Introduction: Spinal Injuries
Rob Orman MD and Anand Swaminathan MD

Highlights

- Complicated skin avulsions in elderly patients may be repaired by using Steri strips to stabilize and suturing through the Steri strips.
- Patients with trauma and persistent midline cervical spinal tenderness despite negative CT imaging may be managed by obtaining a MRI within 72 hours, continuing C-spine immobilization until resolution of pain, or continuing C-spine immobilization with flexion-extension imaging.
- Most vertebral compression fractures are managed non-operatively. TLSO braces may decrease pain and improve quality of life but are controversial.

CASE

It was a busy day in the Emergency Department with multiple traumas and a STEMI. A patient presented with a laceration. She was an elderly woman and had a skin tear to her shin with approximately 50% of the skin avulsed. It was massive.

- These injuries take a long time to repair correctly. This is stressful, especially in situations with single physician coverage. There was no plastic surgeon available.
- Orman remembered the Lin Sessions segment on wound repair. The wound was repaired with Steri strips on each side and sutures. The patient was referred to the surgeon and placed in a walking boot for several weeks. It healed perfectly.

CASE

In the next room, another patient had a spinal fracture.

- How do you clear the C-spine in a trauma patient who is awake, neurologically intact with a negative C-spine CT but still has midline tenderness? There are some available guidelines. The Eastern Association for the Surgery of Trauma (EAST) has published guidelines that provide the option of clearing the C-spine in the non-awake trauma patient based on the CT scan alone. CT scan picks up the vast majority of these diagnoses but may miss ligamentous injuries, subluxations, or dislocations. There are several options:
  1. Get an MRI within 72 hours of the injury. There is not a lot of available evidence to support this as the preferred option. However, if the patient is neurologically intact, they don’t necessarily need an emergent MRI. Many patients with neck pain will have spontaneous resolution of their pain with time.
  2. Continue C-spine immobilization until they no longer have midline neck pain. Patients may be discharged in a collar such as a Miami-J or Aspen collar. Who should the patient follow-up with? A surgeon or primary care doctor? Neurosurgery is a good option for follow-up. Prolonged wearing of collars may result in skin breakdown and ulcers. It is uncomfortable.
  3. Continue C-spine immobilization and obtain flexion-extension imaging. When do you perform this imaging? Most recommend imaging in two weeks. However, patients rarely wear the collar for the entire two weeks. Does the collar even help? Does it improve outcome?

- How do you manage vertebral compression fractures in elderly patients? There are about 1.5 million of these injuries every year. They are typically seen in elderly patients with osteoporosis or malignancy. Most patients are treated non-surgically with bed rest or hyperextension bracing. Neurosurgical intervention is rare and usually due to retropulsion of bone into the vertebral canal. If there are neurologic symptoms, neurosurgery needs to be involved quickly. Retropulsion of the bone will likely be identified on CT scan and you don’t necessarily need an MRI to make this diagnosis.
  1. If the patient does not have neurologic symptoms, avoid bed rest. Make sure they have good pulmonary toilet and they are using an incentive spirometer. Patients should receive adequate pain control. NSAIDs are first line but you will likely need to escalate to narcotics. However, narcotics may increase the risk of falls and may be difficult to titrate. Patients should be referred to physical therapy early. They need exercises to strengthen their extra-axial muscles.
  2. If the patient needs a lot of pain medication, they may need to be admitted. Have a low threshold for admission or observation. These patients may be debilitated at baseline and are at risk for multiple complications.
• You can get a consult to obtain a TLSO or thoracolumbosacral orthosis brace, although this is controversial.
  
  o A randomized controlled trial in 2004 showed improved quality of life, decreased pain, and improved daily function. 
  
  o However, other studies using electromyography showed an increase in muscle spasm, but it was not a patient-centered outcome.
  
  o These can also lead to skin breakdown so be careful.

Make sure the patient is able to ambulate and perform his/her activities of daily living prior to discharge.

Aortic Dissection
Rob Orman MD and Amal Mattu MD

Highlights
• There are no good clinical decision rules to identify patients at very low risk for thoracic aortic dissection.
• D-dimer is not sufficiently sensitive to rule out aortic dissection.
• CTA is equivalent to TEE and MRA in identifying dissection.
• Transthoracic echocardiogram is unable to definitively rule in or rule out aortic dissection.
• Control of heart rate and blood pressure is likely beneficial but there is no literature determining the best target.

We frequently talk about acute coronary syndrome and pulmonary embolism, which may be difficult to diagnose due to varied presentations, but the great mimic is aortic dissection. This may be a challenging diagnosis to make. ACEP has just published a clinical policy on the evaluation and management of patients with suspected aortic dissection.


• In adult patients with suspected acute nontraumatic thoracic aortic dissection, are there clinical decision rules that identify a group of patients at very low risk for thoracic aortic dissection? No. This is a very rare condition, so it is difficult to create a decision rule that is useful. However, we should be aware of high-risk predisposing conditions and features.

• High-risk predisposing conditions include: Marfan syndrome, family history of aortic disease, known aortic valve disease, recent aortic manipulation, or known thoracic aneurysm.

• High-risk pain features include: chest, back, or abdominal pain that is abrupt in onset and severe, ripping, and tearing.

• High-risk exam features are pulse deficits, focal neurologic deficits with chest pain, and chest pain and aortic insufficiency or syncope. Although this study identified systolic blood pressure differential as a high-risk feature, this finding has been studied and found to be poorly sensitive and specific. 15-20% of normal patients will have a blood pressure differential of at least 20 mmHg. This is an extrapolation of original data that focused on pulse differential. Focus on checking pulses; if you feel a palpable pulse differential, you should be worried.

• In adult patients with suspected acute nontraumatic thoracic aortic dissection, is a negative serum D-dimer sufficient to identify a group of patients at very low risk for the diagnosis of thoracic aortic dissection? No. Patients with dissection have intramural clot and early studies suggested that the sensitivity of a positive D-dimer was in the upper 90%. This was then extrapolated to say that a negative D-dimer rules out an aortic dissection. However, subsequent studies show that the sensitivity of D-dimer is closer to 90%, and this is not sufficiently sensitive for such a deadly disease.

• In adult patients with suspected dissection, does an abnormal bedside transthoracic echocardiogram (TTE) establish the diagnosis of thoracic aortic dissection? You can't definitively rule in or rule out dissection with a transthoracic echocardiogram. However, if you do get an echocardiogram and are concerned for a dissection, you should get an immediate surgery consult or arrange transfer to a higher level of care.

• In adult patients with acute nontraumatic thoracic aortic dissection, does targeted heart rate and blood pressure lowering reduce morbidity or mortality? It is probably beneficial. However, there are no definite targets that have ever been successfully studied. There is no good data to suggest a preferred strategy.

• Patients don't die due to the initial dissection but rather due to propagation causing complications such as tamponade, aortic valve insufficiency with cardiogenic shock, or aortic valve rupture. The two major factors associated with progression of the dissection flap are the shear forces and pulsatile flow.
It seems reasonable that if you decrease the blood pressure and heart rate that you will decrease the chance of progression and mortality. However, this has never really been studied and we only have consensus recommendations that advise targeting a heart rate around 60 beats per minute and a systolic blood pressure under 120 mmHg.

In 1962, Everett Rogers proposed the law of diffusion of innovation. Patients can be stratified with a bell-shaped curve. To the left are the innovators (the first 2.5%), then the early adopters (13.5%), then the early majority (34%), and the late majority (34%). Finally, are the laggards (16%). This was designed to evaluate bringing products to market, and they had to reach the early majority before a product was successfully marketed. However, it can also evaluate how physicians practice. Sacchetti tends to be an early adopter or early majority. His department has over 100 journal publications, many of which are early reports on what are now considered mainstream therapies. Their approaches to problems are frequently at odds with published reports and opinion. The late adopters tend to include practitioners who are hesitant to incorporate their clinical experiences into the decision-making process. It can be frustrating when clinicians are unwilling to consider a new therapy until there is good evidence-based medicine support for its use. Why should we wait for years of accumulated data and for someone else to tell us how to practice?

We need to strike a balance between what the literature or authorities state and what happens in our department. There are a number of well-designed, evidence-based medicine studies that are wrong because they measured the wrong parameters or sampled the wrong group.

If you compare published reports against personal clinical experience, you will always be subject to confirmation bias. Confirmation bias is when a specific outcome confirms your belief about a study process. For example, if you believe that intranasal lidocaine therapy works to treat migraine headaches, every time you use it you will expect it to work. If it does, it confirms your belief. If it doesn’t work, you tend to discount those cases. However, if you look at objective outcomes, you can mitigate the effects of confirmation bias. Sacchetti didn’t need a randomized controlled trial to show that insertion of a wire-guided pigtail catheter led to re-inflation of the lung or that central line placement was more successful and safer using ultrasound guidance.

