What did they find? 9 patients out of 187 survived. 6 patients survived to hospital discharge and 3 patients were organ donors. Every survivor had cardiac motion on ultrasound. Ultrasound done at the bedside by the clinician was very sensitive in identifying patients who would survive. If patients had no evidence of cardiac activity or cardiac tamponade, the survival was zero regardless of any other factors.

There is some criticism of this paper. Simon Laing points out that the number of survivors was small. If there was just one survivor in the group with absence of cardiac motion, the conclusion would have been very different. These findings add to the prognostication but shouldn’t be used in isolation. This may be helpful in centers where thoracotomy is not done frequently and presents major challenges for resource utilization.

What do the guidelines say? The two major US trauma guidelines don’t agree. The EAST guidelines say you should do a thoracotomy if you have pulseless patient, irrespective of signs of life, with a penetrating injury. They have conditional recommendations for thoracotomy in pulseless patients with blunt trauma only if they have signs of life. They do not recommend thoracotomy in pulseless patients with blunt trauma and no signs of life. Western Trauma Association has taken a different approach, utilizing time of CPR as a surrogate marker. They recommend not performing thoracotomy if the patient has received 10 minutes of CPR after blunt trauma and 15 minutes after penetrating trauma. They found no survivors after these times.

Take Home Points
- The two major trauma guidelines do not agree on indications for ED thoracotomy. You need to consider mechanism of injury, down time, presence of signs of life, patient characteristics and available resources in your decision.
- Bedside ultrasound showing no evidence of cardiac motion or cardiac tamponade indicates a survival rate of zero after thoracotomy.
- Who should get a thoracotomy?
  - What was the mechanism of injury? Was it a blunt injury? These have very bad outcomes. Or was it a penetrating injury? Slightly less bad. For penetrating injuries, was it a thoracic injury or an extra-thoracic injury?
  - When did the patient lose the vital signs? Some use the duration of CPR as a surrogate marker. How long has the patient been down? This can be difficult to discern.
  - Are there signs of life? These include pupillary response, spontaneous ventilation, movement of the extremities or cardiac electrical activity.
  - What are the resources and infrastructure available? You need to consider your available resources.
  - You need to consider patient characteristics. A 19 year old male with cardiac arrest secondary to a single stab wound to the heart is very different than a 92 year old patient with blunt trauma.
- Can bedside ultrasound help us determine patients in whom thoracotomy will be futile?
  - This was prospective observational study of 187 patients in traumatic cardiac arrest that underwent point-of-care cardiac ultrasound.
Trauma Surgeons Gone Wild: Part 2 - How To Crack The Chest
Mizuho Spangler DO and Kenji Inaba MD

Take Home Points
1. Visual inspection of the pericardium can’t reliably identify tamponade. Always open the pericardium.
2. Thoracotomies are always initiated on the left side, even in the presence of isolated right sided trauma, to allow for cross-clamping of the aorta and resuscitation of the heart.
3. The patient should have a right sided thoracotomy (or thoracotomy if indicated) to rule out hemorrhagic shock from the right chest.
4. The end goal of thoracotomy is restarting the heart into a viable perfusing rhythm. This may involve intracardiac epinephrine, massage and internal defibrillation.

- What are the essential actions in traumatic cardiac arrest?
  1. Taking control of the airway and oxygenating the patient.
  2. Gaining vascular access and infusion of fluids.
  3. Performing ED thoracotomy on the left side to see what is in the left chest and start resuscitating the heart.
  4. Vent the right chest and make sure there isn’t a large hemothorax or pneumothorax.

- How do we set up?
  - You need to protect yourself. These are high risk patients for communicable diseases. You need to have a gown, gloves and protect yourself from splatter. Double glove and cover the area between your gloves and gown.
  - Call for help. Whether it is surgery, anesthesiology or the hospitalist. Another set of hands can make a huge difference.
  - If you have someone skilled who can help manage the airway and right chest, you can focus on the left chest.
  - The ED thoracotomy should be a wide open thoracotomy. You will insert the chest retractor(s). Make sure that you don’t cut yourself or get hurt on the way in. The chest retractor is used to get the best possible view.

- What are you seeing?
  - Is there blood in the pericardium and can you get it out? It can be difficult to see tamponade especially with a fatty pericardium. Always open the pericardium. Incise along the pericardium medially making sure that the phrenic nerve isn’t injured. You should be able to see it. If there is no blood, you are done. If there is blood, you need to open the pericardium all the way and look for the hole. It is very rare to have blood in the pericardium in blunt injury.
  - If there is a hole, you can use 2-0 prolene to repair the hole. Otherwise you can place a foley catheter or your finger in place.
  - Is there hemorrhagic shock from the right side of the chest? Place a chest tube to see if there is a large amount of hemorrhage. If there is, the patient needs a right sided thoracotomy to control the hemorrhage.
  - Why are thoracotomies always initiated on the left side, even the trauma is isolated to the right side? Because we can cross-clamp the aorta and restart the heart. If the left-sided thoracotomy is negative, you can then rule out a source of hemorrhage on the right side. The clamshell incision gives you unimpeded access to all of the thoracic and mediastinal contents.
  - What is the end goal of an ED thoracotomy? The end goal is to get the heart restarted into a viable perfusing rhythm. After entering the left chest and stopping acute hemorrhage, you need to address the heart. Start with cardiac massage. You need to get some of the blood recirculating. Volume and epinephrine are both important. Resuscitation should be done with cross-clamp of the aorta in place. Intracardiac epinephrine is injected directly into the left ventricular cavity followed by cardiac massage. You want to place the needle in the hollow of the left ventricle. All efforts should be focused on getting the heart started.
  - What sequence of medications should we use? It is not well established. At LAC+USC, they use a combination of epinephrine, 20-40 units of vasopressin and bicarbonate. However, there is no supporting evidence.
  - It is not uncommon to develop a fibrillating rhythm. You need a defibrillator with internal paddles. Ventricular fibrillation is determined based on visual inspection and not cardiac monitoring. You need to defibrillate the heart directly. Ventricular fibrillation is better than cardiac standstill. The end goal is sinus tachycardia.
  - When is it time to stop resuscitation? This can be difficult to determine, particularly in pediatric patients. Check with the entire team to make sure everyone is okay with stopping resuscitation.
  - There are three potential outcomes that can occur with successful resuscitation. Survivors. Neurologically intact survivors. Potential organ donors.
Chris Hicks on ED thoracotomy

- **This is procedure where you need a specific script.** We are not as comfortable with ED thoracotomies as trauma surgeons.

- **If you are going into the left chest, have someone decompress the right side of the chest.** You can’t determine tamponade based on the appearance of the pericardium. Open up the pericardium.

- **If the patient is intubated, have the respiratory therapist hold ventilation or advance the tube into the right mainstem.** Drop the left lung and improve your view. Place an NG tube. The aorta can be difficult to distinguish from the esophagus. You don’t want to cross-clamp both.

- **Our primary role is to make the decision to open the chest in a patient that we think is salvageable.** Deliver the heart. Open the pericardium. Examine for cardiac injuries and try to repair or tamponade it. Do internal compressions if it is indicated. You can stop at this point unless you are at a trauma center and feel comfortable with cross-clamping the aorta, doing a hilar twist, etc.

- **If you have a stab wound to the chest with isolated cardiac injury and tamponade, survival with good neurologic outcome is between 30-40%.** That is pretty extraordinary.

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**Cardiology Corner:**

**Cardiogenic Shock**

Rob Orman MD and Amal Mattu MD

**Take Home Points**

- If a patient has an MI with hypotension and pulmonary edema, you need to give inotropic support such as dobutamine.

- Dobutamine may cause tachycardia and vasodilation. You may also need to give norepinephrine.

- Aortic balloon pumps are not routinely indicated but may be used to bridge the patient to the cath lab or definitive therapy.

- Get the patient out of the ED and to the cath lab or cardiac surgeon as quickly as possible.

- Patients with a right ventricular MI are very fluid dependent.

- Congestive heart failure exists on a spectrum. Cardiogenic shock scares us the most as there is little advice on how to manage it.


- Get the patient out of your emergency department. For the most part, there is not much that we can do in the ED except for support the patient until they can go upstairs. For most other types of shock (septic, hemorrhagic, hypovolemic, obstructive shock from massive pulmonary embolism or tamponade, anaphylaxis or adrenal shock), we do a pretty good job of managing in the emergency department. We can almost provide definitive care for those patients in the ED. Cardiogenic shock is different. We can’t provide definitive care for these patients.

- With rare exceptions, these patients need to go upstairs to the cath lab or cardiac surgeon. If you get a patient in cardiogenic shock presenting to the ED, your first step should be to call to get someone to accept the patient.

