools available to trauma surgeons at major trauma centers. It is better than what is currently available when 45 minutes away from the nearest trauma center or in a free-standing ER with a walk-in trauma experiencing significant hemorrhage. It is beneficial when there is proper patient selection. It shouldn’t be given to everyone or when there are better alternatives available; it should be given when there is no better option.

Paper Chase 1:
The Dizzy Patient
Sanjay Arora MD and Michael Menchine MD


This is a Korean study that found that 3.6% of patients presenting to the Emergency Department with dizziness had a central lesion, most commonly a posterior infarct.

Dizziness is a common complaint. About 2.5% of all Emergency Department complaints are for dizziness. History is challenging. Patients often don’t want to cooperate with the neurologic exam. There are some studies suggesting that up to 20% of patients with isolated peripheral vertigo have acute strokes, although work-ups are frequently negative. CT scans are terrible at detecting posterior fossa lesions, especially strokes. The CT scan is approximately 16% sensitive for acute stroke in the posterior fossa.

The authors of this study sought to determine the presence of central lesions, as determined by MRI, in patients presenting to an Emergency Department with a complaint of dizziness. They also sought to determine if there were any clinical features able to distinguish between patients with and without central lesions.

This was a retrospective chart review of 902 patients, who presented to a single Emergency Department with a chief complaint of dizziness during a 6 month period. There were 645 patients who received an MRI. The other patients do not have good data available, but they appear to have been discharged without imaging.

The study looked at the 645 patients who received MRI. The average age of these patients was over 60 years of age. The authors found that 23 patients or 3.6% had a central lesion: 22 were an infarct and 1 was hemorrhage. The large majority were posterior fossa infarcts.

The authors then looked at clinical features associated with a central source of dizziness. They found advancing age had a higher likelihood of a central source: the rate was 3.9% and 3.5% in patients in their 50s and 60s; 7.4% in their 70s; and 16.7% in their 80s. Hypertension was more common in cases with a central cause: 69% versus 36%. Atrial fibrillation was more common with a central cause. 77% of patients with a central cause reported a more vague non-whirling dizziness compared to 40% in patients without central lesions. Other associated neurologic symptoms were present in about 46% of patients with a central cause, compared to only 3%.

Limitations. This was a retrospective chart review with fairly poor methods. They read the doctors’ note. There was no standardized approach and it is difficult to tell how robust these
The incidence of stroke in all-comers with dizziness is pretty low at 3.6% (and this is only in patients who received MRI). The incidence is probably less than 3% overall. This paper suggests the risk of stroke is low enough that most patients with dizziness do not need to be evaluated for a central cause. You should use clinical exam and potential risk factors to stratify for need for MRI. You should have a lower threshold for obtaining an MRI in older patients. Older patients are often unable to comply with neurologic testing, and it can be difficult to identify acute findings in an elderly patient with disequilibrium at baseline.

Pediatric Pearls: Resuscitation of the Sick Neonate – Part 1
Ilene Claudius MD and Sol Behar MD
Interview Courtney Nichols MD

• The neonatal resuscitation is slightly different than PALS. The main difference is a focus on pulmonary support. When a baby is born, the first thing performed is support of respirations. The algorithm is fairly simple.

• Ask three questions. Is this a term gestation baby? Is the baby crying and breathing? Is there good tone?
  • If the answer is yes to all three questions, you can put the baby on the mother’s belly and keep it warm. Blankets should be placed on top of the baby. The mother’s heartbeat can help regulate the baby’s heartbeat with skin-to-skin contact.
  • If the answer is no to any of these questions, you need to start supporting the baby.

• During the first 30 seconds, assess the baby. Keep the baby warm. Clear the airway with bulb suction if possible. Dry and stimulate the baby. Check the heart rate at 30 seconds. If the baby is gasping or has apnea, start positive pressure ventilation. Establishing pulse oximetry at this time is now recommended.

• What do you do if there is meconium aspiration? If you are going to perform tracheal suction, it is best if it is done right away. If the baby is born with meconium, the baby’s respiratory status is first evaluated. If the baby is vigorous despite the meconium: bulb suction, dry, and stimulate as usual. If the baby has poor tone and poor respiratory effort, intubate and suction the trachea for meconium. The idea is to remove some of the meconium before the baby starts breathing spontaneously, to decrease the pneumonitis caused by the meconium.

• How is this performed? An endotracheal tube is placed and a meconium aspirator is used to suction through the tube as the tube is removed. If the baby has improved tone and respiration, the baby can be dried and stimulated as usual. If the baby persists with poor respiratory effort and decreased tone, the baby can be intubated again. The decision is made to suction again or, if the heart rate is dropping, to leave the tube in place and start providing positive pressure ventilation.

• If you are more than a few minutes out, the meconium has likely done its damage and the decision becomes whether or not you need to intubate the baby for respiratory support.

• For babies without meconium who need positive pressure ventilation, you need to suction the nose and mouth, such as with the bulb suction, prior to initiating positive pressure ventilation. Nasal cannula, such as the RAM nasal cannula, or bag-valve can be used for positive pressure ventilation. Give this for thirty seconds, prior to providing cardiovascular support with chest compressions. Very commonly with these infants, positive pressure ventilation is sufficient to improve the patient.

• The RAM device is basically a nasal cannula that can provide positive pressure. An appropriately sized cannula is placed and the baby’s mouth is closed. Positive pressure ventilation can be provided through a T-piece or flow-inflated bag. Ventilation with room air or an oxygen percentage a little bit higher than room air is recommended. Any baby that requires significant respiratory support, such as intubation or chest compressions, can be given 100% FiO2.

• What is a normal pulse ox in a normal neonate? At one minute of life, the baby’s oxygen saturation is between 60-65%; at two minutes of life, 65-70%; and at three minutes of life ,70-75%. The oxygen saturation is approximately 80-85% at five minutes. It isn’t until 10 minutes of life that the baby’s oxygen saturation reaches 85-90%.

• The rate of ventilation with PPV is about 40-60 beats per minutes. “One and two and breath. One and two and breath.”

• If you do need to start chest compressions, you want to do it at a rate of 90 beats per minute. NRP recommends coordination of compressions, regardless of whether or not there is an advanced airway in place. Resuscitation is 120 events per minute: 90 compressions and 30 breaths. “One and two and three and breath. One and two and three and breath.” Breaths are delivered more slowly if the patient is receiving simultaneous chest compressions.

• When do you start chest compressions? After suctioning and stimulating the baby for thirty seconds and providing positive pressure ventilation for thirty seconds, if the baby has
a heart rate less than a 100, you need to take a corrective step. This can be adjusting the mask, repositioning of the airway, suctioning, opening the mouth, increasing the pressure, or giving an alternate airway. If the heartbeat goes below 60 beats per minute, you need to consider intubation and administer chest compressions with coordinated respirations. Intubation medications are not typically given, as there is little room before the oxygenation drops. Atropine is not part of the NRP recommendations. The only medication in the algorithm is epinephrine if the heart rate continues below 60.