There are soft endpoints where confirmation bias is a risk. Do wounds that are closed with fast-absorbing gut really look as good as those closed with nylon? Are patients with renal colic really more comfortable with ketorolac? These are cases where Sacchetti might lean more heavily on the outcomes of clinical trials to guide management, although he maintains some skepticism.

There are some cases where the patients are not followed long enough to know the outcome. Medical expulsive therapy seemed like a good idea for kidney stones, but a prospective study showed that it didn’t work.

If Sacchetti hears an authoritative or evidence-based source describe something at complete odds with his clinical experience, he re-examines his perception of his clinical experience. He either admits he is wrong or continues with the knowledge...
that his current approach is better in his hands. If an innovative solution arises with scientific merit, he will give it a try. If it outperforms his current solution, he will stick with it; otherwise, he will go back to his standby. In both cases, he uses his existing experiences as the basis of his decisions, with the understanding that he needs to be aware of the possibility of confirmation bias.

Lessons from Quality Review
Reuben Strayer MD

Highlights
- Always expose the patient prior to exam.
- Pay attention to the vital signs, especially persistent unexplained tachycardia.
- Review all test results prior to discharge.
- Don’t be afraid to do a higher resource work-up in a patient that is difficult to evaluate.

- More often than not, review of cases identifies a systems problem that can benefit all.
- What issues are repeat offenders? Expose the patient. Be wary of discharging patients with tachycardia. Review results before discharge. Don’t be afraid to do a higher resource work-up. Don’t look at just the immediate problem; why did this problem happen today?

- Expose the patient.

CASE
A 75-year-old female presented with abdominal pain and vomiting. The evaluation was unremarkable. The patient looked fine and they planned to send her home. However, the son arrived and said that she was not right and they needed to do more testing. A CT scan was ordered and diagnosed an incarcerated inguinal hernia. The diagnosis was made more than 12 hours after she arrived. Her outcome was poor. The inguinal hernia was very apparent on exam once they looked. However, they never disrobed the patient on initial assessment.

- If you are looking for ways to reduce errors, have technicians or nurses fully disrobe and gown the patient who presents with a non-isolated extremity complaint, prior to evaluation by a doctor.

- Vital signs are vital. Respiratory rate is probably the most important, but because they are never measured accurately, we have learned to do our jobs without them. The biggest red flag is persistent tachycardia. You can discharge patients with persistent tachycardia but do it reluctantly, carefully, deliberately, and with an explanation.

CASE
A healthy 31-year-old female presented with nausea, vomiting, and abdominal pain. Her abdominal tenderness was distractible. She received symptomatic treatment and felt better. Blood work ordered from triage was normal. She was discharged. She returned two days later extremely ill from perforated appendicitis. This situation happens occasionally. However, her previous differential showed 26% bands, which may have raised the suspicion of a more serious etiology had it been noted prior to discharge. It was missed, as the differential had resulted after the blood count.

- You are responsible for the tests. Review the results prior to discharge.

- Perform a higher resource work-up when your assessment is limited. If you are unable to assess the patient as you normally do, perform a higher resource work-up. The terms “high resource” and “low resource” are unambiguous and are better terms than “conservative versus aggressive” treatment. What patients may be difficult to assess? The very young and the very old. Patients who are intoxicated or have a brain problem. Limited physical exam. Language or cultural barriers. Sometimes you don’t have the time to properly assess the patient. In these cases, you need to do more tests, consider more observation, and have a lower threshold to admit. You can give the patient your phone number if you are comfortable with this.

CASE
A resident repaired a laceration and sent the patient home. He wasn’t entirely comfortable with the repair, so he gave the patient his business card. The patient contacted him several days later, as she was concerned that she had developed drainage and discoloration of the finger. He was able to arrange appropriate hand surgery follow-up for the patient.
Repeat the ECG.

CASE
A 39-year-old healthy male presented to the Emergency Department at 0045am. He had no medical history but presented with a reasonable story of chest pain. He looked okay but not great. The ECG wasn’t diagnostic but still worrisome. It wasn’t clear if he was having a STEMI. 9 minutes later, a repeat ECG showed a clear STEMI.

- Repeat the ECG in 5 to 10 minutes. Don’t repeat the ECG in an hour; time is myocardium.

“Never carry bling through gloomy Antwerp.” What do you need to do in cardiac arrest? The six therapies you need to remember when taking care of a cardiac arrest patient:

- Needle: needle the lung or heart
- Calcium: hyperkalemia is probably the most important and treatable cause
- Blood: give blood or fluids
- Thrombolytics: give thrombolytics for massive PE
- Glucose
- Antidote

You don’t need to be reminded to shock ventricular fibrillation or tachycardia. You don’t need to be reminded to intubate the patient. This will solve the less obvious problems that cause cardiac arrest. You don’t need to be reminded to give epinephrine; this will treat anaphylaxis. If the patient is dead due to pump failure, there is not much you can do, aside from giving catecholamines or initiating ECMO. If the patient is really hot or really cold, you will identify it and treat it. If the patient is dead from sepsis, an aortic or intracranial catastrophe, you aren’t going to bring them back.

Patients and doctors love to attribute patients’ symptoms to what they just ate. We usually eat three or more times a day; for every symptom, including getting hit by a bus, there was a meal prior. The meal rarely causes the symptom, unless multiple people have the same symptoms after eating the same meal.

- Avoid benign specific diagnoses. For example, avoid gastroenteritis for abdominal pain NOS, avoid migraine for headache NOS, and avoid costochondritis for chest pain NOS. The patient above, who had perforated appendicitis, was diagnosed with gastroenteritis on his index visit. When you tell a patient that they have a benign condition, some of them will believe you; when you are wrong, they will not come back. “Sir, I’m not sure exactly what is causing your abdominal pain. Based on my evaluation, after my assessment, I am reassured that it is not serious and will get better on its own. However, there is a chance that I am wrong because sometimes it is too early and we can’t see it. So, if you get sicker, you need to come back.” If you know for sure it is a benign specific condition, you can tell the patient. If you don’t know for sure (and we never know for sure with headache, chest pain, and abdominal pain), don’t pretend you know for sure.

Clinical Sobriety
Matt Delaney MD, Sam Ashoo MD, Greg Henry MD and Anand Swaminathan MD

Highlights
- There is a great deal of variability in management of drunk patients by physicians.
- We are not good at determining clinical sobriety or blood alcohol levels.
- Document that the patient is eating, drinking, walking and appears to be making appropriate decisions and make sure this corresponds with nursing documentation.
- Waiting for the alcohol level to fall to below legal limits can be dangerous in chronic drinkers as it may precipitate withdrawal when they reach sober levels.

Delaney
- Drunk patients can sometimes be a source of entertainment or a soul-crushing resource drain that will bring an Emergency Department to a screeching halt. These are risky patients; they do dangerous things and hide dangerous diseases. There is a lot of variability in the practice of how we deal with these patients. How do we figure out who is drunk and who is sober?
- There are some providers who designate a patient as clinically sober, while others get a blood alcohol level to determine sobriety. There is very little literature to suggest that a clinical approach to sobriety is any better than getting labs. Both of these practice patterns have significant limitations and carry a certain amount of medical risk.
- There is fairly good support in the medical and legal literature for the idea of clinical sobriety. What does this consist of? You look for obvious signs of psychomotor and cognitive impairment and make your best guess. However, it is often unclear who is clinically sober, and we are not good at determining clinical sobriety.
They found that we tend to overestimate the blood alcohol level in patients who have had some alcohol but are not intoxicated. However, the physicians were not asked if the patient was intoxicated or sober, but to identify if the blood alcohol level was over or under 80 mg/dL. There is a lot of variability at the lower end of the spectrum. Alcoholics weren’t excluded. Laboratory determination of BAL does not reflect the extreme individual variability of the effects of alcohol on the population.

- We are not good at guessing the blood alcohol level of patients.

They found that we tend to overestimate the blood alcohol level in patients who appear intoxicated but are not chronic drinkers. We tend to underestimate the BAL in patients who are chronic drinkers. They found poor correlation between the BAL and visible signs of intoxication, across all ranges of blood alcohol level.

- There probably isn’t a huge risk in assuming a patient is more intoxicated than they are. If the patient seems intoxicated, check on them frequently and make sure they are sobering appropriately.
- Document clinical sobriety such as eating, drinking, walking, and appearing to be making appropriate decisions. Check the nursing notes and make sure that you are on the same page. You don’t want to discharge a patient when the nursing notes say the patient is altered with an ataxic, unsteady gait.
- If you are going to get blood alcohol concentrations, don’t ignore a high level. In the United States, all states consider someone to be impaired if their blood alcohol level is greater than 80 mg/dL or 0.08. From a legal perspective, a patient is legally intoxicated above this level, despite their clinical appearance. There is little legal support for the idea that a person could be clinically sober while having an elevated blood alcohol level.
- We see patients who are alcoholics and who present with very high blood alcohol levels, but seem totally fine. From a legal standpoint, this is a shady area. The risk comes from ignoring the elevated blood alcohol concentration.
- Some will observe non-chronic drinkers who are intoxicated for a few hours. You don’t necessarily need to repeat a level to prove they are sober. You do need to watch them for a reasonable amount of time. It is thought that non-chronic drinkers will metabolize ethanol at a rate of 20mg/dL/hr. A patient with a blood level of 200mg/dL may need 5-6 hours of observation until their blood alcohol level drops below the limit of 80 mg/dL.
- This is more difficult in patients who are chronic drinkers. Their metabolism is different than a non-drinker. These patients may not plan to get sober. The patients may go into alcohol withdrawal. If you are strictly following the blood alcohol level, you may make decisions that are not in the patient’s best interest to get them below a legal threshold.
- Below the limit might not be good enough. There is a certain subset of patients who are intoxicated although their blood alcohol level is below the legal limit. This can put them at risk of injury after discharge.