- What is cardiogenic shock? Hypoperfusion of the end organs with a primary cause of failure of the pump. Not all MIs will manifest with cardiogenic shock although STEMI is the most common cause. This typically results from a large anterior MI with an extensive area of involvement.

- Shock may result from a right ventricular MI causing the right ventricle to fail, followed by failure of the left ventricle. These patients will be hypotensive and have pulmonary edema.

  - Mechanical causes of cardiogenic shock include rupture of the papillary muscles leading to valvular insufficiency with failure of forward flow.

- You have patient with an MI and a blood pressure of 70. Bedside echocardiogram shows a floppy, poorly contracting heart.

- If the patient has MI with hypotension and pulmonary edema, you need to provide some type of inotropic support.

- Dobutamine is probably the best inotrope we have. However, dobutamine will increase myocardial oxygen consumption and can increase ischemia. It causes tachycardia. It can produce vasodilation in ⅓ of patients and may worsen blood pressure. Dobutamine is really an inodilator and increases cardiac contractility with peripheral vasodilation.

- If the patient is hypotensive and dobutamine isn’t helping, you need to add an additional vasopressor. Norepinephrine is the vasopressor of choice. This is an inopressor; it increases cardiac contractility as well. Norepinephrine may increase afterload and increase the resistance to the heart pumping. Epinephrine is also in this category.

- Titrate the medications as tolerated.

- There is no simple formula.

- There is theoretical benefit to balloon pumps. These decrease left ventricular afterload which will decrease wall tension and myocardial oxygen demand. They improve diastolic perfusion pressures in the coronary arteries. However, recent studies show that routine use of the balloon pumps is not associated with ben-
eft. The Van Herck paper advises that balloon pumps are not routinely recommended unless there is mechanical complication such as a ruptured mitral valve. They are used occasionally to try to bridge the patient to cath lab or other intervention.

- **Are these patients candidates for thrombolytics?**
  - The literature says that thrombolytics don’t work well in cardiogenic shock. However, if you are at a facility that lacks a cath lab or cardiac surgeon, you may have no other choice but to give lysics to your STEMI patient. This paper suggests that it is a reasonable choice. If you are within 60-90 minutes of a cath lab, skip the thrombolytics and send them to a cath lab.
  - In general, thrombolytics will fail to work in ⅓ of the time in all-comers with STEMI who are hemodynamically stable. This often has to do with the magnitude of occlusion and number of involved vessels. Thrombolytics are less likely to work in critical, proximal LAD lesions or acute left main occlusion and these same patients are at high risk of cardiogenic shock.

- **Should you give anti-platelet medications to these patients?**
  - Everyone should get aspirin and heparin. Unfractionated heparin infusion is recommended. What about the fancy newer anti-platelet agents such as clopidogrel or ticagrelor? These are contraindicated if a patient is going for bypass. Most literature recommends not giving these medications to patients with cardiogenic shock because so many will be going on to bypass surgery.

- **Give the patient aspirin and heparin and let the patient go to the cath lab.** The cardiologists will give the newer agents in the cath lab if they are able to fix the problem. If they can’t fix the problem, the patient will be sent for bypass surgery. Everyone will be happy that you didn’t give those medications.

- **A classic scenario for a patient with MI and shock is the right ventricular infarct. What can you do for these patients?** These patients are very fluid dependent. The purpose of the right ventricle is to pump blood to the left ventricle which produces cardiac output. If the right ventricle is damaged, the only thing providing blood to the left ventricle is preload. If you give nitrates or anything that drops the preload, the patient will get very hypotensive. If the patient is already intravascularly depleted, they may already be hypotensive. These patients need a lot of volume to provide preload for cardiac output.

  - **Keep checking their lungs.** As long as the lungs are clear, you can keep giving fluids. If the right ventricle is dysfunctional, fluid will back up and the patient will have JVD and a plump IVC even though the left ventricle is not getting enough blood.

  - **Dobutamine increases tachycardia and increases myocardial oxygen consumption which increases ischemia. Stay away from pressors or inotropes as long as the patient is fluid responsive.**

- **Are there any other therapies that we should consider in patients with cardiogenic shock?**
  - Therapeutic hypothermia is being studied in cardiogenic shock. There are two studies that suggest that therapeutic hypothermia may be beneficial but it is not currently recommended.

  - ECMO. There are increasing reports on the use of ECMO.

  - Patients in cardiogenic shock are probably going to benefit from endotracheal intubation and ventilation as it takes away the work of breathing. Add PEEP as this will help with pulmonary congestion. Resuscitate before you intubate.

- **Summary.** The majority of patients with cardiogenic shock will have a large MI that is often anterior and involving 40% or more of the left ventricle.
  1. Start an inotrope like dobutamine and an inopressor like norepinephrine if you do not receive enough cardiac contractility from the first drug.
  2. Call your cardiologist and cardiac surgeon to get the patient out of the ED and up to the cath lab or OR.
  3. Have a low threshold to intubate these patients. Be careful. These patients are asking for a peri-intubation arrest. They are hypotensive, hypoxic and acidemic. Resuscitate before you intubate. Give fluids and vasoactive agents before RSI.
  4. Give them an aspirin and unfractionated heparin. Hold off on the other anti-platelet agents.

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**CASE**

A 68 year old man with an inferior/right ventricular MI received 13L of normal saline over 24 hours to maintain his blood pressure. [Ed, note: this seems a bit exceptional, most patients will not require this much fluid!]
T here were no differences in adverse events, length of stay or admissions.

This seems like a safe and easy thing to do and seems more effective than traditional Valsalva. The Lancet website has a video of how to perform the procedure.

**Paper Chase 1:**

**Valsalva for SVT**

Sanjay Arora MD and Michael Menchine MD

**Take Home Point**

- A randomized controlled trial in patients with stable supraventricular tachycardias found using a modified Valsalva maneuver with supine position and passive leg raise resulted in three times the rate of conversion to sinus rhythm compared with standard Valsalva maneuver.

- **Appelboom, A et al. Postural modification to the standard Valsalva maneuver for emergency treatment of supraventricular tachycardias (REVERT): a randomized controlled trial.** Lancet. 2015 Oct 31;384(10005):1747-53. OPEN ACCESS LINK

- This was a randomized controlled trial of 433 patients with stable SVT that compared standard Valsalva maneuver to modified Valsalva maneuver with supine position and passive leg raise. The treatment group had three times the rate of sinus rhythm compared to standard Valsalva maneuver.

- When patients present with stable supraventricular tachycardia, it is recommended to try Valsalva maneuver before treating with adenosine. However, Valsalva is rarely effective and we don’t often do it correctly. The published rates of conversion with Valsalva maneuver are between 5-20%. Clinical experience suggests that this is on the low range. Adenosine isn’t fun for the patient or provider.

- Prior to this study, there were only two trials looking at positioning during Valsalva. There does seem to be some effect.

- In this study, the authors conducted a multicenter, randomized controlled trial examining a sitting position for Valsalva versus lying down with leg lift. Patients were instructed to blow into a manometer with a goal of sustaining 40mmHg of pressure for 15 seconds. They included stable adult patients and excluded those unable to lie down or cooperate with Valsalva. This paper had good methodology.

- Why not start in this position? The authors explained that there are two points in time that Valsalva is likely to work; right at the onset of Valsalva or, more likely, at the end when they take a deep breath.

- 433 patients were eligible and randomized. Spontaneous conversion occurred in about 5% prior to Valsalva and these were excluded. 43% of patients in the intervention group had normal sinus rhythm at one minute, compared with 17% of the control group. Most were on the first attempt. This was an odds ratio of 3.7 and an absolute difference between the groups of 27%.

- Adenosine was ultimately used in 69% of the standard group and 50% of the treatment group.

**Paper Chase 2:**

**CT for Diaphragm Injury**

Sanjay Arora MD and Michael Menchine MD

**Take Home Points**

- Diaphragmatic injuries from penetrating trauma may be difficult to identify and can result in severe complications later on.

- The traditional approach to identifying these wounds is exploratory laparotomy but this has a negative rate of two thirds.

- A case series in patients with penetrating thoracoabdominal stab wounds found that multislice CT scans were only 82% sensitive in identifying injury and are not sufficient to rule in or rule out this injury.


- This is a case series of 43 patients with penetrating thoracoabdominal injuries that found that multislice CT scans were 82% sensitive and 88% specific for diaphragmatic injury.

- Stab wounds to the chest are common. Stab wounds to the thoracoabdominal region between the 4th intercostal space and rib margin anteriorly and the tip of the scapula to the last rib posteriorly can result in diaphragmatic laceration. The incidence of diaphragmatic injuries with stab wounds to the region has been reported to be between 5% to over 30%.