- How long do you continue with ventilation and chest compressions before deciding to give epinephrine? When you initiate chest compressions, someone should be attempting to obtain venous access, either via an umbilical venous line or peripheral IV. When access is obtained, you should consider epinephrine if the heart rate is not improving. Epinephrine can be given every 3 to 5 minutes. If you are unable to obtain IV access, it can be given through the ET tube. The dose of epinephrine (1:10,000) is 0.5-1.0mL/kg for endotracheal administration. 0.1-0.3mL/kg if using intravenous access.

- The NRP recommends that if you have a neonate in asystole and you are unable to achieve a response after 10 minutes of resuscitation, it is time to terminate the resuscitation. What do you do if the patient persists with a heart rate of 30 despite your interventions? There are no great answers for how long to continue the resuscitation. You can get the parents involved.

- What are indications for complete non-resuscitation? The target for resuscitation of preterm babies depends on the institution. Some will use a gestational age of 24 weeks. In the 23-24 week range, they have a discussion with the parents. Less than 23 weeks is considered not viable and is not resuscitated. Weight of about 600 grams usually correlates with dates of 24 weeks.

- Babies are placed in plastic coverings for warming if they are doing well. Do you need to cool a neonate who isn’t doing well? It depends on how far you are from a cooling center. Patients who are candidates for therapeutic hypothermia include: gestational age >36 weeks, physiologic criteria of pH < 7 or base deficit > 16 mmol/L, pH 7.01-7.15 or base deficit 10-15.9 mmol/L with an acute perinatal event, or APGAR scores < 5 or neurologic criteria of signs of encephalopathy or presence of seizures. Cooling should be initiated in the first 6 hours of life, so it may be needed in Emergency Departments transferring patients.

- How can you initiate cooling in the emergency department? Turn off the warmer. No blankets. No hats. The goal temperature is 34-35º Celsius.


- Obtain cord blood gases and an ABG within the first hour of life. Follow blood glucose and calcium. Treat any seizure activity with phenobarbital.

- What fluids do you give a neonate? At birth, they don’t require much. Some start with D10W at 80ml/kg in the first day of life for maintenance fluids in a term neonate. Children of diabetic mothers and preterm neonates may require extra calcium. However, this can usually be started later in the first day of life.

- How do we give glucose in the neonate? 2cc/kg of D10W. Although blood glucose of 25mg/dl (1.4mmol/L) may be tolerated in the NICU, in the Emergency Department, we should consider giving glucose below 35-45mg/dl (1.9-2.5mmol/L).

- Maintenance fluids depend on the age of the neonate. There are no clear guidelines. In the NICU, patients will often receive 80cc/kg of D10W in the first day of life, 100cc/kg on day 2 with some sodium chloride, 120cc/kg on day 3, and 140cc/kg on day 4.

- When should a distressed neonate receive a blood transfusion? This depends on the clinical situation. Did the mother have a ruption? Are there any signs of fetal blood loss? The hemoglobin levels expected in a neonate is a range over gestational age and is higher in preterm infants.

- How much fluid should you bolus the neonate? 10cc/kg of normal saline is reasonable.
wheeling the patient out, the nurse had Hochman come up to the cath lab with them.

- **They arrived at the cath lab.** She was now more short of breath and tachypneic. She had rales throughout. Hochman called for the crash cart and intubated the patient. During the intubation, the patient became slightly hypoxic. She then became bradycardic and arrested. Hochman did CPR. The patient was placed on the cardiac monitor and was found to be in ventricular fibrillation. She was defibrillated once. She then entered a PEA rhythm and received two ampules of epinephrine, with return of pulses. The interventional cardiologists took over the case.

- **On catheterization, the patient had normal coronary arteries.** An echocardiogram showed decreased left ventricular end-diastolic volume and decreased contractility throughout the left ventricle, with severe hypokinesis in the apical region of the left ventricle. The patient had an ejection fraction of about 25%.

- **A balloon pump was placed and the patient was admitted to the CCU.** The balloon pump was discontinued a day later. She was extubated 3 days after that. A repeat echocardiogram showed improved ejection fraction to 45%. She was discharged on day 12.

- **The patient had Takotsubo cardiomyopathy, also known as broken heart syndrome or stress cardiomyopathy.** In Takotsubo cardiomyopathy, the heart does not contract properly. Takotsubo cardiomyopathy is triggered by emotional or physical stress in about 85% of cases. This can be fear, grief, anger, relationship difficulties, or physical distress from other disease processes, such as asthma or stroke. Treatment, such as surgery or chemotherapy, can provoke it. Takotsubo can be caused by administration of exogenous catecholamines.

- **This was initially described in Japan in 1990.** The first reported case in the United States was in 1998. Since 2000, there has been an explosion of case reports.

### Case #2

A 61 year old female presented with substernal chest pressure radiating to the back. Prior to arrival in the ED, paramedics called and sent an EKG that showed ST elevations in the inferior leads. They were concerned for ST-elevation MI. The patient had taken a full dose of chewable aspirin at home. The paramedics administered nitroglycerin en route, with improvement in her pain. They elected not to call a Code STEMI until the patient arrived in the ED and was evaluated.

On arrival, the patient reported substernal chest pressure radiating to her back, which was almost gone with the nitrates. She was non-toxic appearing. Repeat EKG showed ST elevations in the inferior leads, with reciprocal T-wave inversions in lead AVL. At this point in time, the Code STEMI was activated. She had no past medical history and no past surgical history. She was a non-smoker, did not use cocaine, and had no family history. Her only cardiac risk factor was her age and post-menopausal status. Her physical exam was normal.

- **The nurse accompanied the patient to the cath lab, where she was greeted by the cardiology fellow, who was waiting for the interventional cardiologist. The cardiology fellow was not impressed by the patient’s EKG and clinical presentation. He wanted to send the patient back to the ED and cancel the cath.**

- **The Emergency Department nurse called Patel on the phone, and he discussed his discomfort with the plan with the cardiology fellow. They agreed to wait until the interventionalist arrived to decide whether or not to proceed with catheterization.** Fortunately, the interventionalist wanted to proceed with catheterization. The initial troponin was 2.8ng/mL. The second troponin was 4.6ng/mL, before trending down. The catheterization showed very mild non-obstructive coronary artery disease with a 30% mid-LAD stenosis. The patient was later diagnosed with Takotsubo cardiomyopathy. She had an ejection fraction of 40% with hypokinesis of the apex. The patient was admitted, treated medically, observed with improvement in ejection fraction, and discharged home without complications.