Patients with a legal but detectable blood alcohol level had a significant increase in accident severity compared to drivers who had not consumed alcohol.

- Is it totally safe to discharge them? The legal literature is scattered on this. Delaney will let patients with elevated blood alcohol levels metabolize until they should be under the legal limit, document that they are clinically sober, and help arrange a ride home. If the patient is in the Emergency Department solely for alcohol intoxication, Delaney will discharge them home if they are improving and have a sober ride. If the patient is in an accident with a potential missed injury, they should be observed until clinically sober and re-assessed for occult injury.
- Many patients who are drunk want to sign out against medical advice. The courts are reluctant to say a patient does not have capacity. Delaney will sign the patient out against medical advice. He calls the police and tells them he has an intoxicated patient that he is worried about. He does not restrain them due to intoxication.

Ashoo

- There is the correct school of thought and the wrong school of thought. Clinical sobriety is a clinical exam. The number is irrelevant.
Swaminathan

- Is there utility in getting a blood alcohol level from a medico-legal perspective? Blood alcohol levels were adopted by the states to determine who was able to operate a motor vehicle. They selected a number that seemed correct, knowing that there is a wide spectrum. Patients may have co-ingestions of other medications that contribute to altered mental status.
  - We don’t have cases where not obtaining a blood alcohol level made a difference. The examination and evaluation of the patient is what is important. Most legal cases arise due to failure to document the abilities and limitations of the patient.
  - Kowalski versus St. Francis Hospital. An intoxicated patient eloped from the Emergency Department and was hit by a car. He became a quadriplegic. He sued the physician and hospital, saying that he should not have been allowed to leave as he did not have decision-making capacity. The physicians and nurses had documented that the patient was alert, was awake, was oriented, walked normally, and looked fine. His blood alcohol was elevated. The state Supreme Court ruled that they did not have the right to hold the patient against his will if he met the rules of competency. They would have committed the felony of battery.
  - The patient is normal when determined by documented examination and nothing else. The number is useless and variable.
  - However, a patient with an anticipated change in their mental status (such as a patient who received naloxone after opiate withdrawal and is at risk of repeat symptoms when the medication wears off) may be reasonably held against their will, as they present a danger to themselves or others due to a disease entity that may return. This is not the case with alcohol, unless they are consuming more in the Emergency Department.
  - If the patient has neurologic findings, such as ataxia or altered mental status, you won’t discharge them even if they have a low blood alcohol level.

Swaminathan

- Swaminathan does not usually get blood alcohol levels unless he is unsure if the patient is altered due to alcohol intoxication.
- What if the patient had a blood alcohol level drawn that was elevated but they seem sober? This depends on where you work and how the patient will leave. If you work in an area where the patient has to drive home, you can’t send them out to drive home. Getting them a sober driver is probably the way to go. If you didn’t get a blood alcohol level and sent them out to drive home, you aren’t really in a better situation.
- Waiting for the alcohol level to fall to below legal limits can be dangerous in chronic drinkers, as it may precipitate withdrawal when they reach sober levels. Also, when the patient is sober enough to walk around and wants to go home, they will disrupt your department until they leave. Establish that the patient has capacity to leave before they go home. If they can walk, talk, and tell you what is going on, you can establish capacity without them reaching the legal BAL limit.

Henry

- Is there utility in getting a blood alcohol level from a medico-legal perspective? Blood alcohol levels were adopted by the states to determine who was able to operate a motor vehicle. They selected a number that seemed correct, knowing that there is a wide spectrum. Patients may have co-ingestions of other medications that contribute to altered mental status.
  - We don’t have cases where not obtaining a blood alcohol level made a difference. The examination and evaluation of the patient is what is important. Most legal cases arise due to failure to document the abilities and limitations of the patient.

The LIN Sessions: Overtesting
Michelle Lin MD and Hemal Kanzaria MD

Highlights

- Over 85% of physicians feel that too many diagnostic tests are ordered in their own department and 97% acknowledged personally ordering medically unnecessary radiology tests.
- The two most common contributors are fear of missing a low-probability diagnosis and fear of being sued.
- Increased engagement of patients through education and shared decision-making is a promising way to address over-imaging.

- A survey tool was developed using different techniques such as focus groups, expert opinion, and pilot testing. 435 emergency physicians were asked for their perspectives on diagnostic imaging and overuse of imaging in general. Respondents were asked if they personally order CTs and MRIs that they believe to be medically unnecessary. Medically unnecessary testing was defined as a study you would not order if you had no external pressures and were only concerned with providing optimal medical care. Physicians were then asked about contributing factors to ordering unnecessary tests and solutions to this issue.
- Over 85% of physicians felt that too many diagnostic tests were ordered in their own department. Almost all respondents (97%) acknowledged personally ordering some amount of medically unnecessary radiology tests.
- Why? The two most common contributors were fear of missing a low-probability diagnosis and fear of being sued.
- Suggested solutions were tort reform and increased involvement of patients via education and shared decision-making.
- Over-imaging is a systemic problem throughout medicine and not a problem of bad doctors. Over-testing is not due to lack of knowledge or poor medical judgment, but reflects a cultural response to uncertainty and error.
- Most physicians reported that they ordered tests out of fear of being sued and suggested tort reform as a solution. Almost all physicians in high-risk specialties will face a claim during their...
career. Tort reform is necessary but will be insufficient to address this issue.


They looked at Emergency Medicine practices in three states: Texas, Georgia, and South Carolina, where substantial tort reforms were made that provide emergency physicians with significant legal protections. They compared Emergency Medicine practice in these states with surrounding states that didn’t have these reform efforts. They looked at the effect of the legislation on the ordering of CTs and MRIs, Emergency Department charges, and hospital admissions and found no change in outcomes. However, changes may not have manifested yet.

- Fear of malpractice isn’t the only driver. Our collective intolerance of uncertainty is at least as large as a driver. We don’t like uncertainty and we feel we should do something if there is any possibility of disease. This approach is well-intentioned but does contribute to the culture of overtesting and can lead to patient harm.

- In the study, physicians felt that increased engagement of patients through education and shared decision-making was a promising method of addressing over-imaging. Shared decision-making involves increased engagement of patients in their healthcare choices. Physicians and patients collaboratively discuss potential management strategies when more than one option exists, and they work together to reach a decision based on the best available evidence and the patient’s values and preferences. The benefits are multifold. Studies show that patients who are engaged in this process demonstrate increased understanding of their healthcare and satisfaction with their healthcare decisions.

- There are a few studies to show that this may decrease healthcare costs. Hess EP, et al. Implementation of shared decision making in cardiovascular care: past, present and future. Circ Cardiovasc Qual Outcomes. 2014 Sep;7(5):797-803. PMID: 25052074. They randomized Emergency Department patients with low-risk chest pain to usual care versus shared decision-making. Patients assigned to shared decision-making received a decision aid that was used to educate patients in the choice of admission versus outpatient follow-up. Patients with shared decision-making had greater understanding of their condition and lower healthcare utilization rates, without an increase in adverse outcomes. There are many areas of Emergency Medicine that are preference-sensitive, with no right or wrong answer.

- In the next several years, there will likely be more research on where shared decision-making might be most appropriate. A randomized controlled trial is underway at the Mayo group looking at head CT for minor head injury in pediatric patients, who meet criteria for moderate risk. It is up to the physician to explain the different potential harms and benefits of a management strategy.

- This is important topic; 4 of the 10 guidelines of the ACEP Choosing Wisely campaign relate to advanced imaging.

- Avoid a head CT in patients with minor head injury and use validated decision rules to determine low risk.

- Avoid a CT for asymptomatic syncope without trauma and with a normal neurologic exam.

- Do not get a CT pulmonary angiogram in patients with a low pre-test probability for pulmonary embolism.

- Don’t order a CT for patients with a high suspicion for uncomplicated kidney stone.

- The Society of Academic Emergency Medicine will be hosting conferences on optimizing imaging in Emergency Medicine and shared decision-making.

- We are taught to take care of the patient in front of us and not think about cost. On the other hand, many of these efforts say that we should be stewards of resources and minimize waste. There is perceived tension between doing what is right for the individual patient in the Emergency Department and doing what is right for society at large. It is essential that practicing emergency physicians, society, and patients are engaged in these solutions. We need to address widespread societal beliefs that promote the perceptions that error is the cause of any bad outcome, technology can solve all problems, and catching diseases early is always beneficial.