- These injuries are significant for a number of reasons. It is difficult to identify this injury. The diaphragm is thin, the wounds are often small and the patients are usually asymptomatic from the diaphragmatic injury. The diaphragm does not heal well. Missed diagnoses may tear further and a third will progress to have viscus herniation into the chest which can result in complications and be difficult to repair later on. This complication may occur months to years after the initial injury. This is most relevant when the stab wound is on the left side. Injuries on the right are less concerning because the liver prevents herniation into the chest.

- The traditional approach to identifying these wounds is exploratory laparotomy. However, the negative rate in stable patients is two thirds. Other approaches include laparoscopy or a
HEART Gets Put To The Test

Paper Chase 3: CT scanners.

**What did they find?** Of 38 patients, 11 (29%) had a diaphragmatic injury. CT scan identified 9 of these patients and had two false positives giving a sensitivity of 82% (not great) and specificity of 88%. This was using the current generation of multislice CT scanners.

**Take Home Points**

- A randomized controlled trial in patients with symptoms of ACS without STEMI found that utilizing the HEART score and two serial troponins to identify low risk patients led to double the rate of discharge with no major adverse cardiac events by 30 days.
- The HEART score was developed in the Netherlands and involves five components: history, EKG, age, risk factors, and troponin level.
- Patients identified as low risk per HEART score with a negative troponin have a 2% risk of major adverse cardiac event within 30 days. Two negative troponins lowers this to 1%.
- This was a randomized controlled trial of 282 patients with symptoms of ACS without STEMI managed via provider discretion or the HEART Pathway utilizing the HEART score and two serial troponins. Those in the HEART Pathway had double the rate of discharge with no major adverse cardiac events by 30 days.
- The official recommendations from the ACC/AHA guidelines still state that even low risk patients with chest pain should receive provocative testing or CT within 72 hours and preferably within 24 hours. We see a lot of low risk chest pain and management can be frustrating. In response, accelerated protocols are being created which include the use of chest pain units and troponins to help mitigate this process while minimizing legal risk. The need to test these low risk patients is under debate and not part of this discussion. However, if you need an accelerated diagnostic protocol (ADP) at your hospital or a way to categorize these patients, consider using the HEART score.
- The HEART score was developed in the Netherlands and involves five components: history, EKG, age, risk factors, and troponin level. A score of 0-3 indicates low risk for a major cardiac event at 6 weeks. This was found true even in high risk sub-groups of diabetics, elderly and female patients.
- This article evaluated the HEART score in a randomized controlled trial. They included 282 patients with symptoms of ACS without STEMI and randomized them to the HEART Pathway or usual care. The major difference between the HEART pathway in this study and the original article was the addition of a second troponin at 3 hours. They did this to be more in line with common practice in the United States. Patients who were low risk were discharged home, medium risk patients were sent to an obs chest pain unit and high risk patients were admitted. The outcome was the rate of advanced cardiac testing at the 30 days and major adverse cardiac event by 30 days.
- There were some issues with the follow-up of patients, assessment of major adverse cardiac events and how patients lost to follow-up were handled but overall the methodology was not too bad.
- What did they find? 40% of patients in the HEART Pathway were discharged compared to 18% of patients with usual care. About 60% of patients in the HEART pathway eventually received provocative testing compared with 70% in the usual care. The ACC/AHA guidelines recommend all patients receive provocative testing. However, there is a growing body of literature suggesting that this may be overkill as none of the patients who were discharged home had any adverse cardiac events at 30 days.
- This was a small trial. There were some protocol violations.
- To date, over 6000 patients have been reported to have been...
evaluated with the HEART pathway and it seems that with negative troponin, the possibility of major adverse cardiac events (MACE) within a month is about 2%. The addition of a second troponin dropped the risk to about 1%. It is important to know these numbers and the HEART score so that you can discuss this with patients. If you discharge a patient home after two negative troponins and a HEART score of 0-3 with a plan for provocative testing within 72 hour follow-up and it doesn't actually occur, don't freak out because the chances of something bad happening are very low.

- It is possible to have a HEART score of 0-3 with a positive troponin. If a patient has positive troponin, you should probably admit the patient regardless of the HEART score.

### Paper Chase 4: Blood Clots and Cancer Screening
Sanjay Arora MD and Michael Menchine MD

**Take Home Points**

- A randomized controlled trial in patients with unprovoked blood clots found augmented intensive screening with CT scans was no better than the usual age and gender appropriate screening for identifying occult cancer.

- The incidence of cancer in patients with unprovoked thromboembolic disease is low at 3.9%.


- This was a randomized controlled trial in patients with unprovoked blood clots randomized to age and gender appropriate screening or augmented intensive screening with CT scans. The incidence of occult cancer was about the same in both groups suggesting that a big work-up for cancer beyond the usual is unnecessary.

- This is a large, multi-site randomized controlled trial that demonstrates the incidence of occult cancer following apparent unprovoked DVT or PE is low at 3.9%. The incidence is not significantly different whether the cancer screening is performed with an age/gender specific protocol or an age/gender specific protocol augmented with CT scans.

- Unprovoked VTEs arise in the absence of cancer, known thrombophilia or transient risk factor such immobility or surgery. We diagnose these frequently. There is an increasing push to discharge these patients home when they are hemodynamically stable. Not much happens during the inpatient management of DVT. However, the internist often works up the patient for an occult cancer. This may not happen if the patient is discharged home from the emergency department. Some literature suggests that the risk of diagnosis of cancer in the year following an unprovoked DVT is upwards of 10%. This might make some ER doctors nervous about outpatient management. Should these patients be screened in the ED prior to discharge? Does this increase liability for ER physicians?

- In general, screening patients with unprovoked VTE for cancer is thought to be a good idea. Most of the conflict arises in what constitutes adequate screening. For a young, otherwise healthy patient, performing a history and physical may be sufficient. Have you had unexplained weight loss? Are you coughing? If the patient is elderly with risk factors for colon cancer, they may need colonoscopy. However, others have argued that the work-up needs to be more extensive and involve CT imaging.

- In this study, the authors looked at 854 patients from several hospitals in Canada who were diagnosed with unprovoked VTE. 67% had DVT and 33% had pulmonary embolism. Patients were randomized to a simple cancer work-up including history and physical exam, basic blood tests and chest x-ray. Older women received a mammogram. Older men received a PSA. This was compared to a similar approach plus a very fancy CT scan. The CT scan was a virtual colonoscopy and gastroscopy, cystoscopy, pancreatography and multiphase liver CT scan. The work-up was performed within 3 weeks of the initial diagnosis of venous thromboembolic disease.

- The key outcomes were missed occult cancers diagnosed within one week. How many cancers were diagnosed with each screening strategy up front and how many additional cancers were diagnosed in the following year? It was thought that more aggressive screening would diagnose more cancers initially and fewer later on.

- What did they find? Overall, the incidence of occult cancer was small in the cohort at 3.9%. The majority were found on the initial screening phase. Very few were missed. This was independent of the screening method used. Only 4 patients in the limited screening group and 5 patients in the augmented screening work-up had a cancer diagnosed within a year after initial negative screening. This is equivalent to the frequency of cancer diagnosis in the general population.

- This study helps us understand the incidence of cancer with unprovoked VTE (about 1 in 30 and not 1 in 10 as previously thought) and that a general screening approach conducted within a month after diagnosis is appropriate.
Paper Chase 5: Pediatric Seizures and Brain Badness
Sanjay Arora MD and Michael Menchine MD

