The Written Summary of the EM:RAP Monthly Audio Program

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Editor-in-Chief: Mel Herbert, MD
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EM:RAP
Emergency Medicine : Reviews and Perspectives

Paper Chase 1: Decision Rule for SAH

Sanjay Arora MD and Mike Menchine MD

- *Perry JJ et al. Clinical decision rules to rule out subarachnoid hemorrhage for acute headache. JAMA. 2013 Sep 25;310(12):1248-55.* PMID: 24065011.
- **Headaches make up about 2% of ED visits**. Subarachnoid hemorrhage is probably the most devastating potential diagnosis and only makes up 1-3% of these visits. It is a rare diagnosis but missing it is horrific. It is likely that they will have a clinical deterioration within 30 days that will be devastating. There is 50% mortality.
- **If you perform CT/LP in all of the patients presenting with vague headache, you will get a bunch of false positives due to traumatic taps.** Then you are committed to getting a CT angiogram and going down that pathway. This is a big problem.
- **The authors of this study looked at 3 decision rules that had already been derived.** These are fairly simple. Do a work-up if the patient has one or more of several high-risk findings.
  - **Rule 1.** Age >40yr, neck pain or stiffness, witnessed loss of consciousness or onset during exertion.
  - **Rule 2.** Age >45yr, arrival by ambulance, vomiting (>1 episodes) or diastolic blood pressure >100mmHg.
  - **Rule 3.** Age 45-55yr, neck pain or stiffness, arrival by ambulance or systolic blood pressure >160mmHg.
- This was a prospective evaluation from 10 Canadian medical centers. 2131 patients with headache were enrolled. Inclusion criteria included: no trauma, new headache that peaked in less than an hour, and GCS of 15. Patients with chronic headaches were excluded.
  - **These are the patients that you are worried about**: they will have the smallest amount of blood and be least sensitive on CT scanning. Many of the papers that have looked at ruling out SAH with CT have included all comers and patients with a lower GCS. You are not going to miss the subarachnoid hemorrhage in the patient with a GCS of 3.
- **6.2% of the patients in the study had subarachnoid hemorrhage.** They had each physician record the data points of the decision rules and then determined the sensitivity and specificity.
- **What did they find?**
  - 84% of the patients got a CT scan and only 37.5% received an LP. This seems a little low. However, it depends on what they were looking for; the paper doesn't specify that the physician was looking for subarachnoid hemorrhage.
  - **The rules did very well overall;** they were 98.5%, 95.5% and 97.0% sensitive. However, the specificity was pretty bad; 27.5%, 30.6% and 35.6%. No particular rule was that different. They were not quite 100% sensitive and they were so nonspecific that you couldn't rule out SAH.
  - **They did post-hoc analysis and determined that if they added two different variables (limited neck flexion on examination and thunderclap headache)** to the first decision rule it was 100% sensitive but only 15% specific. (i.e. 85% of patients that are positive on the screen do not have subarachnoid hemorrhage).
- **The Ottawa SAH rule says investigate if >1 high-risk variables are present:** age >40yr, neck pain or stiffness, witnessed loss of consciousness, onset during exertion, thunderclap headache (instantly peaking pain), and limited neck flexion on examination.
- **We don't know how effective this rule can be.** It is completely untested whether physicians would actually apply these rules in practice. The physicians in the study recorded the elements but didn't act on them. It is unclear if the rule would lead to an increase in CT scans; in this study only 84% were scanned. It is so nonspecific that if it was applied broadly, it perhaps would result in more CT and LPs.
- **The study did not address how many of the patients who did not receive a CT or LP were found to have SAH on follow-up visit.**
- **The take-home points?**
  - This highlights the challenge of developing decision rules for complicated diseases that are rare. They need to be 100% sensitive and they can't quite deliver. **Are we doing a good**

enough job in detecting subarachnoid hemorrhage already? If we are, we need a decision rule to help us with specificity which these do not. We need a rule to help us do fewer work-ups, not more.