- **Nearly 90% of Takotsubo cases are in women, usually 50 years or older.** The symptoms mimic MI. The EKG may appear exactly like a STEMI. Cardiac enzymes often are elevated. The pathophysiology is not fully known but thought to be the result of physical or emotional stress. Current theory suggests that a surge of catecholamines causes damage to the heart muscle, decreasing contractility. It is generally a transient phenomenon. The heart typically returns to normal contractility within 1-2 weeks. The name Takotsubo comes from the Japanese word describing a ceramic octopus trap, which is similar to the appearance of the heart on echocardiogram. The treatment has been beta-blockers and ACE inhibitors, although it is questionable whether these provide any benefit. Anti-coagulation is sometimes indicated, as embolic or thrombotic events may occur. Intra-aortic balloon pumps may be used to support cardiac output. The normal treatments for pulmonary edema are used, if indicated and appropriate.

- **The prognosis is good.** The chance of recurrence is very low (less than 5%). Medications are not thought to prevent recurrence. There is no evidence of cumulative damage to the myocardium.

  - This included 136 patients, who were seen over about 10 years at a single center in Minnesota. All had acute cardiac
Physical contributors included exogenous beta-agonists, such as albuterol, dobutamine, or phenylephrine.

Nearly half of the patients had anterior ST elevations. Inferior ST elevations are less common. 92% had troponin elevation on admission.

Average ejection fraction on admission was 32%. All of the patients had follow-up echocardiograms, and almost 100% had return of function, with an ejection fraction of at least 50%.

There was very low mortality; only 3 of the 136 patients died. At two year follow-up, only 17 out of 136 had died, and none were from cardiac causes. Only 6% had recurrent events.


This article outlined conditions encountered in the Emergency Department that can mimic ST elevation, such as hyperkalemia, pericarditis, left bundle branch block, Prinzmetal’s angina, among others. Takotsubo’s cardiomyopathy did not make this list, but, as the Sharkey article indicates, ST-elevation is a fairly common presentation.


This case report described a 57 year old teacher with a history of hypertension, who presented with chest pain and ST elevations in the anterior leads. She was toxic on presentation with dyspnea and hypotension. Her troponin was elevated to 4.2ng/mL. She was diagnosed with Takotsubo cardiomyopathy and did surprisingly well.

Takotsubo cardiomyopathy patients may present with a wide spectrum of disease severity. However, the end prognosis is generally excellent.

Clean arteries on cardiac catheterization does not rule out an acute coronary event; patients may have sustained vasospasm or had a clot that broke up. Cardiologists may be able to determine Takotsubo cardiomyopathy as the etiology, based on the classic appearance of the left ventricle. This can’t be explained by a single vessel distribution.

Take-home points. Be careful with a crashing patient going up the cardiac cath lab. Takotsubo cardiomyopathy is not an uncommon entity. These patients look exactly like STEMI patients. Treat them as you would treat any other STEMI patient; you do not have time to get an echocardiogram. This is often seen in elderly females between 50 and 70 years of age, with an emotional stressor.

Pediatric Pearls: Resuscitation of the Sick Neonate – Part 2: Jaundice
Ilene Claudius MD and Sol Behar MD
Interview Courtney Nichols MD

The AAP has some great guidelines on when to start phototherapy and exchange transfusions in infants. You want to initiate phototherapy as quickly as possible. It can sometimes be faster to get the lights from upstairs than to transfer the baby up to the NICU. Children who are approaching the criteria for exchange transfusion will often get phototherapy first, and it may be possible to prevent exchange transfusion.

25 is the magic number; this is when it is an emergency for any baby and you want to start phototherapy as soon as possible. These infants have a higher risk of neurologic injury. A history of a bilirubin level over 25 qualifies these infants for high risk follow-up.

A 2 day old term infant presents with yellow skin. What do you do? There is a good reference on the internet; bilittool.org. You can input the child’s age in hours and serum bilirubin levels, and it will tell you recommendations for treatment and level of risk. You want to evaluate the child’s neurologic status, hydration status, and weight loss from birth.

When do you work up for other causes such as hemolytic anemia? Start with a total bilirubin and direct bilirubin, blood type and direct Coombs test. You can also test their CBC and reticulocyte count to see if there are signs of a hemolytic anemia. Children with hemolytic anemia may require closer monitoring. Also, if they are approaching criteria for exchange transfusion due to hemolytic anemia, IVIG may be beneficial.

Is there ever a reason to initiate an exchange transfusion in the emergency department? No. There are significant risks associated with this therapy. Focus on intense phototherapy and exchange transfusions in infants. You want to initiate phototherapy as quickly as possible. It can sometimes be faster to get the lights from upstairs than to transfer the baby up to the NICU. Children who are approaching the criteria for exchange transfusion will often get phototherapy first, and it may be possible to prevent exchange transfusion.

The babies should continue feeding. If they are unable to continue feeds, you can rehydrate them with IV fluids.

Breast feeding jaundice is usually seen in the first week of life. These babies tend to be dehydrated and are unable to excrete bilirubin, due to decreased feeding while waiting for the mother’s milk to come in.

Breast milk jaundice happens after the first several weeks of life. The beta-glucuronidase in the breast milk can
increase the bilirubin levels in infants. The levels don’t usually approach levels concerning for kernicterus. Mothers should continue to breastfeed. The levels can be monitored and will usually decrease over time. The yellow color of the skin and eyes can persist for several weeks.

- Distribution of jaundice is so variable that you can’t determine the level of bilirubin based on clinical exam. You should still test the serum. Also, after babies have received phototherapy, you can’t follow exam of their skin.

- Transcutaneous bilirubin is used in many newborn nurseries as it is non-invasive. It is less accurate at the extreme ranges. If you have a high transcutaneous value, you should check a serum level.

OSU Grand Rounds Case 1: Whole Body Weakness
Mike Weinstock MD, Kim Cunagin DO, Alex Fox MD, Shari Robbins MD, Victoria Lawson MD

Case #3

A 26 year old male presented to the Emergency Department complaining of arm and leg weakness, as well as whole body weakness or maybe numbness. He also complained of some sharp chest pains the previous day. He then complained of numbness to his leg and then his whole body.

- We have all had these patients. This patient was an extremely difficult historian. He had a new complaint with every question that he was asked. It was difficult to redirect him back to his original complaint, in order to ask him follow-up questions. Eventually, they determined that he was experiencing numbness that started in left leg, which went to his left arm and maybe to his right fingertip. The numbness had started the previous day, resolved, and then returned.

- On physical exam, his vital signs were all stable. His neurologic exam was completely normal, without subjective findings of paresthesias. No numbness or tingling.