Take Home Points
- Although children between the ages of 1 month to 18 years with an unprovoked seizure had a rate of 11% for having clinically relevant intracranial abnormalities identified on imaging, less than 1% had an emergent or urgent condition.
- Patients with a focal seizure or high-risk history (history of brain tumor, prior stroke, coagulopathy, sickle-cell disease and prior surgery with a shunt) are more likely to have relevant intracranial abnormalities.
- Children between the ages of 1 month to 18 years with seemingly unprovoked seizures had a rate of 11% for clinically relevant intracranial abnormalities but less than 1% had something emergent or urgent.
- Focal seizures are a bad prognostic sign.
- This study looks at unprovoked seizures not associated with fever or trauma. The rate of positive findings on imaging ranges widely in the literature from 10% to 33%. The rate of emergent findings is between 1% to 10%. These estimates are not very good and obtained primarily from retrospective data.
- This was a prospective study to determine the prevalence and risk factors for clinically relevant abnormalities in kids with first time, unprovoked seizures. This was a prospective study conducted at six urban EDs over two years. They included patients between one month and 18 years. Prior to initiation of the study, they determined findings considered benign and urgent/emergent. Examples of benign abnormalities included Chiari malformation. Examples of urgent/emergent abnormalities included tumor, hemorrhage and edema. Although they tried to enroll all comers, they enrolled 475 of 625 eligible patients (about 75%). There was no protocol dictating work-up; it was left to physician discretion. 80% of patients received imaging at some point within 4 months after ED visit. They do not say how many of these patients received their imaging study from the ED.
- What did they find? 11% had some clinically relevant abnormality. Only 3 children (0.8%) had an emergent or urgent condition. These included two tumors and an intracranial hemorrhage. The children that did not receive imaging were followed up and none required surgical intervention. However, some were lost to follow-up.
- Which kids should be scanned? They performed multivariate regression on a bunch of potential historical exam features including headache, behavior change, altered mental status, etc. They found only two significant features; focal seizure and high-risk history. What was a high risk history? History of brain tumor, prior stroke, coagulopathy, sickle-cell disease and prior surgery with a shunt. These findings should be interpreted with caution as there were very few true positives here. Some variables were significant in the univariate analysis such as duration greater than 50 minutes that did not hold true during the multivariate analysis. They didn’t examine age.
- Really emergent findings are rare. If parents refuse or we decide not to do CT imaging and defer the decision to a neurologist or PMD, the chances of missing something really bad are very low. Most of these children should probably receive MRI.

The UTI That Isn’t
Rob Orman MD and Dave Glaser MD

Take Home Points
- Asymptomatic bacteriuria is very common in adults, occurring in up to 20% of healthy women and 50% of long term care residents.
- A urinary tract infection is a positive urinalysis with signs and symptoms of a urinary tract infection.
- Watchful waiting is an appropriate strategy in asymptomatic patients. The likelihood of cystitis progressing to pyelonephritis is 1 in 38.
- An 80-year-old female presents after mechanical fall resulting in a pelvic fracture. She denies dysuria or increased frequency and urgency. She is afebrile and asymptomatic. As part of her work-up, she receives a urinalysis that shows 10-15 wbc/hpf and bacteria. Diagnosis? Pelvic fracture and urinary tract infection. She is sent home with a walker and a script for antibiotics.
- You have two 80 year old patients. Both patients have 3+ bacteria and 15 wbc/hpf. One patient has dysuria and the other does not. Does it make a difference? We view urinary tract infection as a laboratory diagnosis. We forget that the differential of a positive urinalysis also includes asymptomatic bacteriuria. We have been trained that bacteria and white blood cells in the urine indicate an infection. This is wrong.
- What is the prevalence of asymptomatic bacteriuria in adults? It is very common. It is bacteria with or without white blood cells in the urine and no infection. Asymptomatic bacteriuria is more common with older age. A study found a rate of asymptomatic bacteriuria of 5% in sexually active young women. This rate increases with age. Up to 20% of healthy women in the communi-
ty will have asymptomatic bacteriuria. 15% of men over the age of 75 will have it. The incidence can reach 50% of women and 40% of men in the long-term care population.


- There are commensal bacteria that live in happy harmony in the bladder of these patients. This may not be continuous. Patients may clear their bacteria only to have recurrence later.

- What is a urinary tract infection? UTI is a positive UA (usually a positive urine culture or at least 10 wbc/hpf and bacteria) with signs and symptoms of a urinary tract infection. Older patients may have altered mental status and not genitourinary symptoms. We are often happy to ascribe vague symptoms in the elderly such as dizziness, nausea or falls to a positive urinalysis and treat as an infection. Older patients may not develop specific urinary tract symptoms. However, you need some clinical symptom in the otherwise healthy, alert patient population to call it a UTI. We are probably doing harm by treating these patients.

- The degree of positivity doesn't help in the diagnosis of UTI. Patients in long-term care or chronic incontinence are likely to have asymptomatic bacteriuria. It is the clinician’s job to determine if they have a urinary tract infection. It is likely pyelonephritis if you are going to ascribe altered mentation or other systemic symptoms to urinary tract infection. Simple cystitis does not cause systemic symptoms.

- Watchful waiting is a good idea in many of these patients. Some studies have shown that 25-50% of women with typical cystitis symptoms and positive urinalysis will have spontaneous resolution of their cystitis within a week. The likelihood of progressing from cystitis to pyelonephritis has been cited as 1 in 38 times. Often times a positive urinalysis will not have positive cultures.


- A study found 43% of patients with a diagnosis of urinary tract infection did not have positive urine cultures. 95% of these were given antibiotics.


- There is concern for complications of antibiotic use such as C. difficile. Some believe that some bacteria may be uroprotective and keeping more pathogenic bacteria at bay. Killing these bacteria may increase the risk of infection with more pathogenic bacteria. We like to act on findings but this can result in harm over a global population.

- If asymptomatic bacteriuria grows out bacteria on culture, is it considered a UTI? No. The culture does not distinguish between asymptomatic bacteriuria or infection. You need the clinical signs and symptoms.

- When should you send urine? If the patient presents with symptoms of infection, they need urinalysis. If the patient is elderly with a fall but alert without urinary tract symptoms, they don’t need a UA. The specificity of UA for a UTI is quite low.

- What should you do with patients with indwelling catheters? These are always positive for everything. These patients have a higher rate of asymptomatic bacteriuria. You need to decide clinically if they are infected. If they don’t have any other signs of infection, you are better off not sending the urine.

- Get the urinalysis if they have signs and symptoms referable to a urinary tract infection or as part of work-up of stone. Don’t get the urinalysis for vague symptoms. You will overtreat and hurt a lot of your patients. Don’t treat the lab, treat the patient.

### Amiodarone Pulmonary Toxicity

**Mike Weinstock MD, Rob Orman MD, Chris Frank MD and Ruth Greenwald**

**Take Home Points**

- Patient and families are more likely to sue based on negative perceptions of the interaction with the physician than the science or diagnostics.

- Amiodarone use may rarely result in toxicity to the lungs.

- Amiodarone lung toxicity may mimic a variety of medical conditions such as congestive heart failure or pneumonia. It usually improves with discontinuation of the drug.

**CASE**

Fred Greenwald was a former stockbroker who moved to Florida after retirement. He had some medical issues such as coronary bypass, atrial fibrillation and melanoma but was doing well. One day he reported that he had experienced shortness of breath upon exiting a store earlier in the day. His wife encouraged him to go to the doctor. His cardiologist obtained a chest x-ray that showed "double pneumonia". The patient went to the emergency department. He was well-appearing and not hypoxic. He had no fevers or cough. The ED physician told him that he could have "walking pneumonia" and confirmed the diagnosis made by the cardiologist. The chest x-ray showed involvement of both lungs. The pulmonologist came by and evaluated the images. He thought that it was due to amiodarone toxicity and not pneumonia.
When the nurse arrived with his pills, Fred refused the amiodarone. The cardiologist was rude and dismissive when informed of the decision to stop amiodarone. Fred had taken amiodarone for his atrial fibrillation for two years. When the family did further research, they learned that amiodarone could result in lung toxicity and death.

Fred was admitted to the hospital. He received antibiotics and oxygen. However, he became increasingly dyspneic. After 12 days, the inpatient team had a discussion with his wife Ruth. They reported that Fred wasn’t improving. They recommended hospice which the family agreed to. Fred was made comfortable. When he realized that he was dying, he said, “Well, I’ve had a good life.” He died 14 days after hospital admission.

His cardiologist had never discussed the potential for lung toxicity with amiodarone. His wife decided to sue the cardiologist after the death. She spoke with a lawyer who advised that Fred was 78 years old, semi-retired, had lived a long life and they would be unable to get enough money from the case. The reason Ruth decided to sue was based on her interactions with the cardiologist. She regarded him as a cold man who didn’t have good bedside manner and was not informative.

When we think about lawsuits, we focus on the medicine. However, the real reason patients sue often has little to do with the science and diagnostics. If the cardiologist had been more conciliatory or had a different approach. She would have viewed it differently if he had said, “I gave him the amiodarone for his atrial fibrillation. I didn’t expect it to cause a problem. I told your husband to come to me for any problems.”

Although we don’t prescribe long term amiodarone, there are lessons we can learn from this case. Shortness of breath with what looks like pneumonia could also be due to another etiology. There was diagnosis momentum in this case.

How can we do better when there is a decision to be made with the potential for good and bad outcomes? Inform the patient as much as possible. If you don’t know the answer, say so. “I will try to find the answer out for you.”