### Sepsis Decision Pathway: Recognition – Part 1
Cam Berg MD and Rob Orman MD

#### Highlights

- Studies investigating each of the elements of early goal-directed therapy (EGDT) found that several elements of the EGDT bundle do not help.

- Early and appropriate antimicrobials and early and aggressive crystalloid fluids are important.

- Sepsis is a leading cause of death; patients with septic shock have mortality of 30% and those with severe sepsis have mortality of 25%.

- Patients with sepsis don’t present with a clear-cut chief complaint. Many Emergency Departments struggle with how to effectively triage patients with potential sepsis. If you aren’t careful, you could bring everyone back to the resuscitation room in flu season.
• In 2001, Manny Rivers changed the world of sepsis and initiated much of the work that has happened in the last decade. They showed that we can make a huge difference by paying attention to our sepsis patients. Rivers proved a number needed to treat of 6 to save a life with EGDT. There are few interventions in Emergency Medicine with that kind of outcome difference.

• A lot of new concepts were advanced in a single publication.

• Lactate was also emphasized; patients may be sicker than our clinical gestalt suggests. A lactate of 4mmol/L or more had comparable outcomes with patients with full-blown hemodynamic shock; mortality is over 30%.

• However, multiple interventions were bundled together: lactate screening, early antibiotics, aggressive and protocolized fluid resuscitation, invasive hemodynamic monitoring, CVP, ScvO2, blood transfusions and inotropes. What really works?

• In recent years, a lot of work has been done to investigate each of the elements of EGDT and found that several elements of the EGDT bundle did not help. CVP is not a good marker of volume status. ScvO2 doesn’t seem to unmask any secrets. Blood transfusions seem to be detrimental in sepsis. Pure inotropes like dobutamine are seldom needed in sepsis and the arrhythmogenic properties can be problematic.

• The PROCESS and ARISE trials were randomized trials performed in multiple centers in various countries that demonstrated that less complex protocols are equally effective to EGDT. Berg, in collaboration with others, has developed a local protocol modeled on the evidence obtained over the last 15 years and attempts to standardize the treatment of sepsis patients. The pathways are applied to patients with all severity of sepsis.

• The pathway highlights two things: early and appropriate antimicrobials and early and aggressive crystalloid fluids.

• EGDT works well for the sickest sepsis patients but is cumbersome. Prior to development of the pathway, they didn’t have a standard resuscitation algorithm for less sick patients. They didn’t have a system for lactate screening. There was no standardized disposition. Central line utilization varied widely and was often placed for monitoring only.

• Does it matter whether the patient presents via EMS or to triage? Yes. Time matters. Ambulance patients are typically roomed immediately whereas triage patients may wait in a queue.

• Berg’s group places a physician in triage, and sepsis patients are roomed earlier. The EMS system has also established a sepsis recognition and resuscitation pathway using point-of-care lactate in the ambulance.

• What patients enter the pathway? For inclusion in the pathway, any adult patient is eligible. Screening is performed with modified SIRS criteria. Patients with two or more criteria are considered for sepsis as a cause versus something else. Do they have SIRS and a suspected clinical infection? If yes, the patient enters the pathway.

• The modified SIRS criteria replace the white blood cell count with other vital sign parameters. SIRS is described by two of more of the following clinical characteristics: temperature >100.4°F (38°C) or < 96.8°F (36°C), heart rate >90, respiratory rate >20, oxygen saturation <90%, MAP <65 or new altered level of consciousness. This allows patients to be identified earlier and without laboratory testing.

• The EMR is able to flag suspected patients based on vital sign parameters. The nurse is able to highlight patients with a suspected clinical infection.

• Sepsis is a leading cause of death and the largest inpatient DRG (diagnosis-related group). Patients with septic shock (sepsis and a low blood pressure) have a mortality of 30%. Patients with severe sepsis (sepsis and a lactate greater than 4mmol/L) have a mortality of approximately 25%. Patients with an intermediate lactate (abnormal but below 4mmol/L) have a mortality of about 10-15%. Patients with a normal lactate have a mortality of about 5%. Patients with STEMI have a mortality of about 2%.

• Once patients are entered into the pathway, an IV is placed and a lactate and single blood culture is drawn along with labs. The blood culture is kept and labeled but not necessarily sent to the lab. They are primarily interested in obtaining cultures of the suspected infection site. If the blood is sent for culture, they perform two sets of cultures from separate sites.

Sepsis Decision Pathway: Recognition – Part 2
Cam Berg MD and Rob Orman MD

Highlights

• Protocols can help differentiate septic patients and provide organized, evidence-based care for all patients from all providers.

• Patients with persistently elevated lactate have a very high in-hospital mortality.

• The mean arterial pressure (MAP) is the major branch point. If the patient has a MAP less than 65, they are immediately roomed in a bed. Aggressive resuscitation is initiated. Diagnostics are initiated. The patient is given two liters of IV crystalloid fluids and then the blood pressure is rechecked.

• Septic shock is defined as persistent hypotension despite the initial fluid bolus. The literature is scattered regarding what constitutes an initial resuscitation effort. This protocol uses two liters. If the MAP remains less than 65, it is called septic shock. The patients have sterile central line placement in the internal jugular or subclavian. They are started on nor-
Patients will likely have a normal MAP.

- They do not use CVP to guide fluid resuscitation but rather perform empiric fluid loading. All patients are given the same moderate dose of fluid. They use age and medical comorbidities to determine fluid amount. Patients with age greater than 75 years or a history of systolic congestive heart failure (defined as an EF of less than 40%) receive three liters. All other patients receive 4 liters.

- They do not look for fluid responsiveness with ultrasound of the IVC, although this may be a good strategy if resources are available. CVP tends to underresuscitate certain patients, such as patients with heart failure and pulmonary hypertension, even if they are intravascularly depleted and in distributive shock. A young patient in distributive shock may need 8-9 liters to reach a target CVP, which is not good either.

- Either normal crystalloid or lactated Ringers is fine.

- Patients with presumptive septic shock get blood cultures and cultures of the presumptive site of infection.

- They are given broad-spectrum antibiotics. The antibiotic regimen is standardized based on the hospital susceptibility data and suspected site of infection. The goal is antibiotic administration within an hour.

- These patients are dispositioned to the ICU.

- In practice, patients may stabilize after a third liter and these patients would not necessarily have a central line placed. It is still prudent to call these patients septic shock. Patients do not need a central line unless they need vasopressor infusion or IV access. Lines are not placed for monitoring or due to the diagnosis.

- If the MAP is greater than 65, the serum lactate determines the next decision point. Lactate is very predictive in sepsis and correlates linearly with mortality. Patients with a normal lactate (less than 2mmol/L, depending on assay) have uncomplicated sepsis. Patients with intermediate lactate (2mmol/L to 3.9mmol/L) have sepsis. Patients with a lactate greater than 4mmol/L have severe sepsis.

- Causes of elevated lactate other than sepsis: any dramatic increase in metabolism, such as vigorous physical activity or seizure, trauma, use of beta-agonists, and chronic liver disease.

- What do you do during flu season? Patients will likely have screening with lactate and chest x-ray. Patients are excluded from the protocol if they do not have a suspected bacterial infection. Patients with upper respiratory infections are often triaged as ESI 4 or 5 and these triage levels are also excluded from the pathway.

- What happens to a patient with a lactate greater than 4 mmol/L with a normal MAP? These patients receive non-invasive resuscitation. They do not have central line placement unless it is mandatory for vascular access. They also receive empiric fluid loading similarly to patients with septic shock (3-4L). They have cultures obtained of their blood and the suspected site of infection. They receive broad-spectrum antibiotics. These patients are also dispositioned to the ICU. A lactate greater than 4mmol/L meets criteria for critical care reimbursement. The outcomes of these patients are comparable to patients with septic shock.

- What happens to a patient with a lactate between 2mmol/L and 3.9mmol/L? None of the studies, such as PROCESS or ARISE, addressed this category of patients. They receive 2 liters of IV crystalloid. They have cultures obtained of the suspected site of infection only and do not have routine blood cultures sent. They receive antibiotics targeted to the infected organ. These patients are diagnosed with sepsis. Admission is recommended. The lactate is repeated after the patient has received two liters.

- Lactate clearance is defined as a decrease of at least 10%. Lactate has a very short biologic half-life.

- If the lactate remains persistently elevated, it means that the patient’s disease state has continued to result in its production. These patients have a very high in-hospital mortality. In Berg’s data set, these patients have an in-hospital mortality of more than 50%. This subset of patients, who fail to clear their lactate, go to the ICU. The rest of the patients are admitted to the floor.