- Work-up. He had laboratory testing, including a CBC and chemistry panel to check his electrolytes. CT scan of his head was normal. EKG was normal. He was asymptomatic. His exam was normal. They had a lengthy discussion about the findings and differential diagnoses of his symptoms. At this point, he was very frustrated that they had not identified a diagnosis and decided to go home.

- Could the patient walk? The initial nursing note reported that the patient ambulated with a limp but by the time of discharge, he was ambulating to and from the bathroom without difficulty.

- This is a difficult case. Given the completely normal findings, it is difficult to make a case to admit the patient for additional work-up. Most would have felt comfortable discharging him home.

- The patient returned and complained that they hadn’t done anything for him previously. The only unusual finding on exam was an absence of reflexes in his lower extremities. The patient was not able to walk and fell forward when they attempted to ambulate him. This was clearly different than his initial presentation. The patient had a flu shot two weeks prior.


- The patient reported that the weakness had progressed over about 24 hours. A lumbar puncture was performed and they noted elevated protein. The diagnosis was narrowed to Guillain-Barre Syndrome.

- It is not uncommon for patients with Guillain-Barre to have multiple visits prior to diagnosis. The symptoms are very non-specific. Patient may have very ill-defined paresthesias (hot feels cold, cold feels hot, they feel swollen, etc.). Neurologic exam may initially be normal. They may have a completely normal sensory exam. Patients may have a subjective feeling of weakness without loss of muscle strength on manual testing of muscle. Manual testing isolates each muscle, which is different than standing the patient up. Unlike other neuropathies, this is a patchy inflammatory attack and is proximal and distal. Guillain-Barre can mimic other neuromuscular issues.

*Editor’s Note: Classically, GBS is a progressive ascending symmetric muscle weakness starting in the legs, with absent or decreased deep tendon reflexes.

- How do you make the diagnosis? The necessary criteria are symmetric weakness, areflexia, and progression over time. You may have atypical variants, like the Miller-Fischer variant or sensory-only GBS. Areflexia may not be present early in the disease course. It can mimic a spinal cord problem or stroke due to asymmetry. Rapid progression is characteristic. You can consider observation if you aren’t sure what is going on. They may have autonomic dysfunction and this may be life-threatening.

- The confirmatory test is CSF analysis demonstrating albuminocytologic dissociation. Nerve-conduction study may also show changes.

- Other diagnoses to consider in this case. Spinal cord issues. Toxic neuropathies. This patient had exposure to IV drugs. Hexane has been associated with acute-onset, demyelinating neuropathy. An infectious polyradiculopathy.

- Treatment. IVIG and plasma exchange are equally efficacious. The sooner the treatment is given, the better the prognosis.
The LIN Sessions:
tPA in Pregnancy
Michelle Lin MD and Zlatan Coralic PharmD

---

**Case #4**

You work in a busy Emergency Department. You are notified that there is a stroke patient being transported by EMS. The patient arrives. It is a woman in her mid-thirties presenting with slurred speech. The only thing she can say is, “Help me.” She has obvious left-sided paralysis.

- **What can cause paralysis to one side of the body?** Todd’s paralysis after seizures. Trauma. If nothing else pans out, you need to think about stroke.
- **Would you activate a Code Stroke with the neurology team?** Yes. The neurology team arrived and agreed with the assessment of probable stroke. A CT scan was ordered. Then, they found out the patient was 10 weeks pregnant.
- **If this is an ischemic stroke, do you give tPA?**
- **Ischemic stroke is relatively rare in young patients.** However, in young patients with stroke, women are more likely to experience it than men. This correlates with childbearing age. The reported incidence ranges widely, from 5 in 200 to 5 in 100,000. Hormonal changes in pregnancy may affect the hemostatic cascade. This is usually more prominent in the third trimester. In general, pregnant women are slightly more hypercoagulable, which resolves about three weeks postpartum.
- **The risk factors for stroke in young patients are similar to those in older patients:** hypertension, diabetes, sickle cell and other blood disorders, and substance abuse. Risks factors associated with pregnancy include preeclampsia and eclampsia. Also, women who have a cesarean section are at increased risk of stroke in the peri- and postpartum period.
- **tPA in pregnancy:** Pregnancy is not an absolute contraindication to tPA. The NINDS 2 and the ECASS 3 trials don’t specifically mention pregnancy as a contraindication, although pregnant patients were not included in those trials. The American College of Emergency Physicians policy statement does not offer guidance on giving tPA to pregnant patients: “Exclusions (or cautions) to tPA use that were not specifically mentioned in either study but are generally used: … pregnancy and early postpartum period.”
  - The package insert for tPA refers to it as pregnancy Category C. High-dose tPA has been shown to have embryocidal effects in rabbits when intravenously administered. There was no maternal or fetal toxicity at 1mg/kg (the dose used for stroke). They recommend that tPA be used in pregnancy only if the potential benefit justifies the potential risk to the fetus.
  - You should look at your hospital protocol to see if pregnancy is listed as an absolute contraindication or a relative contraindication.
- **There is limited data available.** A review article published on the subject seemed to favor giving tPA to pregnant patients.
  - Arguments in favor of tPA: tPA has such a large molecular weight that it theoretically does not cross the placenta (they cited rabbit studies where it did not cause harm at the standard dosing of 1mg/kg).
  - Arguments against tPA: we don’t know the ultimate effect on the placenta, premature labor, or fetal demise. tPA can be a source of many medicolegal issues in the emergency room.
- A case series looking at the use of tPA for ischemic stroke, myocardial infarction, pulmonary embolism, and thrombosis of cardiac valve prosthesis found a similar complication rate to non-pregnant patients and low rates of maternal mortality, fetal loss, and preterm delivery.
  - A case series looking at the use of tPA for ischemic stroke, myocardial infarction, pulmonary embolism, and thrombosis of cardiac valve prosthesis found a similar complication rate to non-pregnant patients and low rates of maternal mortality, fetal loss, and preterm delivery.
  - It can be difficult to attribute harm: was fetal loss due to the low blood pressure associated with a massive pulmonary embolism or the tPA?
- **What would you do in a pregnant patient with a right MCA occlusion?** This has to be a case-specific, decision-making process. The patient and family should be included in the decision-making process.
- Dystonia can be a mimic of stroke.
Undifferentiated nausea and vomiting in the ED.