What do we need to know about amiodarone? We frequently see patients on this medication. It is the most commonly used anti-dysrhythmic and is used worldwide for atrial fibrillation and ventricular tachycardia. The reported rate of pulmonary toxicity is about 1-2% per patient/per year. At best, anti-arhythmic don’t kill people. We use amiodarone so frequently because it has a neutral effect on mortality. All other anti-dysrhythmics have an increased mortality under certain circumstances. There should be a discussion of risks and benefits with the patient that includes discussion of need for frequent monitoring with labs and imaging.

There are formal guidelines on appropriate monitoring for amiodarone use. These have become less intense with time.

- Thyroid and liver function is tested prior to initiation of therapy, at three months post-initiation and every six months thereafter.
- Patients should be monitored for visual toxicity such as corneal deposits.
- The most feared complication is lung toxicity. There is no good screening for this although patients will often receive a chest x-ray every year. Ideally, patients should receive pulmonary function testing prior to starting amiodarone. Patients used to receive pulmonary function testing yearly but this was not very helpful except in patients who were impaired at onset. “Your chance of having problems with your lungs is 1-2% and it can present with any combination of fever, hypoxia, dyspnea, cough or other symptoms. It can result in any chest x-ray finding. If you have a lung disease, especially pneumonia, and it isn’t getting better, you need to consider amiodarone toxicity.”

How do you make the diagnosis of amiodarone lung toxicity? Stop the drug and wait several weeks. There are no diagnostic imaging, laboratory, biopsy or bronchoscopy findings. “This seems clinically consistent with the diagnosis so I want to stop the drug and see what happens.”

Rapid progression is unusual. There is an acute presentation of lung toxicity that typically occurs within a few months after starting the drug. The majority are subacute presentations with low grade fevers, cough, not improving with antibiotics or diuretics that improve after stopping the drug.

What is the half-life? Amiodarone has extremely complicated pharmacokinetics with multiple compartments. The terminal half-life is measured in weeks to months. In a chronic user, the drug will not be fully out of the system for six months. It deposits in fat tissues and may have a longer half-life in people with chronic use and increased adipose tissue.

What can we do to avoid missing this diagnosis in the emergency department? Nothing. This is a very difficult diagnosis to make in the ED. If you see a chest x-ray that appears consistent with pneumonia or congestive heart failure, you need to consider if this makes sense clinically. Amiodarone toxicity may be very difficult to differentiate from pneumonia; it may present as a lobar pneumonia with fever, leukocytosis and a cough. Radiologists are often quick to determine physiology based on the imaging appearance but it is not that simple. The diagnosis needs to be placed in clinical context. If the patient does not respond to antibiotics, amiodarone toxicity should be considered.

Early closure of the diagnostic process is a common error. Don’t let the diagnosis made by the internist or cardiologist influence your work-up and evaluation of the process. Keep an open mind and make sure that other diseases are considered in the differential.
• **Amiodarone lung toxicity is often reversible.** Mild cases may improve with simple discontinuation of the drug. Steroid use is controversial and reserved for severe cases. The second step is supportive care. Steroids may be considered if the patient is not improving.

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**Pediatric Pearls:**

**Pediatric Rheumatology**

Ilene Claudius MD, Sol Behar MD and Caroline Chang MD

**Take Home Points**

- Always consider the possibility of infection in patients with juvenile idiopathic arthritis presenting with a flare.
- Macrophage activation syndrome (MAS) is due to a cytokine storm and can be life-threatening. JIA and lupus may be associated with MAS.
- Well-appearing patients with JIA and a mild flare may respond to NSAIDs. A pulse of oral steroids may help more severe flares.
- Rituximab is administered in two doses given two weeks apart but the immunosuppressive effects can last up to a year.

- **What is JIA?** Juvenile idiopathic arthritis. This term encompasses seven different subtypes.
- A patient with a known diagnosis of JIA presents to the ED with a complaint of a flare. What should you do?
  - Lab testing is a good idea. In most types of JIA, CBC with differential, ESR, CRP are adequate. This is to help confirm a flare and look for other things. In some types of JIA, patients may have elevated ESR with normal CRP. Other patients may have very active disease with normal CRP and ESR. Normal labs do not mean the patient is not experiencing a flare.
  - JIA is primarily a clinical diagnosis and the patient should be examined. What joints are involved? How does the patient look?
  - Always consider the possibility of infection. Infection can mimic a flare or trigger a flare. Treatment of flares often involves steroids which suppress the immune system so you want to know if the patient has an infection. Patients may also already be on immunosuppressive therapies which may increase their risk of certain infections and affect their ability to fight them.
  - How can you distinguish between an arthritis flare and a septic joint? It may be very hard to differentiate. Typically, patients with oligo JIA do not have fever or elevated inflammatory markers. The CRP is important. With most infections, the CRP is elevated if the ESR is elevated. If both markers are elevated, the patient is febrile and the joint is very hot and red, you need to presume it is a septic joint until proven otherwise. If the patient is otherwise well-appearing, the joint is warm but not hot and the labs are normal in a patient with a known diagnosis of JIA, it is safer to assume a flare. When in doubt, don't rely on the markers and aspirate the joint.

- **What is macrophage activation syndrome?**
  - This is a cytokine storm. The macrophages are activated and unable to turn off. It is similar to sepsis; everything is activated and patients may go into DIC. These patients are not well-appearing. They have very high inflammatory markers and may develop renal failure, cytopenias, abnormal liver function and the entire body may be involved. This is life threatening. It may be triggered by infection or certain autoimmune diseases. Lupus and systemic JIA patients may get MAS.

  - If you suspect MAS in a patient, you should check a CBC with differential to look for cytopenias, ESR and CRP. If the patient is very early in their presentation, the ESR should be very high. As their disease progresses, the ESR may drop and this is a sign that the patient may decompensate.

  - If you are unable to get an ESR, there are other things that you can check. Ferritin and triglycerides are also elevated.

  - How do you treat it? If the macrophage activation syndrome is caused by a rheumatologic disease, the first-line treatment is high dose pulse steroids. Also, supportive care.

  - Does this happen to adults? It can. It may look like sepsis and it can be difficult to distinguish between the two.

  - If a child with lupus or JIA looks really sick, treat for infection and call your rheumatologist for recommendations on treatment of MAS.

- Should we start oral steroids in well-appearing patients with oligoarticular JIA and a flare with normal CRP? This depends on how severe the flare is. If the patient is not taking NSAIDs on a regular basis, the first line treatment is NSAIDs. If it is a severe flare (for example, the patient has difficulty walking or functioning), a short course of steroids is probably safe and effective.

  - Steroids can be given either as a small pulse of methylprednisolone at a dose of 1-2mg/kg via IV in ER or 1-2 mg/kg divided BID or TID for a 3-5 day oral pulse.

  - When should patients be admitted for inpatient management of their flare? If they really can't walk or there is concern for an underlying infection.

  - What are the risks of the various immunomodulators? If the patient is on oral methotrexate or anti-TNF, the degree of im-
munosuppression and subsequent risk of infection is moderate. If the patient is receiving an IV agent such as rituximab or chemotherapy, there is a much higher risk. Rituximab is administered in two doses two weeks apart but the effects last for up to a year.

- **If you have a well-appearing child with a temperature of 103 and some upper respiratory infection symptoms who received rituximab a few weeks ago, what should you do?** Draw labs such as CBC with differential, ESR, CRP and blood culture. If the ESR and CRP are only a little elevated and the child looks ok, it is probably safe to monitor. If the patient is leukopenic or neutropenic, they need antibiotic coverage and a rule out of serious bacterial infection.

- **If the child has a definite source such as otitis media, you can treat with antibiotics.** If patients are on antibiotics, they have to hold their immunosuppressive medications for that week until they are better. Close follow-up is very important. You don’t necessarily have to send labs if they are otherwise well-appearing.

- **What happens when these kids become adults?**
  - In the past, these children had long term bone damage when they became adults and required joint replacements or wheelchairs. Now with the use of biologics, we think we can catch the disease early and treat early to prevent a lot of these long term morbidities.
  - Some of the other diseases such as lupus, dermatomyositis and vasculitis may have other long term consequences.
  - Patients with lupus are at increased risk for atherosclerosis and coronary artery disease.
  - Some children may continue to have active disease as adults or experience remission followed by flares as an adult.

- **Are there any differences in the management of patients with lupus in the ED?**
  - Lupus is a spectrum of disease. It is important to get a good history of their typical lupus symptoms and what medications they are on. Some patients just experience a rash or arthritis and are only on methotrexate. These patients would be treated differently than patients with more severe forms of the disease such as renal failure or who are using cyclophosphamide.
  - If the patient presents with a fever, you should get a blood culture. If they have respiratory symptoms such as chest pain or difficulty breathing, get a chest x-ray as they are at increased risk of pleural effusions or pericardial effusions. They may develop pericarditis and should receive an echocardiogram and EKG if they have symptoms.
  - You need to be very careful to perform a thorough infectious work-up depending on how the labs look, how sick the patient looks and if they are on high dose steroids, IV cyclophosphamide or mycophenolate.