- Do you need to culture the blood of a patient with pneumonia? This goes back to a CMS quality recommendation. It was interpreted to mean that all patients with pneumonia who are admitted need to have blood cultures drawn prior to antibiotics. What the CMS core measure actually said (based on no data) was that patients who are admitted for pneumonia should have blood cultures obtained prior to antibiotics if blood cultures are obtained in the first day. You are safe not sending blood cultures, as long as the admitting physician also does not obtain blood cultures. You need a consensus. If the patient has complicated nosocomial risk factors or chronic lung disease and community acquired pneumonia, they will have blood cultures drawn prior to antibiotic administration.

- What happens to a patient with a normal lactate? Treatment and disposition is per the physician. These patients tend to do well. The in-hospital mortality for admitted patients with a normal lactate is 5%. In some cases, observation or discharge home may be appropriate options.

- Patients with hemodynamic shock refractory to fluids receive the full treatment, regardless of lactate.

- What has happened since the introduction of the protocol? They aimed to maintain or improve sepsis-related mortality while decreasing hospital length of stay and total ICU utilization. So far, they are meeting all targets. Their overall sepsis mortality has
improved slightly from 17% to 14%. The length of stay has decreased by a day. They have seen improved reimbursement due to use of the correct terminology in their charting; for example, patients are diagnosed with sepsis rather than pyelonephritis or pneumonia. More patients are admitted to the ICU from the ED. However, despite admitting more patients to the ICU from the ED, their overall ICU utilization has decreased. Prior to the protocol, more patients were admitted to the floor, under-resuscitated, and then transferred to the ICU when they worsened.

- This is not the only way to differentiate septic patients but it is organized, is evidence-based, and gives a baseline level of care for all patients from all providers. The Surviving Sepsis campaign has a 2015 update that reflects this: CVP and ScvO2 are out; patients in septic shock should have a lactate and blood cultures drawn prior to antibiotics within three hours of presentation; and patients should receive 30cc/kg of IV crystalloid for hypotension or lactate > 4mmol/L.

- Sepsis Resuscitation Pathway Diagram (See Next Page)

Pharmacy Rounds: Rate control for atrial fibrillation
Brian Hayes PharmD

Highlights

- Studies comparing calcium channel blockers to beta-blockers found that both were effective in controlling rate.

- There was a trend towards better efficacy of diltiazem but this was not statistically significant.

- Although there are no published cases in the literature, giving a beta-blocker after calcium channel blocker or vice versa is not recommended due to concerns about heart block.

- Calcium channel blockers versus beta blockers. There is some literature available. The 2014 Atrial Fibrillation Guidelines recommend a non-dihydropyridine calcium channel blocker or beta-blocker as first-line therapy in rate control. They both receive 1B level recommendations.


- This study included 45 patients with atrial fibrillation or atrial flutter. It was a randomized, open-label study and patients received either esmolol or verapamil. They found a significant and equal decrease in heart rate with either agent.

- They found that 50% of the patients who received esmolol converted to normal sinus rhythm versus only 12% of patients with verapamil. This was statistically significant.

- Mild hypotension was observed in both groups.

- This was a small group of patients. Diltiazem is used more commonly in the United States, while verapamil is used more commonly in Australia and other countries.


- This study included 40 patients: 20 received IV pushes of metoprolol and 20 received IV pushes of diltiazem. All patients had atrial fibrillation. This was prospective, randomized trial performed in the Emergency Department.

- They found the success rate of metoprolol was 80% and diltiazem was 90% at 20 minutes after administration. However, this was not statistically significant. At two minutes, the success rate of diltiazem was 50% compared to 15% of metoprolol, and this was statistically significant.


- 52 patients were included: 28 patients received diltiazem and 24 patients received metoprolol. Patients had atrial fibrillation or atrial flutter. This was a prospective, randomized, double-blinded trial. They used rigorous methodology.

- Within 30 minutes, 96% of patients in the diltiazem group reached the target heart rate of less than 100 compared to 46% of patients in the metoprolol group.

- Two studies with less rigorous methodology comparing calcium channel blockers to beta-blockers found that both were effective in controlling rate. Medications and doses used were not reported. They had similar hospital rates and safety.


- There is an ongoing randomized, placebo-controlled, double-blind trial that compares diltiazem and metoprolol in the Emergency Department currently underway and scheduled for completion in December.

- Based on the available data, diltiazem may work a little better but both are appropriate.

- Calcium channel blockers and beta-blockers: what happens if one doesn’t work – do you give the other? There is a concern for heart block. What does the evidence say? Hayes was unable
SEPSIS RESUSCITATION PATHWAY

- SIRS = ≥2 of T >100.4 or < 96.8, HR > 90, RR > 20, O2 sat <90%, MAP < 65, New ALOC
- System-based examples of clinically suspected infections:
  - Intra-abdominal
    - Abdominal pain
    - Rectal pain or purulent discharge
  - Urinary
    - Dysuria, urgency, or frequency
  - Constitutional
    - Rigors
    - Reported measured fever
    - Monoarticular non-traumatic joint pain


Modified SIRS ≥ 2 and suspected infection?

- Yes
  - Draw and order lactate and draw rainbow per protocol

- No
  - Exit protocol

MAP < 65

- Yes
  - Patient roomed to bed. Initial fluid resuscitation (2L). Broad spectrum antibiotics. Obtain blood culture and cultures of suspected source.

- No
  - lactate results ≥ 4
  - MAP < 65
    - Yes
      - Severe Sepsis Non-Invasive Protocol
        - SIRS + infection + serum lactate ≥ 4
          - Patient roomed to bed.
          - Empiric Fluid Load:
            - if age > 75 OR history of systolic heart failure -> give a total of 3L IV crystalloid within first 6 hours
            - if age ≤ 75 AND no history of systolic heart failure -> give a total of 4L IV crystalloid within first 6 hours.
          - 2 peripheral IV lines should be placed unless a large and reliable line is possible (16g AC).
          - Broad spectrum antibiotics. Obtain blood culture and cultures of suspected source.
          - Central line NOT mandatory. Admit to ICU.
          - Reassess patient and recheck lactate upon completion of fluids

  - MAP < 65
    - No
      - Severe Sepsis Invasive Protocol
        - SIRS + infection + hypotension (MAP<65) refractory to 2L IVF
          - Central line placed.
          - Empiric Fluid Load:
            - if age > 75 OR history of systolic heart failure -> give a total of 3L IV crystalloid within first 6 hours
            - if age ≤ 75 AND no history of systolic heart failure -> give a total of 4L IV crystalloid within first 6 hours.
          - Initiate norepinephrine and target MAP ≥ 65. Admit to ICU.

  - MAP < 65
    - No
      - Uncomplicated Sepsis
        - SIRS + infection
          - Patient roomed to bed in ED. Initial fluid resuscitation (2L). Targeted antibiotics per suspected source. Obtain cultures of suspected source.
          - Reassess patient and recheck lactate upon completion of fluids

To obs / floor for ongoing care

- ≥ 10%
  - Repeat bolus = 1L lactated ringers IV. Admit to ICU.

- < 10%
  - Lactate clearance

- To obs / floor for ongoing care

- Treatment and Dispo per physician
to find a single study or case report that demonstrated heart block after giving both. However, most recommend avoiding this combination.

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### Critical Care Mailbag: Intubation Post-ROSC

Scott Weingart MD and Rob Orman MD

#### Highlights
- If you are unable to obtain a end-tidal CO₂ reading in a patient after cardiac arrest and ROSC, check for a pulse.
- Supraglottic airways may be maintained throughout resuscitation during cardiac arrest, but patients with ROSC should have placement of a definitive airway prior to transport to the cath lab.
- Drugs that may be used include ketamine and etomidate in lower doses. Propofol may be used at a low dose but is less ideal. Fentanyl and midazolam don't work.
- Any patient who is not in cardiac arrest should not be intubated without drugs.

#### CASE

An 88-year-old with out-of-hospital ventricular fibrillation cardiac arrest was shocked three times by Emergency Medical Services (EMS). Return of spontaneous circulation (ROSC) was achieved. On arrival, the patient had a supraglottic airway. The patient was in a fast, broad-complex atrial fibrillation at 140, with good oxygen saturation. They were considering acute myocardial infarction versus pulmonary embolism. The patient maintained output after ROSC, and bedside echocardiogram showed a globally dilated heart with poor ejection fraction. The decision was to secure the airway, obtain a head CT, and consider transport to the cath lab.

- The first attempt with direct laryngoscopy had no end-tidal CO₂ reading post-intubation, so the decision was to remove the tube and repeat.
- The first question you need to ask is, does the patient actually have a pulse? You will get a flat end-tidal CO₂ in patients who do not have a pulse, regardless of where the tube is.
  - Rather than immediately trying to reintubate or check tube placement, palpate for a pulse. If they have a pulse, then remove the tube and reoxygenate the patient.
  - Also, consider equipment failure. You can blow into the end-tidal tube to confirm it is working prior to use. Or you can use a colorimetric end-tidal CO₂ detector.

#### Should you leave the supraglottic airway device in place or establish a more definitive airway?
It is reasonable to continue the supraglottic airway during the active code. Don’t send the patient with ROSC to the cath lab with the supraglottic airway, unless there is an anesthesiologist accompanying the patient.