  - These were found to be equally efficacious for the treatment of undifferentiated nausea and vomiting.
- What do you do if you give a dose of ondansetron but the patient continues to experience nausea and vomiting? Some will repeat a dose one or two times. Others will switch agents in an attempt to target different receptors.
- Every time we give ondansetron for undifferentiated nausea and vomiting in the Emergency Department, we are using it off-label. Ondansetron was approved for prevention, not treatment, of post-operative nausea and vomiting, or chemotherapy-associated nausea and vomiting. We give it all the time.
- Should you give diphenhydramine if you are giving prochlorperazine?
  - Prochlorperazine is a commonly used medication in Emergency Medicine. It works well in some patients with migraines, and it is a great choice for patients with vomiting refractory to ondansetron.
  - Prochlorperazine has anti-dopaminergic activity, increasing the chances of extrapyramidal symptoms. The most common symptoms seen in the ED are: akathisia or restlessness, dystonia, Parkinsonism, and rarely tardive dyskinesia. Most of the literature on extrapyramidal symptoms associated with prochlorperazine is from the psychiatric literature, where it has been used for more than 70 years. There is some data for the single dose given in the Emergency Department.
  - What is the risk of extrapyramidal symptoms with prochlorperazine? A study, including 229 adult patients receiving intramuscular or intravenous prochlorperazine for nausea and vomiting or migraines in the Emergency Department, found that 11% developed akathisia in the Emergency Department and an additional 6% developed akathisia within two weeks after discharge. Overall, there was a 1 in 5 chance of an unpleasant reaction after prochlorperazine. Olsen, JC et al. Frequency of adverse reactions to prochlorperazine in the ED. Am J Emerg Med. 2000 Sep;18(5):609-11. PMID: 10999579.
- The definitions for akathisia could be debatable in these studies, as it is such a subjective finding.
- Overall, although prochlorperazine is effective for nausea and vomiting, patients may experience adverse reactions even with a one-time dose.
- What can you do to prevent this? A randomized controlled trial, including 100 patients, looked at whether or not giving Diphenhydramine 50mg decreased symptoms, and found an absolute reduction of akathisia in 22% of the diphenhydramine group. The number need to treat was about 1 in 5. However, this did have an increased risk of sedation. Vinson, DR et al. Diphenhydramine for the prevention of akathisia induced by prochlorperazine: a randomized, controlled trial. Ann Emerg Med. 2001 Feb;37(2):125-31. PMID: 11174228.
- We don’t know if giving a smaller dose of diphenhydramine or administering it orally will show benefit. We also don’t know if giving a smaller dose of prochlorperazine decreases risk of akathisia.
- These studies did have very strict inclusion criteria.

Cardiology Corner:
Endocarditis Update
Rob Orman MD and Amal Mattu MD

- 99% of patients presenting with a viral syndrome and lack of energy have nothing bad, but some have something far worse…
This can be a difficult diagnosis to make in the Emergency Department. Aside from a transthoracic echocardiogram (TTE), there is no standard diagnostic test and making the diagnosis depends on your index of clinical suspicion.

Endocarditis tends to present with a lot of non-specific signs and symptoms. Patients often will have a fever. They frequently will complain of muscle aches, especially in the large muscle groups. They are often not toxic and will complain of malaise. The cases we tend to miss are those without the classic risk factors, like IV drug abuse. A murmur is often not documented or heard and may be intermittent or not present. It is easy to misdiagnose these patients as influenza or viral syndrome. By the time the diagnosis is made, they may have suffered embolic complications.

When should we suspect endocarditis? About 80% of patients with endocarditis will have a fever. Take patients seriously when they say that they are having fevers at home, even if they are afebrile when you see them. Consider this diagnosis in patients with fevers and myalgias.

The worst-case scenario for patients with fever, myalgias, and a non-toxic presentation is endocarditis or spinal epidural abscess. These two diseases are often misdiagnosed as influenza or viral syndrome. Spinal epidural abscess is an increasingly litigated missed diagnosis. On average, patients have 2 to 3 visits before a correct diagnosis is made.

What do you need to do if you are considering endocarditis as a diagnosis?
- Listen very carefully for a new murmur (present in 50%). Look for hematuria. Is there splenomegaly on the abdominal exam? Look for splinter hemorrhages in the fingernails, as well as Janeway lesions and Osler nodes. Look for Roth spots in the eyes.
- Consider in patients with infections in more than one site, such as multi-lobar pneumonia or a patient with perinephric abscess and pneumonia.
- Consider risk factors such as IV drug use, recent instrumentation or procedures, indwelling lines, hemodialysis, history of IV drug abuse and prosthetic heart valves. Staphylococcus and streptococcus account for about 80% of cases.
- The recommendations for empiric antibiotics for native valves with presumed endocarditis are ampicillin-sulbactam, or amoxicillin-clavulanate, and gentamicin. If you are worried about MRSA (and you should be worried about MRSA), use vancomycin, along with ciprofloxacin and gentamicin.
- For prosthetic valves within one year of placement, use vancomycin, along with gentamicin and rifampin. Rifampin adds more gram-positive coverage and penetrates the biofilm associated with the prosthetic valve.
- If the prosthetic valve was placed more than one year ago, you should treat the patient as you would native-valve endocarditis.

Prophylaxis for endocarditis. Recommendations have changed dramatically with each guideline revision. In general, patients who need endocarditis prophylaxis meet two criteria: 1) they are receiving a specific procedure; 2) they are at high risk. The guidelines in the United Kingdom no longer recommend antibiotic prophylaxis in any situation.

The only procedure that requires endocarditis prophylaxis is an invasive dental procedure.

Who qualifies for prophylaxis? Patients with prosthetic heart valves, a prior history of infective endocarditis, or unrepaired cyanotic congenital heart disease.

Can lab tests help you? The most important lab work to obtain is blood cultures. In general, the more cultures, the better. Obtaining three sets of blood cultures results in identification in 90% of cases. The white blood cell count will only be elevated in about 50%. Elevated ESR and CRP are present only in about two thirds of patients. Half of patients will have some degree of anemia. If there is renal involvement, there may be a slight bump in the serum creatinine.

What are the different pathogens?
- Staphylococcus is generally listed as the number one pathogen for most types of endocarditis. It is most common with IV drug abuse and prosthetic heart valves. Staphylococcus and streptococcus account for about 80% of cases.
- Other less common organisms include: Bartonella, Brucella, Coxiella, and bacteria in the HACEK group (Hemophilus, Aggregatibacter actinomycetemcomitans previously known as Actinobacillus, Cardiobacterium, Eikenella and Kingella). *Editor’s Note: Hemophilus aphrophilus is now known as Aggregatibacter aphrophilus and Aggregatibacter paraphrophilus.*

Your patient has endocarditis. What antibiotics should you use? We don’t have good data to say that immediate antibiotics make a difference (unlike sepsis, pneumonia, and meningitis). You don’t want to start antibiotics prior to obtaining blood cultures.

- The recommendations for empiric antibiotics for native valves with presumed endocarditis are ampicillin-sulbactam, or amoxicillin-clavulanate, and gentamicin. If you are worried about MRSA (and you should be worried about MRSA), use vancomycin, along with ciprofloxacin and gentamicin.
- For prosthetic valves within one year of placement, use vancomycin, along with gentamicin and rifampin. Rifampin adds more gram-positive coverage and penetrates the biofilm associated with the prosthetic valve.
- If the prosthetic valve was placed more than a year prior, treat the patient as you would native-valve endocarditis.