- **Don’t assume that fever is due to a flare.** Fevers are more likely to indicate infection.

- **What should you do with a child who is undiagnosed and has non-specific symptoms such as vague joint pains?**
  - Persistent symptoms are concerning for rheumatologic disease. If there has been no improvement in 3-4 weeks, you should consider a rheumatologic diagnosis. There are a lot of conditions that cause joint pain without swelling that are not usually rheumatologic. If there is true arthritis, you need to consider other diagnoses such as septic arthritis and malignancy.

- **Leukemia can mimic JIA.** Warning signs of leukemia include high inflammatory markers, pain out of proportion to their presentation. Most JIA patients continue their daily activities as usual but the parents notice that joints appears unusual. Any cytopenia on CBC is very atypical for JIA and requires a work-up for malignancy.

- **X-rays may be helpful.** Patients may have a metastatic tumor or mass. Leukemia may show signs on x-ray such as leukemic lines. These are transverse radiolucent lines across the width of the metaphysis.

- **Try to identify the chronicity of the complaint and refer for further assessment.**

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**De-Escalating Agitation**

Rob Orman MD and Daniel McCollum MD

**Take Home Points**

- You can categorize psychiatric patients into DEFCON categories 1-5.
- Aggressively manage substance abuse problems; nicotine patches for smoking, alcohol withdrawal protocol for alcohol abuse, clonidine or pain medication for opiate abuse or chronic pain and benzodiazepines for sympathomimetic abuse.
- Paying attention to patient comfort with warm blankets, phone calls and food can improve patient compliance without medication.

- You are busy repairing a laceration when the nurse notifies you that the police are bringing in an agitated patient in handcuffs. There are no psychiatry beds available. The disposition will take days and not hours. You consider ordering a B-52; intramuscular diphenhydramine, haloperidol and lorazepam even prior to evaluating the patient. **Is there a better way to manage psychiatric patients?** Can we avoid physical restraints and oversedation?
• Many approach psychiatry patients like John Wayne. "Now listen here, I have the right to take away your freedom. If you don't listen to me, I have the right to tie you to the bed and jab you with a medicated needle." What happens? It leads to immediate confrontation. The patient is restrained to the bed, receives a big slug of meds and the disposition drags on as the patient bounces back and forth between oversedation and agitation. What if we made an effort to ally with a select group of patients and address their needs?

• You can classify agitated patients with the DEFCON categorization used by the United States Armed Forces. DEFCON 5 is the usual level of preparedness. DEFCON 1 is nuclear war. Sometimes you can meet the patient's needs with warm blanket and other times you need the B-52 or IM ketamine.

• DEFCON 1. These are the severely agitated patients. You can't reason with them. They are out of control.

  • The police bring in a young man who is handcuffed. He is attempting to kick the several officers who are trying to place him into the exam room. He is yelling obscenities and demanding to be released. There is no name, age or identification other than a history of "psych problems". He was found downtown running in and out of traffic. He has a profusely bleeding laceration on his forehead and won't let you examine it.

  • This is DEFCON 1. There is no time for discussion. This patient must be immediately protected from harming himself or the ED staff. Some will give antipsychotic or benzodiazepine. Some will give the B-52. Others will give midazolam or droperidol if available. Some use ketamine. In dire circumstances, you may have to intubate the patient with RSI to protect the patient.

  • Do things in the interest of the patient and not for your own convenience.

• DEFCON 2.

  • The patient is a 32 year old male with schizophrenia presenting with severe paranoid delusions. He is responding to internal stimuli and having a conversation with himself. He is sitting in the chair and is constantly moving. His personal care home attendant reports that he has refused all medications for a week and is threatening to kill "everyone who ever wronged me". He is starting at the security guard and saying, "You better get that guy away from me."

  • There is a high risk of deterioration to DEFCON 1, especially if you handle the situation like John Wayne. Some of these patients are able to reason and ally with you. Before jumping straight to the B-52, you can say, "You seem a bit anxious. Would you mind if I give you something for your nerves?" A surprising number of patients will say yes. This opens up a more complete pharmaceutical armamentarium for you to use.

  • You can consider atypical agents such as olanzapine or oral medications.

  • A show of force to get the patient into the room can be helpful if they are initially resistant. Humans are proud creatures. If you bring just 1 or 2 security guards into the room and order the patient to do something they don't want to do, part of them will think, "I can take them". The use of a large show of force as gently as possible can have a calming effect.

  • After delivering the medications, seclude the patient in a safe room. If possible, dim the lights and reduce external stimuli. A nice calm environment will give your medications time to work without escalating to DEFCON 1.

  • Don't let your guard down as you may need to escalate if the patient does not respond to your initial interventions.

• DEFCON 3.

  • A 23 year old female with bipolar disorder presents with increased auditory hallucinations. She stopped taking her lithium two weeks ago due to side effects. Her mother brought her in due to increasing behavior problems at home. She is mildly uncomfortable during your history and physical exam. She is fidgeting in her chair and appears to be worried about the security guard a few feet away.

  • This is the sweet spot for this approach. These patients have a risk of deteriorating to a worse DEFCON level but are often rational enough to respond to efforts to ally with them.

  • "You seem anxious. Would you like something for your nerves?" Oral lorazepam is a good option. Some of the atypical oral agents such as olanzapine may be effective but can take longer to work. This should be tailored to the individual patient and you may have time for a quick chart review to see what has worked in the past.

  • This group needs aggressive management of substance abuse problems. If they smoke, replace their nicotine deficit with patches or gum. This is not the time for people to quit smoking. If they are a heavy drinker, starting an alcohol withdrawal protocol with early use of benzodiazepines is a great idea. If they are using illicit drugs, consider adjunctive therapies such clonidine for withdrawal symptoms or benzodiazepines for sympathomimetic abuse. Use oral opiates such as oxycodone if they have a medical indication so that their pain is treated without withdrawal.

• DEFCON 4.

  • A 63 year old alcoholic man is brought in by his ex-wife. While drinking earlier in the evening, he called his ex-wife and said that he wished "it would all end. Maybe I should just kill myself." She was concerned enough to call 911 and the police brought him in. He states his last drink was 6 hours ago and he drinks 1 to 2 pints per day. He is reasonably coopera-
These patients can be very challenging. They have gone from being completely free to do what they want to being held against their will. They are very upset and feel victimized. These are among the most common psychiatric patients in the emergency department. They are often brought in by the police.

"You seem anxious. Would you like something for your nerves?" Oral lorazepam is a good option in this situation. The big change for these patients is to address concerns about comfort. These patients are rarely dispositioned quickly. Many of them stay in the ED for many hours up to several days. How can we keep the peace with the patient for that long?

"We both want the same thing: to get you home as soon as safely possible." Pass the buck and blame the law for keeping the patient against their will. "I'm required by the law to keep you due to the report of you being suicidal. I'm legally not allowed to discharge you yet."

Substance abuse issues often need to be addressed. Make sure the patient is on their home medications including drugs for their general medical problems.

Make them as cozy as possible. A nice blanket straight from the warmer or delicious turkey sandwich is worth 2mg of lorazepam. Allowing the calm patient to make a phone call is better than any dose of diphenhydramine. Have some empathy for these patients and realize how much it must suck to be in a strange ED for over 24 hours. Make their stay as pleasant as possible. Does this encourage patients to return to the ED?

Make them feel like the ED is a place they can turn to when they need help. Make sure they feel welcome and not like a burden. Warm blankets, turkey sandwiches, nicotine patches and caring words. You don't want to see the same patient a week later with a fatal overdose.

Many of us are pressured to see a large number of patients in an ED that is bursting at the seams. Imagine if these patients were presenting with a urinary tract infection rather than psychiatric complaints. DEFCON 1 is urosepsis requiring an ICU admission. DEFCON 5 is simple cystitis. If the patient has to spend 24 hours in the ED because of a simple UTI, you would do everything possible to make them as comfortable as possible. Why would it be any different for our psychiatric patients? Consider how you would like to be treated or your family member to be treated.

The LIN Session: THAM
Michelle Lin MD and Curtis Geier PharmD

Take Home Points

- THAM or tromethamine is a weak base that accepts free hydrogen ions. It can be used to scavenge acid in severely acidotic patients such as after respiratory arrest.

- Dosing may be calculated using a formula or by giving a bolus of 250mL followed by an additional 250mL administered slowly.