#### The endotracheal tube was removed and the patient was spontaneously breathing.
The resident suggested that the patient needed medications to facilitate intubation.

- **What are the best drugs to use?** The patient had poor cardiac function. Propofol could cause the patient to crash. Ketamine was suggested, but acute myocardial infarction was on the differential and they didn’t want to increase her heart rate.
- The patient was given midazolam and fentanyl but no paralytic. Her blood pressure dropped after intubation. She received phenylephrine and improved.

- **Would ketamine be reasonable or make things worse?** Ketamine gets a bad rap. This patient was borderline or frankly hypotensive. She would likely be placed on vasopressors soon. The patient was likely given an enormous amount of inotropes and vasopressors during the resuscitation. The amount of sympathetic surge that you are going to get from the ketamine dose used is inconsequential. If ketamine made any difference in the vital signs, it would probably be a benefit to this patient who will be converted from negative pressure to positive pressure ventilation.

- **Weingart recommends a half dose of ketamine; why?** In these hemodynamically unstable patients, Weingart reduces the dose by half. This will augment the safety. The difference between this and other sedation agents when used in a half dose is a semi-dissociated state, not under-sedation. Patients have no real perception of pain and their awareness, if any, is a fog. They won’t remember the intubation and the potency of the analgesia will prevent discomfort.

- **Should the patient with ROSC be intubated without drugs?** Any patient who is not in cardiac arrest should not be intubated without drugs. Even if the patient is unconscious, they still have tone in the muscles you need to move out of the way, in order to get your best view for first-pass success.

- **The patient received midazolam and fentanyl for induction prior to intubation. What about this choice?** These don’t work. Even in the best circumstances, these medications take about 3 minutes for full effect. They likely didn’t give the induction dose of 20-30mg of midazolam and 500mcg of fentanyl. No one gives these doses in the Emergency Department. If you use these in the small doses used in the Emergency Department, you are essentially intubating the patient without any drugs.

- **How do you proceed in patients with recurrent arrest?** This is becoming increasingly common with mechanical CPR devices, due to improved cardiac output. Their brain gets perfused and...
they wake up. You stop the machine to check a pulse, there is no pulse, and then they re-arrest. You need to give these patients something.

- **What should you use?** No one has a great answer.
- Ketamine is a good option.
- If the patient is interfering with your CPR attempts despite the ketamine, you can paralyze them.
- Etomidate is a great drug. Weingart recommends using a half-dose of etomidate as well.
- Some Emergency Departments only have propofol immediately available. You can give it, but you should give a dose that is one-tenth of the usual dose (if you normally give 100mg, give 10 mg). Studies have shown that patients with poor cardiac output (these are mostly in hemorrhagic shock patients) will have the same level of brain sedation as healthy patients despite lower doses.

**Take-home points.**

- The patient should have been paralyzed with succinylcholine or rocuronium on the first intubation attempt.
- Always use a muscle relaxant unless you are performing an awake intubation.
- Always give an induction medication to a patient who may have some perception of the intubation.
  - Half-dose ketamine is a good choice.
  - Half-dose etomidate is a good choice.
  - Propofol is an ok choice; you can make it work but reduce the dose.
  - If you want to use midazolam or fentanyl you can, but it is purely for its amnestic effect; the patient may have some perception but he/she probably won’t remember it afterwards.

**Paper Chase 1:**

**LP Findings that Rule-Out SAH**

Sanjay Arora MD and Michael Menchine MD

**Highlights**

- **CT scans and lumbar puncture are not without complications; there are a lot of false-positives.**
- The combination of no xanthochromia and < 2000 red blood cells rules-out subarachnoid hemorrhage in a prospective cohort of patients with suspected subarachnoid hemorrhage.


- This study showed that the combination of no xanthochromia and less than 2000 red blood cells rules-out subarachnoid hemorrhage, in a prospective cohort of patients with suspected subarachnoid hemorrhage.
- **This is an important paper on an important topic.** Subarachnoid hemorrhage, as a cause of headache, is a big problem. Headaches are frequently presenting complaints making up 2% of ED visits. 1% of headaches are due to subarachnoid hemorrhage. If you miss a subarachnoid hemorrhage, the patient can return moribund. It is estimated that approximately 5% of subarachnoid hemorrhages are missed initially in the Emergency Department.
- **Unfortunately, performing CT scans and lumbar puncture are not without complications.** They are not fun for patients or doctors. There are a lot of false-positives.
- **Perry published a paper saying that a modern head CT scan could rule-out subarachnoid hemorrhage, if performed within six hours.**
- **They reported a sensitivity of 100% if CT was performed within 6 hours. After six hours, sensitivity drops to 85%. Unfortunately, the large majority of patients present after 6 hours. Lumbar puncture will remain part of the diagnostic strategy for subarachnoid hemorrhage for awhile.**
- **30-40% of lumbar punctures will have some amount of blood.** People have been trying to determine how much blood is due to traumatic tap versus subarachnoid hemorrhage. Xanthochromia is sometimes used as a marker of subarachnoid hemorrhage, as it is due to the breakdown of red blood cells. However, this is only 47% sensitive. Other studies have looked at the number of red blood cells in tube 4 or the decrease from tube 1 to tube 4. None of these studies have been particularly compelling. All have been retrospective in nature and results were varied.

- **This study was a prospective cohort of patients being worked up for subarachnoid hemorrhage.** Patients evaluated for acute headache were enrolled in Canadian Emergency Departments over a ten-year period. Acute headaches were defined as having maximum intensity within an hour of onset. Patients with chronic headaches were excluded. 4,141 patients were enrolled and 3.5% had subarachnoid hemorrhage. The majority of patients received CT scans. 1,739 had lumbar puncture performed. Of the patients who received lumbar puncture, 37% had some amount of blood or xanthochromia on the lumbar puncture.
• Patients had an aneurysmal subarachnoid hemorrhage if they had an aneurysm on CT angiography and required neurovascular intervention. Patients were followed for six months to rule-out subsequent aneurysmal subarachnoid hemorrhage.

• What did they find? Of the 641 patients with red blood cells in their CSF, the vast majority were traumatic taps. Only 15 had aneurysmal subarachnoid hemorrhage. The patients with subarachnoid hemorrhage had a lot of blood in their CSF, with a median was approximately 20,000 cells, compared to patients with a traumatic tap who had a median of 20 cells.

• What is the cutoff to differentiate trauma versus subarachnoid hemorrhage? 2000 red blood cells had a sensitivity of 93% and specificity of 93%. This identified 14 out of 15 patients. If xanthochromia was added, they identified all 15 cases. There were only 15 true-positives and the confidence intervals were wide.

• They determined that no xanthochromia and less than 2000 red blood cells is a low-risk finding. If your pretest probability is low, you can probably discharge the patient. However, if your pretest probability is high (such as a patient with a history of a Berry aneurysm), you should probably do additional testing.

• Aortic dissection is bad. Mortality is estimated at 1-2% per hour without treatment. However, it is very uncommon. There are three basic types of aortic dissection: the intimal flap tear type (which is the majority of cases), the intramural hematoma (which is unlikely to be identified by D-dimer), and the penetrating ulcer type (which is rare).

• Risk factors include hypertension, collagen tissue disorder like Marfan syndrome or vasculitis. Chest pain is the most common presenting complaint: 73% of patients in the IRAD (International Registry of Acute Aortic Dissections) database with aortic dissection had chest pain. This was classically described in the database as sharp pain, rather than tearing or ripping. Painless dissection is very uncommon and accounted for about 5% of cases.

• Can you use D-dimer to rule-out aortic dissection? Other studies looking at this topic have had mixed results due to poor quality, small numbers of patients, retrospective studies, and multiple endpoints for what constitutes a positive D-dimer.

• The authors performed a meta-analysis looking for articles with the following characteristics: original research; designed to evaluate a diagnostic test; prospective enrollment of patients with suspected acute aortic dissection; and D-dimer was performed as well as some type of gold standard such as CT, MRI, or TEE. Also, the values to calculate true- and false-positives and negatives were provided. They found 5 studies that met these standards.

• The total number of patients was 1,557. Most of the studies used a cutoff of 0.5 ug/mL (500 ng/mL).

• What did they find? Using D-dimer in a low-risk patient gave a sensitivity of 98% and a negative likelihood ratio of 0.05. The specificity of the test is low. The prevalence of aortic dissection in these studies was very high, about 20-50%.

• The paper is subject to the usual limitations of a meta-analysis. For example, different study settings, different assays, different timing of blood draws, etc.

• What do they recommend? In a low-risk patient as defined by the AHA criteria, a negative D-dimer makes your post-test probability of the disease 0.3%. The upper limit of the confidence interval (i.e. what if the test works as poorly as possible in the sample) was 0.6%. These are pretty good numbers. However, patients who are low-risk based on this criteria have no signs of dissection, including chest pain, so why are we even considering dissection? The proportion of patients for whom a D-dimer is appropriate is very, very low. D-dimer is unlikely to catch patients with intramural dissection. A positive test can be problematic as well, due to its poor sensitivity; you will do more work-ups than necessary.