What patients are candidates for surgery? Patients who are starting to develop severe regurgitation involving the aortic valve or mitral valve. You should involve surgery early. If you have cardiothoracic surgery available, call them. If you don’t have a cardiothoracic surgery service, think about transferring these patients. If patients have severe AR or MR, they will likely be in acute heart failure, pulmonary edema, or cardiogenic shock. Involve the surgeon as quickly as possible.

Take-home points. This is a difficult diagnosis to make in most cases because they present so non-specifically. Fever is unreliable. Labs are unreliable. The murmur can be unreliable; a new
murmur is documented only about 50% of the time. Before you diagnosis a patient with fever or myalgias as influenza or viral syndrome, consider this diagnosis. Listen for a murmur. Look for associated signs and symptoms. Consider the patient’s risk factors.

† If you are going to send home a non-toxic patient, who has any risk factors for endocarditis, you need to make sure the patient has close follow-up and consider sending off blood cultures prior to discharge.

Paper Chase 2:
IV Antibiotic Associated Diarrhea in ED Patients
Sanjay Arora MD and Michael Menchine MD


This was a prospective, observational, non-randomized study of patients discharged home from the Emergency Department after receiving antibiotics. Patients who received IV antibiotics in the Emergency Department were twice as likely to develop diarrhea after discharge, compared to those who received oral antibiotics. There are lots of limitations to this study.

† The overall rate of diarrhea with antibiotics was about 1 in 5 patients, regardless of whether they received IV or oral antibiotics. We need to keep this in mind when giving patients antibiotics, and we need to educate our patients about the possible risks and side effects.

† There are risks associated with giving antibiotics to patients: cost, side effects, changing patterns of resistance, setting patient expectations, and practicing bad medicine.

† This addresses the risk of harm, specifically antibiotic-associated diarrhea and C. difficile. This contributes to patient discomfort, overall morbidity and mortality, antibiotic noncompliance, and overall healthcare costs. What is the role of IV antibiotics?

† This is a prospective, observational, non-randomized study of adult patients discharged home from the Emergency Department with a prescription for oral antibiotics. They didn’t intervene in terms of management but collected information at the time of enrollment and at one month follow-up. They were specifically looking at diarrhea, defined as three or more loose stools for 2 days. C. difficile infection was determined by review of the medical records.

† They enrolled 247 patients. Of these, 45 patients (18%) developed diarrhea; 10 patients (4%) had mild diarrhea that did not meet their definition criteria; 2 patients (1%) developed C. difficile colitis. These were outpatients.

† They looked at several variables to identify predictors: IV antibiotics were a risk factor. Twenty-six percent of patients who received IV antibiotics developed diarrhea after they left, compared to 12% with oral antibiotics. The number of doses of antibiotics didn’t seem to matter, but the sample size was too small to adequately examine this.

† The authors reported that the two groups were similar in terms of how sick they were, but the way they determined this by was looking at the average Emergency Severity Index (ESI) categorization, which is fairly nonspecific. If you look at the actual tables, the two groups were very different, which confuses the message of this study. The percentage of patients who were febrile in the IV group was much higher: 14% versus 6%. The diagnoses were skewed: there was more cellulitis in the IV group (47% versus 25%) and much less urinary tract infection (only 5% versus 22%). It would have been nice to see some other measures of how sick the patients were, such as blood pressure, time in the Emergency Department, or IV fluids.


† The general message is that we need to be careful and try to limit IV antibiotics. Narrow the spectrum if you can. Limit IV antibiotics to patients who need them. The bioavailability of oral antibiotics is pretty good. The overall rate of diarrhea is much higher than expected. Don’t overprescribe antibiotics. “I can’t give you medication that might harm you, when I know it won’t help you.”

Case #5

The patient was a 38 year old woman who drove herself to the hospital and was unable to speak. There was no other history available. A review of her records showed that she had visited the Emergency Department once previously and had left prior to evaluation.

† Physical exam. She was alert and able to move her hands and feet. She did not cooperate with the entire neurologic evaluation. She was able to drive to the hospital but was unable to speak. She had isolated aphasia with no facial asymmetry. No focal neurologic deficits.

† Work-up. Laboratory testing was unremarkable. Urine toxicology was negative. Urine dip and pregnancy were normal. Vital signs were normal. CT scan of the brain was negative. The social worker approached Weinstock and reported that she was making arrangements to transport the patient for mental health
evaluation. The patient was texting on her phone. He looked to see what she was texting, and it showed “########.” He canceled the transfer.


- They retrieved some numbers from her phone and called. A friend finally arrived. The patient had a job. The friend did not believe that this had ever happened before and did not know of any previous mental health problems.

- The patient was admitted. She removed her IV and left the hospital without notification. She returned the following day with some improvement in her speech but with residual deficits, and she was readmitted. Neurology and psychiatry were consulted. Psychiatry thought it may be conversion disorder.

- An EEG was normal. MRI showed diffuse leptomeningeal enhancement concerning for meningitis. Acyclovir was started. They considered a lumbar puncture. The differential diagnosis included chronic infection, tuberculosis, HIV, Cryptococcus, HSV, VZV, and CMV. Autoimmune problems such as sarcoid, vasculitis or paraneoplastic syndrome. Neoplasm. Paraneoplastic limbic encephalitis. Mycoplasma. Viral.

- The patient’s grandmother was contacted, who reported that the patient had a history of pulmonary sarcoidosis, which had been confirmed on biopsy. She reported that she had had previous episodes of similar behavior, which had resolved. They determined that the patient had neurosarcoidosis.

- Neurosarcoidosis is very difficult to diagnose. It affects the central and peripheral neurologic system and can present in a variety of ways. It is unique in that it is diagnosed on biopsy. It can be difficult to obtain tissue for biopsy. CT of the chest can demonstrate hilar adenopathy.
  - Treatment is steroids and clinical response is evaluated.

---

**Medical Legal 101 – Part 5 – The Trial**
*Mike Weinstock MD with Mark Kitrik (plaintiff attorney) and Karen Clause (a malpractice defense attorney)*

### Case #6

A 41 year old female presented to the Emergency Department with a chief complaint of chest pain “from the waist to the head, neck, and arms.” Her vital signs showed that she was hypertensive at 186/96, which improved to 130/82 without therapy. She reported a pain score of 8 out 10. She had a past medical history of hypertension and stroke and was smoker. She was non-compliant with her medications and had not taken them for 3 months.

- **History of the present illness.** Chest pain over the last day. The pain is a tightness across the chest and upper arms, worsened by deep breaths, radiating to the left arm.