- Bicarbonate requires adequate ventilation to correct acidemia.

- The supporting data is fairly limited and mainly case series.

- THAM or tromethamine is a base for a number of drugs used every day such as ketorolac. It can be used on its own as well. It is a weak base that will accept free hydrogen ions. In an acidic patient, it can scavenge some of the acid.

- A patient took pain medication and then went into a respiratory PEA arrest. The patient was resuscitated with CPR. When ROSC was achieved, a blood gas showed the pH was 6.8. He had a pulse but was prone to dysrhythmias and hypotension. He was

- The outcome of mental illness in people that are appropriately treated prior to advancing to more severe stages is quite good. The majority of these patients can be successful-ly treated as outpatients. The worst thing you can do is make them feel alienated or not welcome.

- These patients often come in voluntarily because things are out of control. They often don't have an emergency yet and often don't require involuntary holds at this time. They do need help. Making them feel like the ED isn't a safety net if they worsen is the worst thing you can do. Burning a bridge with them by being hostile or less than welcoming is a mistake. These patients often take many hours to disposition.

- DEFCON 5 is simple cystitis. If the patient has to spend 24 hours in the ED because of a simple UTI, you would do everything possible to make them as comfortable as possible. Why would it be any different for our psychiatric patients? Consider how you would like to be treated or your family member to be treated.

- DEFCON 5. A 42 year old mother of three with a long history of depression presents to the ED. She has felt increasingly depressed and feels that things are getting out of control. She brought herself to the ED due to thoughts of hurting herself. "But I would never hurt myself because I have to care of my kids." She is currently taking citalopram but feels like it isn't working anymore. She feels increasingly anxious recently. She is well groomed and very cooperative during the exam but repeatedly states "I don't want to be here. When can I go?" He has a mild hand tremor and appears to be very frustrated.

- These patients can be very challenging. They have gone from being completely free to do what they want to being held against their will. They are very upset and feel victimized. These are among the most common psychiatric patients in the emergency department. They are often brought in by the police.

- "You seem anxious. Would you like something for your nerves?" Oral lorazepam is a good option in this situation. The big change for these patients is to address concerns about comfort. These patients are rarely dispositioned quickly. Many of them stay in the ED for many hours up to several days. How can we keep the peace with the patient for that long?

- "We both want the same thing: to get you home as soon as safely possible." Pass the buck and blame the law for keeping the patient against their will. "I'm required by the law to keep you due to the report of you being suicidal. I'm legally not allowed to discharge you yet."

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given a 250mL bolus of THAM and then the rest of the 500mL bottle was infused over the remaining 30 minutes. A repeat blood gas 15 minutes later showed correction of his pH to 7.4.

- **Some people would reach for bicarbonate in this situation.**  
  **How does bicarbonate correct acidemia?** Bicarbonate gets converted to CO₂ and H₂O. The CO₂ has to get blown off. If the CO₂ does not get exhaled, you will not see a change in the serum pH. Often these patients can’t blow off the excess CO₂. Patients who just arrested will have a high CO₂ from apnea and retention, so increasing the CO₂ won’t be helpful. If you think about patients with severe acidemia such as in DKA or aspirin overdose, they are already maximally hyperventilating. You can’t blow off more CO₂ unless you intubate them and hyperventilate them.

- **THAM binds the hydrogen ions and this is exclusively excreted renally.** You aren’t relying on the patient’s ability to get rid of the CO₂. THAM can scavenge carbon dioxide. It is almost the perfect agent in a patient with a mixed respiratory and metabolic arrest.

- **How quickly can this be administered?** It is easy to administer as long as it is available. It is kept in the emergency department at SF General for this situation.

- **How much does it cost?** About $200 a dose. This is considerably more expensive than bicarbonate.

- **Why isn’t it used more often?** The supporting data is controversial. It has never been shown that treating an acidosis without addressing the underlying cause has improved outcomes. This holds true for both THAM and sodium bicarbonate.

- **This is not a cure-all.** It is not going to fix your septic, acidemic patient. You will have to treat the underlying pathophysiology.

- **It has a more limited list of indications approved by the FDA.** It is FDA approved for metabolic acidosis associated with cardiac arrest or correction of acidity of blood in cardiac bypass surgery. This will be a targeted tool in a unique patient population.

- **The data in humans is fairly limited to case series and case reports.** There is one good study that included 18 patients.

- **This study enrolled ICU patients with mild metabolic acidosis defined as a serum bicarbonate less than 20 mmol/L and randomized them to bicarbonate or THAM at equivalent doses.**

- **They found that both agents were equally effective in correcting the acidosis but THAM had some unique features.** It seemed to decrease serum sodium. This may be due to the volume load. Sodium bicarbonate increases the serum sodium.

- **THAM did not affect potassium.** It may not be the best option in patients with hyperkalemia as sodium bicarbonate decreases the potassium level.


- **Can THAM be given to any cardiac arrest patient?**
  - It can be considered in these patients. THAM will work in an open or closed system. If the patient is ventilated, the removal of CO₂ should not be an issue. Sodium bicarbonate may work as an effective buffer in the ventilated patient but it can freely pass across cell membranes and can increase intracellular carbon dioxide and create an intracellular acidosis. THAM may not be necessary in the ventilated patient.

  - If you have a patient who is not intubated or who has a component of respiratory acidosis, THAM would be an appropriate treatment. It is approved for the treatment of metabolic acidosis after cardiac arrest.

- **What is the correct dosing?**
  - The approved dosing is calculated via the formula: THAM solution (mL of 0.3M) required = body weight (kg) x base deficit (mEq/L) x 1.1.
  - If you are treating a patient with severe metabolic acidosis, the dosing would be in the range of 300-600mL. You can start with a bolus of 250 mL and slowly administer the remaining 250mL taking care to not overcorrect the acidosis.

- **What are the adverse effects of this?** You may depress the patient’s ventilation by binding carbon dioxide, which decreases the respiratory rate of the patient. Make sure you don’t overcorrect the acidosis. It can be a venous irritant so use a good IV site if possible. There are cases of hypoglycemia but this is usually associated with large doses or continuous infusion over a long period of time.

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**C-Spine Clearance in the Obtunded**

Mel Herbert MD, Scott Weingart MD, Billy Mallon MD, Peter Viccellio MD, Ian Stiell MD, Kenji Inaba MD, and Anand Swaminathan MD

**Take Home Point**

- Patients who are altered secondary to alcohol intoxication with a negative CT scan of the cervical spine do not require an MRI for clearance unless they are symptomatic upon reassessment, have a concerning mechanism of injury or focal neuro deficits.

- If a patient is altered (such as alcohol intoxication) and has an adequate CT that is normal, do they need an MRI to exclude ligamentous injury before removing the collar? MRI access may be limited in most facilities. Although it is rare, ligamentous injuries can be present despite normal CT scan. Leaving the C-collar on in the patient can also be problematic. As the patient wakes up and becomes more agitated, they may start flopping around on the bed and you aren’t protecting the neck. Why not just take the C-collar off?
The Eastern Association of Trauma did a nice review earlier this year.

- **What are their recommendations?** “In obtunded adult blunt trauma patients, we conditionally recommend cervical collar removal after a negative high-quality C-spine CT scan result alone.”


Anand Swaminathan.

- The best thing to do is to wait until they wake up and are sober. Re-examine them. If they don’t have any midline tenderness, you are done.

- If the patient has tenderness to palpation with a normal neurologic examination, they are discharged in a collar with two week follow-up for flexion/extension films or MRI if they still have midline tenderness. Most of the patients will discontinue the collar on their own and not return for follow-up.

- If the patient has a focal neurologic deficit, they need an MRI.

Peter Viccellio.

- You would get an MRI in a sober patient if they had sustained a stinger injury or transient bilateral symptoms. Wait until they wake up and re-examine them.

Billy Mallon.

- Getting an MRI in all of these patients takes time, costs money and will destroy the emergency department. Don’t do it. Stick them in an Aspen collar if they still have pain when they wake up and have them follow-up in a week.

- It depends on the mechanism of injury. If the patient was in a low speed car accident without pain initially and then later had midline pain despite negative CT, the patient is low risk and doesn’t need an MRI. If a kid dove into a pool and whacked their head on the bottom with a negative CT but persistent pain upon sobering, they are high-risk and should get an MRI.

Ian Stiell.

- Use common sense and clinical judgment. Look at your pre-test probability and make your decision based on that.

Scott Weingart.

- When the patient wakes up, examine them. Weingart uses the Canadian C-spine instrument. He feels for midline tenderness. Don’t ask them if it hurts because they always say yes. Just press on each vertebra and observe their reaction. Do patients with tenderness or inability to range their neck need an MRI? We don’t know. This group has a higher pre-test probability. Some places will still clear them after negative CT scan and others will get an MRI.