Paper Chase 2: D-dimer and Aortic Dissection
Sanjay Arora MD and Michael Menchine MD

Highlights

• A negative D-dimer in isolation has test characteristics sufficient to rule-out acute aortic dissection in a patient with low pretest probability, as determined by the American Heart Association Criteria.

• The proportion of patients for whom a D-dimer is appropriate is very, very low.

• D-dimer is not sensitive enough for any other risk category other than low-risk patients.


• The authors concluded that a patient with low pretest probability, as determined by the American Heart Association Criteria, with a negative D-dimer in isolation has test characteristics sufficient to rule-out acute aortic dissection. However, the proportion of patients (i.e. low-risk) that we see that this applies to is very low. The test is not sensitive enough to be used in any other risk category.

• American Heart Association Acute Aortic Dissection Risk Score (See Next Page)

This is a validation of the aortic dissection risk score. They included 1,035 patients. They found that none of the low-risk patients had an aortic dissection with a negative D-dimer. The sensitivity was 100%.

However, the miss rate increased if the patients were moderate-risk (one criteria in one category); two cases were missed out of 152.

In patients who were high-risk (one or more criteria in two or more categories), the D-dimer was falsely negative in 2 out of 48 cases of aortic dissection.

If you are going to use a D-dimer in the work-up of patients with suspected aortic dissection, it should only be in patients who are very, very low-risk.

Paper Chase 3: Blunt Trauma Thoracotomy
Sanjay Arora MD and Michael Menchine MD

Highlights

- Emergency Department (ED) thoracotomy is a well-established therapeutic option for patients with penetrating injury that results in cardiac arrest.
- Recommendations regarding ED thoracotomy in blunt traumatic arrest are divergent.
- Only between 0.9% and 1.5% of patients survived with good neurologic outcome.
- Highest survival rates were found in patients who had signs of life in the ED, compared to patients who only had signs of life in the field.

Case

A 50-year-old male was an unrestrained driver involved in a high-speed collision into a telephone pole. He was ejected and unconscious on Emergency Medical Services arrival. He was intubated in the field on the first-pass. Two IVs were placed. Blood pressure was 60/palp and heart rate 120. He had a small laceration to the right forehead and an obvious femur fracture. The patient loses his pulse.

• This systematic review of the literature focuses on the outcome of ED thoracotomy for blunt traumatic arrest and concludes that the overall survival rate with good neurologic outcome is 1.5%. The authors performed some subanalysis and offered guidelines to determine in what cases to consider ED resuscitative thoracotomy for blunt traumatic arrest.

• ED thoracotomy is a well-established therapeutic option for patients with penetrating injury that results in cardiac arrest. The main goals are to relieve a cardiac tamponade, clamp something bleeding in the chest (gain control of intrathoracic hemorrhage), and compress the aorta to push blood to vital organs while reducing blood loss. You can’t gain control of a bleeder in blunt trauma through an ED thoracotomy.

• All of the major organizations recommend ED thoracotomy for penetrating traumatic cardiac arrest, providing there hasn’t been an inordinate amount of time between the loss of vital signs and the procedure (about 10-15 minutes). Blunt traumatic cardiac arrest is different, as the outcomes are much more dismal. The Western Trauma Association recommends doing the thoracotomy in blunt trauma if there is any sign of life, including electrical activity, or there is no sign of life but less than ten minutes of CPR. ATLS does not recommend thoracotomy. These recommendations are very divergent. This only applies to adults.

• The authors conducted a systematic review and meta-analysis of the literature and focused on two key questions: 1) What are the chances of a good or bad neurologic outcome following ED thoracotomy for blunt traumatic arrest? 2) Are there any factors that make a substantial difference, such as injury type or duration of CPR? The methods were good and they adhered to meta-analysis best practice guidelines.

• They found 27 articles to review overall and included 13 in the meta-analysis. All of these were case series. None of them were trials and none of them had control groups. The meta-analysis was limited to those studies that had a specific protocol for when to do a thoracotomy.

• They identified 1,369 patients who received ED thoracotomy and 1.5% survived with good neurologic outcome. This is greater than previously thought. This is somewhat biased as one of the studies allowed ED thoracotomy on patients who weren’t quite dead. In this study, they performed thoracotomy on patients who were hypotensive and these patients had better outcome. If this study is excluded, the number of survivors dropped but stayed at 0.9%.

• Highest survival rates were found in patients who had signs of life in the ED, compared to patients who only had signs of life in the field. Survival was almost universally zero for patients with no signs of life in the field. In particular, 2 out of 500 survived with dismal neurologic outcome. Patients who did poorly were those who had cardiac arrest due to massive head trauma.

• They developed resuscitative guidelines despite the poor quality of evidence. In patients with vital signs in the ED with short CPR time (less than 15 minutes) and no obvious head injury, you can attempt an ED thoracotomy. However, even in this highly selected cohort, the chance of survival is very low. If you don’t have the technical capability, don’t do it.

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**Paper Chase 4:**

**Clinda vs TMP/SMX for Soft Tissue Infections**

Sanjay Arora MD and Michael Menchine MD

**Highlights**

- A large, multicenter, randomized controlled trial of adults and children with large abscesses (greater than 5 cm) and/or cellulitis found no significant difference in cure rate or side effect profile between clindamycin and trimethoprim-sulfamethoxazole.

- Sick patients were not included in the study.

- There was a difference favoring TMP-SMX in the abscess group and a similar trend favoring clindamycin in the cellulitis group although neither achieved statistical significance.


- This is a large, multicenter, randomized controlled trial of adults and children with large abscesses (greater than 5 cm) and/or cellulitis. The authors found no significant difference in cure rate or side effect profile between clindamycin and trimethoprim-sulfamethoxazole (TMP-SMX). However, there were some trends toward difference.

- Skin and soft tissue infections are very common. Most are treated as outpatients. There are consequences for treatment failure such as bacteremia, hospitalization, and need for more invasive procedures. In the MRSA era, clindamycin and TMP-SMX are recommended due to their low cost and high activity against MRSA and MSSA. There are few trials that compare these two medications head-to-head.

- These authors conducted a multicenter study across the country. It was a prospective, double-blinded, randomized controlled trial. Eligible patients had signs of cellulitis or an abscess for greater than 24 hours. Superficial skin infections such as impetigo were excluded, as well as infections at body sites that require specialized management (such as hand infections). Animal bites were excluded. Patients who were sick with fever or potentially immunocompromising conditions, such as diabetes or renal failure, were excluded. This was a very uncomplicated group of patients.
Before randomization, the patients were stratified into big abscesses (greater than 5 cm in adults or 3-4 cm in kids depending on age) and/or cellulitis versus small abscesses. The data presented was just for the large abscess-cellulitis group. Patients were randomized to receive clindamycin two 150 mg tabs PO twice daily or TMP-SMX one double strength tab PO twice a day with a placebo dose. Demographics were obtained. Cultures were sent if there was pus. Patients were seen at the end of treatment (ten days later), had a test-of-cure visit (another ten days later), and at 40 days for a final follow-up.

The primary outcome was clinical cure at the test-of-cure visit with resolution of the infection, no occurrence of side effects that required discontinuation of the meds, and no need for surgery or hospitalization.

524 patients were enrolled: 264 in the clindamycin group and 260 in the TMP-SMX group. There were no significant differences in demographics between the groups in terms of age, ethnicity, gender, etc. About 1/3 had abscesses, cellulitis in half, and a mixed picture in 1/5. When cultures were obtained from the abscesses, about 75% were staph and 83% of these were MRSA. The rates of cure were about 80% with clindamycin and 78% with TMP-SMX, when they used the intention-to-treat population. However, in patients who followed-up, the difference was better but still marginal (89.5% to 88.2%). There was no difference in the subgroups such as kids, abscesses, or mixed-lesions. However, every time they divided patients for subgroup analysis, the numbers of patients decreased as well as the power to detect differences. In the group with clindamycin-resistant bacteria who received clindamycin, 11 of the 15 patients still had complete resolution of their symptoms.

Adverse events were similar: 18.9% in the clindamycin group versus 18.6% in the TMP-SMX group. The most common adverse event was diarrhea, about 10% in both groups. There were no cases of C. difficile.

They concluded that clindamycin performed a little better across the board but the study was not powered to detect it, so they determined that the antibiotics were non-inferior. Of note, TMP-SMX worked well for cellulitis, which is usually a strep infection. The traditional teaching is that TMP-SMX does not work well here. The authors cited some basic science data saying that TMP-SMX may work better than we think.

However, there was a difference favoring TMP-SMX in the abscess group and a similar trend favoring clindamycin in the cellulitis group, although neither achieved statistical significance. Why combine everyone? Why give antibiotics to everyone? There are many abscesses in which we perform incision and drainage and send home without antibiotics; their cure rate will be great regardless. There are some simple cellulitis that may resolve spontaneously. The people who need antibiotics are the sick patients who were excluded from this study.