- **Physical exam.** The patient had some wheezing on exam. An EKG was read by the computer as having a septal infarct, but a repeat did not. The patient received an albuterol aerosol.

- **Progress note.** “Chest x-ray and EKG are reviewed. Albuterol aerosol. Captopril 25 mg PO.”

- She was diagnosed with hypertension, bronchospasm, and given a prescription for Lisinopril. She was sent home. Unfortunately, the next day, her daughter awoke to the patient’s boyfriend performing chest compressions. She was taken to the Emergency Department in cardiopulmonary arrest, and they were unable to resuscitate her. The autopsy showed acute myocardial infarction.

- **How do family members initiate a case?** Usually it is via a phone call. The defense attorney was very interested in the case due to the chest pain, heart attack, and death in a young patient. Was there an autopsy? Was there a history of heart problems? The defense attorney is more likely to take a case if there is social consciousness that makes the case seem obvious or basic to a layperson. Most focus groups and laypeople associate chest pain and arm pain with a heart attack, even if the clinical scenario is more complex.

- **The family member provides the defense attorney with the records.** After the records are obtained, the defense attorney finds an Emergency Medicine physician to review the case and identify the positive and negative aspects. Kitrik looks for doctors who are more defense-oriented because if they say it is a good case, it is more favorable. Payment for the physician’s services is from the attorney’s firm.

- **Malpractice defense attorneys are usually reimbursed by an insurance company.** They obtain all of the records to learn as much as they can about the doctor who is going to be
defended and the plaintiff. They want to see what the doctor did and why he/she did it. They want to meet with the doctor as soon in the process as possible. They send the case for review by an expert. They discuss management with the doctor. Why didn’t you get a troponin? You got two EKGs, was that enough? Why didn’t you keep the patient in the hospital for observation? They want to anticipate what the plaintiff attorney will ask.

- **Some features of this case.** The blood pressure came back down with minimal intervention. The pain didn’t present as consistent with a heart attack. Her EKG was not consistent with an MI.
- **What could the physician have included in his chart to make the defense easier?** “The history was reviewed with the patient and is not consistent with coronary artery disease or evolving MI. The patient is discharged home with instructions to return to the ER if her symptoms persist and follow-up with her family physician.”
- **The records did not contain much medical decision making.** The generalized complaint of pain can be complex for doctors; it could be pulmonary embolism, MI, costochondritis, or reflux. There was no differential diagnosis ruling out the worst-case scenario. This patient had a lot of medical problems. This can help the defense; the patient didn’t take care of herself, she smoked, she didn’t take her medications. The case depends on proximate cause and damages, and the burden of proving this is on the plaintiff.
- **It is helpful for the physician to discuss why he/she doesn’t think it is a particular diagnosis, even if he/she is wrong.** “I don’t think it is pericardial tamponade. The patient has good heart tones. There is no low voltage on EKG. There is no JVD.” The rarer the problem, the more difficult it is for the physician to discuss why he/she doesn’t think it is a particular diagnosis, even if he/she is wrong.
- **Prior to discharge, the physician should step back and review what he/she has done and the tests leading up to discharge.** You may have lost sight of the initial complaint. In this case, the patient was diagnosed with hypertension and bronchospasm, but her presenting complaint of chest pain remained unaddressed.
- **An excerpt from the plaintiff attorney opening statement.** “Good morning everybody. One of the reasons people come to the Emergency Department in the United States is because of chest pain. Not all of it is fatal, and it is not always easy to diagnose. The rule in Emergency Departments is that you treat chest pain as a heart attack until you rule it out. That is the rule. The evidence is going to show that when Stacy left the hospital with her friend, they were in shock. Her friend regrets to this day that she did not bar the door and say, ‘No, we are not leaving. We are not leaving.’ But they did.”
- **You can ask the patient, “Do you feel comfortable going home? Have we answered all your questions?”** Consider discussing the case with your colleagues and get their opinions. Why didn’t you get a troponin? You got two EKGs, was that enough? Why didn’t you keep the patient in the hospital for observation? They want to anticipate what the plaintiff attorney will ask.
- **It has been said that risk factors don’t matter in the evaluation of chest pain. You can’t use lack of risk factors to be reassured that a patient does not have cardiac disease.** This is supported by the American College of Cardiology and the American Heart Association guidelines in 2010 for Emergency Department evaluation of low risk chest pain. However, there is a study by Han in Annals of Emergency Medicine in 2007, including over 10,000 patients with 8.1% experiencing acute coronary syndrome. Patients with four or more risk factors have an increased likelihood ratio, especially in younger patients. “In patients younger than 40 years, having no risk factors had a negative likelihood ratio of 0.17, and having 4 or more risk factors has a positive likelihood ratio of 7.39.”
- **An excerpt from the trial of the plaintiff attorney questioning the physician defendant.**
  - Q. Let’s count the risk factors. One, diabetes, right?
    - A. Yes sir.
  - Q. Two, smoking, correct?
    - A. Yes sir.
  - Q. Three, hypertension or blood pressure, correct?
    - A. Yes sir.
  - Q. Four, stroke?
    - A. Yes sir.
  - Q. Five, age over 40?
    - A. Yes sir.
  - Q. Six, family history?
    - A. Yes sir.
  - Q. It’s a bunch isn’t it?
    - A. Yes sir.
  - Q. It has been said that risk factors don’t matter in the evaluation of chest pain. You can’t use lack of risk factors to be reassured that a patient does not have cardiac disease. This is supported by the American College of Cardiology and the American Heart Association guidelines in 2010 for Emergency Department evaluation of low risk chest pain. However, there is a study by Han in Annals of Emergency Medicine in 2007, including over 10,000 patients with 8.1% experiencing acute coronary syndrome. Patients with four or more risk factors have an increased likelihood ratio, especially in younger patients. “In patients younger than 40 years, having no risk factors had a negative likelihood ratio of 0.17, and having 4 or more risk factors has a positive likelihood ratio of 7.39.”
- **Han, JH et al. The role of cardiac risk factor burden in diagnosing acute coronary syndromes in the emergency department setting. Ann Emerg Med. 2007 Feb;49(2):145-52. PMID: 17145112.**
The physician often practices the deposition beforehand, especially in cases like this where it is a difficult record to defend due to gaps in the record. With practice, the doctor anticipates what is coming and the best ways to answer it.

- The physician often practices the deposition beforehand, especially in cases like this where it is a difficult record to defend due to gaps in the record. With practice, the doctor anticipates what is coming and the best ways to answer it.

- Depositions do make a difference whether or not the case goes to trial, based on how well the plaintiffs, physician, and experts come across. A lot of preparation should go into it. Surprisingly, some physicians are not prepped well. If doctors have an arrogant personality, it is hard to hide. If the doctor is humble, caring and kind, it will come out. Juries are biased in favor of doctors who are humble, caring, and kind.