Peter Viccellio.

- Most would agree that a small subset of patients with a normal neurologic exam and persistent neck pain need an MRI. We can exclude many patients based on our history and the absence of severe pain.

- If it is just alcohol intoxication with a head bonk and negative CT, wait until they wake up and re-examine them. If the patient has severe, intractable pain or a concerning mechanism, you need to do something more, like an MRI or C-collar with follow-up.

Annals of Emergency Medicine

Paul Jhun MD and Clare Roepke MD

**Take Home Points**

- Abdominal compartment syndrome is the development of organ dysfunction at high intra-abdominal pressures.

- Intra-abdominal pressure (IAP) can be measured in the emergency department using a Foley catheter.

- The definitive management of compartment syndrome is surgical decompression but decompression of air or ascites in the ED may be necessary as a temporizing measure.
CASE
A 78 year old male with a history of Ogilvie’s Syndrome and congestive heart failure presented to the emergency department with four days of abdominal pain, constipation, abdominal distension and shortness of breath. Vital signs were significant for a blood pressure of 71/31 mmHg and oxygen saturation of 90% on room air. Abdominal exam was significant for a tender, distended and firm abdomen. A portable single view chest x-ray was obtained and showed cardiomegaly and mild pulmonary vascular congestion without free air under the diaphragm. A portable abdominal x-ray was also obtained and demonstrated massively dilated loops of colon in the mid-abdomen. Despite aggressive fluid resuscitation, the patient remained hypotensive. He was taken emergently for CT scan, which demonstrated massive pneumoperitoneum consistent with hollow viscus perforation with compression of intra-abdominal contents by free air. Diagnosis: tension pneumoperitoneum.

- This is a rare diagnosis.

- The abdomen, just like the brain, is contained in a closed, finite space with little room for rapid expansion. The skin can only stretch so much so fast. A rapidly expanding process that squishes the intra-abdominal viscera may decrease abdominal perfusion pressure (APP). The APP = MAP – IAP (intra-abdominal pressure).

- What conditions can increase the IAP? The IAP rises either by external compression of the bowels (from massive volume in the abdomen such as third-spacing of fluids after burns, aggressive fluid resuscitation, rapidly accumulating gas) or increased pressure in the bowels themselves.

- Abdominal compartment syndrome is the development of organ dysfunction at high IAPs (generally greater than 20-25 mmHg). This leads to intra-abdominal organ failure and can be life-threatening with mortality as high as 60-70%.

- What red flags can alert you to abdominal compartment syndrome?
  o These patients are sick. They are not asking you for a sack lunch. It is most often found in critically ill patients who are in shock, badly burned or have sustained polytrauma.
  o In addition to a tense and distended abdomen, the force on the diaphragm can mechanically compress the lungs causing dyspnea and possibly hypoxia in awake patients or high peak airway pressures in intubated patients.
  o Pressure on the IVC impairs cardiac return and output. Patients may have hypotension.
  o Decreased perfusion and direct compression of organs can lead to oliguria or renal failure. The intestines may have decreased mesenteric perfusion.
  o How can you measure abdominal compartment pressures?
    o The gold standard is via the bladder. Use a Foley catheter with a pressure monitor and an arterial line kit. This can be quickly and easily performed at the bedside.
    o Insert the Foley into a flat and supine patient, drain the bladder fully and clamp the Foley tubing. Insert 25cc of sterile water into the side port (this is commonly used as a port to flush the Foley) and clamp the side port (to keep the fluid in the bladder). Hook the Foley up to the pressure transducer and connect it to the arterial line. Zero the pressure to the level of the bladder.
    o You can’t do continuous monitoring. You need to unclamp the side port and Foley tubing to allow the urine to drain.
  o What do you do if you can’t find or don’t have an arterial line kit or special Foley? Keep the patient supine and flat. Insert the Foley. Drain the urine. Instill the 25cc saline into the side port and clamp that. Raise the Foley tubing up in the air perpendicular to the patient, unclamp the tubing and measure how far the fluid rises against gravity. Measure the distance in centimeters from the bladder to the fluid meniscus in the tubing and convert the cm to mmHg. 1mmHg is equal to 1.36 cm H2O and you can find calculators online.
  o Consult surgery for immediate decompression.
    o While waiting for the surgeon, you can place an NG tube and Foley catheter to decompress the stomach and bladder. If the patient is intubated, sedate them and consider paralysis to decrease agitation and increase abdominal wall compliance.
    o The standard of care is surgical decompression (usually via an exploratory laparotomy) but abdominal drain placement can be considered in select patient populations or extenuating circumstances. This is less effective.
    o If the surgeon is not available, alleviating pressure with drainage of ascites is not unreasonable. Repeat the measurement of IAP. This is temporizing measure however. Exploratory laparotomy is the definitive intervention.

CASE CONTINUED
The patient had continued hypotension and respiratory distress. He underwent needle decompression with transient improvement in hemodynamics and improved work of breathing. After stabilization, surgical exploration identified colonic perforation at the level of the splenic flexure.

- The patient had a history of Ogilvie’s syndrome which is an acute dilatation of the colon in the absence of an anatomic lesion. This may have been a contributing factor.
Extensor Tendon Injury
Mizuho Spangler DO and Brian Lin MD

Take Home Points

- Injuries to the central slip of the extensor tendon over the proximal IP joint may be difficult to diagnose and result in a lot of morbidity.
- The modified Elson's test can be used to identify these injuries.
- Brian Lin MD is the founder of the website lacerationrepair.com.
- A 30 year old female who was right hand dominant presented with a laceration to her right hand. She put up her hand to defend herself and sustained a stellate laceration over the extensor surface of the proximal interphalangeal joint of the index finger on her right hand. The laceration was 2 cm and deep over the second knuckle.
- You don't want to miss extensor tendon injuries. These are a common orthopedic injury. They tend to occur in young, healthy working age patients. The extensor tendons are very superficial and thus more susceptible to injury. They are also accessible for primary repair. Minor and partial extensor tendon injuries are under the jurisdiction of emergency medicine.
- How can you recognize extensor tendon injuries?
  - Always make sure that you examine the wound after it has been properly anesthetized, irrigate to remove any dirt and look at the wound in a bloodless field. Use a tourniquet as needed. Ensure good lighting.
  - Make sure you see movement of the joints around that injury through a full range of motion to identify an injury that might not be obvious by staring at the wound. The cut ends of a tendon may retract away from the margins of the wound. Move the joint into the position that will allow you to see a tendon injury.
  - Make sure to examine the patient in the position the injury happened as this may be your best chance to see the tendon injury.
- You are still at risk of missing tendon injuries in certain special situations such as injuries to the extensor tendon over the proximal interphalangeal joint. The extensor tendon at this level is not one simple tendon but a complicated network of tendons. It divides into three separate parts as it traverses the proximal interphalangeal joint. This includes two lateral bands which are held on the dorsal aspect of the hand by a single central slip. This is thin and prone to injury. If it is lacerated, it can be difficult to detect by primary inspection of the wound.
- A specialized test to detect this was developed by Elson. This is commonly used to diagnosis a central slip injury. Have the patient take the injured finger and rest their hand on a flat surface and bend their second knuckle at the bend of a 90 degree surface. Apply pressure to that middle phalanx and ask them to try to extend their finger. If they have a central slip injury, there is weakened extension of the finger at the proximal interphalangeal joint. You will also notice that the distal phalanx hyperextends. Why? The lateral bands migrate in volar direction to the flexor surface and act as extensors at the distal interphalangeal joint. The lateral bands act like a buttonhole and the head of the proximal phalanx pushes through the buttonhole.
  - What happens if you don't diagnose this injury? This can lead to a Boutonniere deformity. It is often described in rheumatoid arthritis or Ehlers-Danlos syndrome. It takes about two or three weeks for the chronic deformity to develop. Once it develops, it is difficult for the hand surgeon to reverse it.
- The modified Elson's test.
  - This is easy to do. It doesn't require you to remember rules. Take the injured finger and have the patient make a shape like a fish hook (extension at the metacarpal-phalangeal joint and flexion at the phalangeal joints). Have them make the same shape on the opposite hand. The patient presses the middle phalanges of their fingers together. Ask them to try to extend their middle phalanges. Look for asymmetry between the two fingertips. They should be symmetric like an isosceles triangle. If there is an injury to the central band, you will see inappropriate extension of the distal phalanx and it will appear asymmetrical.
- In our case, the patient had an abnormal modified Elson's test. She had a central slip injury. The patient was splinted in extension and referred to the hand specialist.