There is a nice commentary piece in this issue asking for more information.

Take-home points. TMP-SMX probably works better against strep than we think but may not be the first choice for cellulitis. Use judgment when giving antibiotics for abscesses after incision and drainage. Cover strep in any sick cellulitis patient and cover staph in any sick abscess patient.

**Paper Chase 5:** Are Steroids Effective in Allergic Reactions
Sanjay Arora MD and Michael Menchine MD

**Highlights**

- A retrospective cohort study found that giving patients steroids for allergy or anaphylaxis was not associated with decreased ED visits.
- This study was likely subject to selection bias and may underestimate steroid benefit.
- They did not study more patient-centered outcomes, such as time to resolution of symptoms or improved symptoms.
- Biphasic reactions are extremely rare.

- This was a large, observational cohort study that asserts that giving patients steroids for allergy or anaphylaxis was not associated with decreased or increased repeat Emergency Department (ED) visits.
- Allergic reactions make up about 1% of Emergency Department visits. Anaphylaxis is seen in 1 out of 300 to 400 cases. Epinephrine, antihistamines and steroids are the main treatments. Antihistamines are the first choice, and steroids are the second most popular drug. Epinephrine is reserved for more serious cases.
- The reasons for steroid use include: reducing risk of biphasic reaction, decreasing the severity and duration of symptoms, and decreasing return visits. The authors note that none of these outcomes have been scientifically tested in allergy. Even short courses of steroids can have side effects, including hyperglycemia and psychological effects.
- A study found an increase in the use of steroids from 22% to 50% from 1993-2004.

• **Do steroids prevent patients from bouncing back to the ED?**
  This was a retrospective cohort study from two hospitals in British Columbia serving about 100,000 patients a year. They identified allergy cases through their electronic health record and conducted a chart review adhering to the standards of Gilbert et al. They differentiated allergic reaction from anaphylaxis for subgroup analysis using a standard definition. They excluded patients with ACE-inhibitor-related symptoms. They then queried their electronic health record and the provincial database; this allowed them to determine if patients had returned to another Emergency Department and they were able to link 99% of the patients to the database.

• **What did they find?** During the five-year period, they identified approximately 3,000 cases and included 2,701 encounters. Steroids were given to 1,288. Some received the steroids in the ED, some received steroids via IV, and some received steroids at discharge.
  
  Overall, 5.8% of patients who received steroids had a return visit compared to 6.7% of the group without steroids. This was not statistically significant.

• **Of patients with anaphylaxis, 4.3% had a bounceback, compared to 5.6% in the control group, and this was not statistically significant.** There were a total of 5 biphasic reactions in the cohort: 4 with steroids and 1 without. This was a very small amount.

• **In an observational study, there will be selection bias.** Sicker patients are more likely to get steroids, and the groups won’t be perfectly balanced. They tried to adjust for this by using observable variables and a propensity score. This is a reasonable method, but it never completely eliminates the selection bias. This is likely an underestimate of the benefit of steroids.

• **This is a mildly patient-centered outcome.** Return visits in Canada may not be the most important endpoint but rather time to symptom resolution may be a better outcome. Some patients may have had to go to their general practitioner instead.

• **Steroids aren’t a panacea.** They don’t prevent all ED visits. They may be marginally helpful. The authors concluded that more clinical trials were needed to evaluate the true treatment effect of steroids and will likely need to evaluate other outcomes aside from ED visits.

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**Annals of Emergency Medicine**

**Annals of Emergency Medicine: Pyoderma Gangrenosum: An Inside Job**

Paul Jhun MD and Brittney DeClerck MD

**Highlights**

• There are three components necessary for a functional skin barrier: intact epidermis, healthy dermis and adequate blood supply. Compromise of any component can lead to skin ulceration.

• Consider external trauma, infection, inflammation and neoplasm in the differential diagnosis of ulcers.

• Pyoderma gangrenosum is a neutrophilic dermatosis, characterized by neutrophils infiltrating the dermis.

• Pathergy is a rapid, exaggerated skin response of pustules and blistering after very minor trauma.

**CASE**

A 23-year-old female with a history of arthralgias and inflammatory bowel disease presented to the Emergency Department with a progressive, painful, atraumatic leg lesion. It was coin-sized when first noticed two weeks prior. It slowly grew with ulceration. The patient was initially treated with antibiotics and cyclosporine, but the wound rapidly expanded, up to one inch of wound margin extension daily. It now involved most of her lateral lower leg. The patient was afebrile with normal vital signs. Initial laboratory testing was unremarkable. She was admitted for aggressive wound management and evaluation. Biopsy supported her presumptive diagnosis of pyoderma gangrenosum.

• **How do you approach ulcers?** These may be overwhelming due to the broad differential diagnosis and potentially extensive work-up. However, you can simplify them by considering the pathophysiology of ulcers and streamlining your algorithm for your differential diagnosis.

• **There are three components necessary for a functional skin barrier: intact epidermis, healthy dermis and adequate blood supply.** Compromise of any component can lead to skin ulceration.

• **Step 1: Is there an internal cause of the ulceration or an external cause?** There are many internal causes and anything leading to derangement of one of the three components could cause ulceration. External causes include trauma (for example, from chronically picking at the skin or pressure breakdown) and venom injection such as brown recluse spider bites. Take a good history.
• Step 2: What are some internal causes? Go back to your internal medicine algorithm: consider infection, inflammation or neoplasm.
  
  - Infection: there are a lot of infectious causes. Some of these are more atypical. Consider bacteria, including treponema or strep infection, such as typical ecthyma. Viruses such as herpes or coxsackie. Pseudomonal sepsis with ecthyma gangrenosum can lead to ulceration. Deep fungal infection like coccidiodymositis or mucormycosis. Depending on location in the world, leishmaniasis, amoebiasis and schistosomiasis. Mycobacteria.
  
  - What are the most common bacterial infections that cause ulceration? Streptococcus or staphylococcus causing impetigo or ecthyma. Pseudomonas sepsis causing ecthyma gangrenosum is less common but can occur.
  
  - Inflammation: there are two types of inflammation that can cause ulceration.
    
    - The first involves the vasculature. Vasculitis occurs anytime there is inflammation of the vasculature and can cause compromise leading to ulceration. Vasculitis affecting the medium-sized vessels, such as polyarteritis nodosa, microangiopathic vasculitis, Wegener’s granulomatosis or rheumatoid-associated vasculitis, can lead to ulceration. Also, consider embolic or thrombotic diseases, such as antiphospholipid syndrome.
    
    - The second type of inflammation is pyoderma gangrenosum, which is part of the neutrophilic dermatoses.
      
      - Neoplasms on the skin can cause ulceration. These are usually non-melanoma skin cancers, like basal cell carcinoma or squamous cell carcinoma. These are common and a chronic condition.

• Pyoderma gangrenosum is a rare condition. It is part of the neutrophilic dermatoses, which involve large amounts of neutrophils hanging out in the dermis. They release elastase and other enzymes, which result in breakdown of the skin leading to an unhealthy dermis and causing ulceration. It is very painful and causes much morbidity and scarring. It can be difficult to treat.

• There are classically three neutrophilic dermatoses: pyoderma gangrenosum, Sweet’s syndrome and Behçet’s disease. They have a similar pathophysiology but have different clinical manifestations.

• What are red flags in the history and physical examination for pyoderma gangrenosum? Comorbidities such as inflammatory bowel disease, arthritic conditions and hematologic malignancies. These lesions start as a small pustule and rapidly expand to a large ulcer. There is a violaceous undermined border, as the neutrophils are in the dermis and progress peripherally. The epidermis is hanging on above and the advancing process is below. The lesions most commonly appear on the legs and around stoma sites but can occur anywhere on the body.

  - Pathergy is nearly diagnostic and is an exaggerated skin response causing blistering and ulceration after very minor trauma. A pathergy test can be performed by injecting some saline under the skin and assessing the patient in 48 hours. Development of a pustule or ulceration at the site indicates pathergy. Patients with pyoderma gangrenosum and Behçet’s disease classically get pathergy.

• Pyoderma gangrenosum is a diagnosis of exclusion as there are no truly diagnostic findings aside from pathergy and classic clinical history. You need to exclude things like infection and vasculitis which can be dangerous to the patient.

• What should you do with a patient with a known diagnosis of pyoderma gangrenosum in the Emergency Department? Be gentle with them and avoid unnecessary needlesticks, debridement, and trauma. Make sure their pain is controlled. They need proper wound care, such a moisture-retaining permeable dressing, such as hydrocolloid or wet-to-dry dressings. Make sure they are managed by their dermatologist or gastroenterologist with topical or intralesional steroids or oral immunosuppressive medications.

• The patient had escalation of her immunosuppressive regimen to include methylprednisolone, cyclosporine and azathioprine with rapid improvement of the wound.

Figure. Progression of left leg pustule to ulcer, with demonstration of well-demarcated, undermined violaceous margins.

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