- While the patient and family may elicit sympathy from the jurors, it can be mitigated if the doctor presents well and the jury believes that doctor did everything he/she could, to take care of the patient. If there isn’t strong testimony from the doctor to counteract testimony from the plaintiff or plaintiff’s witness, it makes it more difficult to prevail. The defense attorney would likely recommend settlement in this case.

- When the patient and family may elicit sympathy from the jurors, it can be mitigated if the doctor can present well and the jury believes that the doctor did everything he/she could, to take care of the patient. If there isn’t strong testimony from the doctor to counteract testimony from the plaintiff or plaintiff’s witness, it makes it more difficult to prevail. The defense attorney would likely recommend settlement in this case.

- The defense usually tries to get a demand from the plaintiff prior to settlement negotiations. The amount of the settlement often depends on the financial loss to the heirs. The demand would likely be in the seven figures, but would probably settle between $500,000 to $1 million for this case.

- The defense attorney would likely recommend settlement in this case.
patient has inadequate blood pressure despite good chest compressions, epinephrine makes sense. **Weingart is utilizing hemodynamic dosing of epinephrine. This is supported by guidelines from the American Heart Association.**

- Hemodynamic dosing of epinephrine is not included in the regular ACLS protocol because it requires end-tidal CO₂, or arterial line monitoring, and few institutions have this capability.


- There is a study currently underway in Great Britain that will likely solve this question. The Jacobs trial didn’t pan out as it was unable to get the necessary numbers. Jacobs, IG et al. Effect of adrenaline on survival in out-of-hospital cardiac arrest: A randomised double-blind placebo-controlled trial. Resuscitation. 2011 Sep;82(9):1138-43. [PMID: 21745533].

- Extend your administration of doses to the maximum time interval, about every 5 minutes. This is a good compromise between using it aggressively and not using it. If your patient has good end-tidal CO₂ on the monitor, consider holding additional doses of vasopressors. If the end-tidal CO₂ starts to flag despite good chest compressions, give a dose of epinephrine and vasopressin.

- As Norm Paradis says, “if” you are doing good CPR with a reasonable end-tidal CO₂ of 20, but the patient is not coming back or entering a shockable rhythm, maybe this end-tidal CO₂ is not good enough and you should aim for a higher value during the next round of “resuscitation.” Nothing good will come from an end-tidal CO₂ of 10.

- The patient with chest pain has a ventricular fibrillation cardiac arrest, and you get the patient back. The patient is awake and the EKG shows STEMI. The patient is going to the cath lab. Should you intubate the patient?


- The patient needs to be defibrillated. Start chest compressions and defibrillate as soon as possible. Don’t worry about the airway. If you did this right, the patient had a minimal arrest time and should wake up and talk to you. Should we intubate them or not? This is a tough call. This is a patient with a failing heart, and such patients do not do well with intubation. However, we don’t want to stick them in the elevator and have them code again. If they do code again, we know they are probably back in ventricular fibrillation.

- ▶ How you handle this scenario depends on your facility. One strategy is to have a doctor that can manage the airway accompany the patient to the cath lab with an LMA or supraglottic airway. This is easy to insert in the elevator. Bring an airway kit. Anesthesia should probably be notified. The cardiologists can always bag the patient if necessary.

- ▶ If you intubate, you need to be concerned, especially if the patient has involvement of the right heart. These patients like to die during intubation. You need to be careful. Use an agent that is as cardiac-stable as possible, such as etomidate or ketamine, if they are in cardiogenic shock. Be careful with your intubation. If they are intubated, they will probably need anesthesia to monitor them in the cath lab anyway. You can discuss with your anesthesiologists; they are going to have to be there regardless, so they can meet you in the cath lab and take over.

- ▶ There has been a lot of discussion about Emergency Department ECMO. Who is a good candidate? Sample sizes are small, but there is increasing evidence coming out regarding this. Data is about to be released showing neurologically intact survival rates of up to 50% in selected patients. Who is a good candidate? The patient should be relatively young (usually less than 65 to 70 years), with a witnessed arrest and CPR initiated right away. Duration of CPR has not been shown to be prognostic. Patients should have a potentially reversible cause. ECMO is a bridge to a more definitive treatment and gets them to the cath lab or embolectomy. The heart is a fairly resilient organ and you can often bring these patients back, as long as they have been receiving CPR.

- ▶ When do you turn off the insulin drip in diabetic ketoacidosis? When the anion gap is closed. In general, Weingart prefers to use pH and base excess. For most, anion gap is the answer because your goal is clearance of ketones, and this is reflected in the anion gap. Base excess and pH can be misleading due to chloride ion imbalance resulting from resuscitation. Severe DKA patients will have a profound acidosis from the ketones flooding into their bloodstream. When you start them on the insulin drip, it allows the liver to start clearing the ketones. The anion gap will get lower. The normal anion gap varies depending on the center and assays, but is usually between 10 and 12. When you get there, you know the ketones are gone. This is when you stop the drip. The glucose usually reaches less than 250 mg/dL (13.9mmol/L) before the anion gap closes.

- ▶ Why can’t you use pH or base excess? Most use normal saline for initial fluid hydration. This makes patients acidic due to hyperchloremic acidosis. Most patients have
functional kidneys, so this is okay. In general, as the ketosis improves, the hyperchloremic acidosis worsens. Sometimes the pH won’t change, even though you have fixed their DKA. It can take a few hours for the kidneys to catch up.

**Do you correct the sodium when calculating the anion gap? No.** The sodium in hyperglycemia is a correct measurement of the patient’s actual serum sodium. It is not erroneous. When the glucose is elevated, it causes the sodium to decrease in order to maintain normal osmolarity. The sodium is lowered because your body is attempting to compensate for the osmotic load of glucose. If you want to know if your patient has a relative hypernatremia or hyponatremia despite the hyperglycemia, you can use the correction factor. This doesn’t really matter until the glucose is fixed. Don’t use anything to calculate the anion gap but the measured sodium. It reflects what is actually present in the bloodstream. Use sodium – (chloride + bicarbonate).

**The patient has a low pH and is being resuscitated. Why shouldn’t you push amps of bicarbonate?** There are countless studies saying that bicarbonate doesn’t work and may make things worse. You can’t fix patients this way. Bicarbonate fixes pH by converting metabolic acidosis to a respiratory acidosis and you can breathe it out. In patients who are maximally compensating with a respiratory alkalosis, they are already blowing down their CO₂ as much as they are capable. Bicarbonate is not the solution. Give insulin and fix the acidosis. When you do finally fix their DKA, you will have a lot of residual alkalosis. You are shifting the acidosis into the cells and the heart may not like it.

**Do you have a question?** Go to the EMRAP site and submit a question. You can also comment in the previous mailbag segments.