- You can consider some of the data elements in these rules and add them to the list of questions you like to check off when considering subarachnoid hemorrhage. If the patient is concerning to you, this study can't tell you not to do a CT/LP on them. There is a concern that this study could be applied to headaches that are not concerning and result in more work-ups.

- **Jonathan Edlow MD, who wrote the ACEP guidelines and published a recent editorial in JAMA, said that if you have a person with rule out subarachnoid hemorrhage and you have a new generation scanner with a negative scan by a good reader within 6 hours of headache onset, you do not have to do an LP.** The number of LPs you would have to perform to pick up an additional SAH would be hundreds (maybe 600-700).

- **Additional criticism of the study.** They did not perform an LP on everyone. **What was the follow-up?** Some argue that the follow-up in centers like Ottawa is actually pretty good and the likelihood of a miss is low.

Airway Corner
Procedural Sedation

Darren Braude MD and Jim Miner MD

- **Procedural sedation usually involves giving analgesia in addition to the sedation medications. Is this the right approach?** We don't know how important pain that you don't remember is. Pain is bad for you when you have a lot of it over prolonged periods of time. Most of the research in this area is from procedures such as thoracotomies and laparotomies. Shorter procedures don't show the same physiologic effects.

- *Miner JR et al. Randomized clinical trial of the effect of supplemental opioids in procedural sedation with propofol on serum catecholamines. Acad Emerg Med. 2013 Apr;20(4):330-7.* PMID: 23701339.

  - This study was performed in response to the observation that patients receiving propofol with adequate sedation did not remember the pain of the procedure and administering opioids along with the sedation resulted in increased frequency of complications like respiratory depression.

  - **They looked at whether or not giving supplemental opioids decreased the wcatecholamine surge. They did not.**

  - This was a small study so they would only have been able to detect a big difference. They did a see a difference in the patients with respiratory depression. **Regardless of whether or not they received opioids, patients with respiratory difficulty that required some sort of intervention had the highest catecholamine surge.**

  - All of the patients received opioids up to 20 minutes prior to the procedure. This study looked at giving an additional bolus of opioids in addition to the sedation medication at the time of the procedure.

- **Are there any problems with giving pain medication until the pain is well controlled and following with the sedation medications in 2-3 minutes?** One problem is that sedatives cause more respiratory depression during the brief procedure. Many painful conditions requiring procedural sedation are less painful after the procedure. The patient has less baseline pain after the procedure. This can result in respiratory depression after the patient no longer has the respiratory stimulus from their intense pain. Providers may be impatient and not wait for peak effect, even with rapid acting agents.

- There is a theory that undertreated pain may lead to chronic pain syndromes in the future although there is no evidence to support this.

- It is reasonable to give baseline pain control to get the patient comfortable in their bed, then perform the procedure using an amnestic agent so they will not have recall of the procedure. The brief period of pain during the procedure probably does not have negative consequence relative to the consequences of respiratory depression.

- **The downside to using catecholamines as a marker is that they can increase for a variety of reasons and do not differentiate how much stress you are experiencing.** Hypoxia caused an increased catecholamine surge. However, we don't know if it is bad for you in the short term (we know it is bad for you in the long term).

- **How are painful conditions such as fractures requiring reduction managed by Miner's group?** Pain is controlled initially by using hydromorphone or morphine until the patient is comfortable in the bed. For the procedure, propofol is given at a dose of 1mg/kg in a fast bolus. For deeper sedation, patients are given an additional 0.5mg/kg every 90 seconds as needed. Once the patient wakes up, pain is reassessed and the patient is given more pain medication as needed.

- *Miner JR et al. The effect of the assignment of a pre-sedation target level on procedural sedation using propofol. J Emerg Med. 2007 Apr;32(3):249-55.* PMID: 17394986. This study randomized patients to moderate or deep sedation using propofol but found that after the first dose, both had equal chances of winding up in moderate or deep sedation regardless of what level they were randomized to. A lot of patients are deeply sedated with 1mg/kg.

- **Why not start with a smaller dose such as 0.5 mg/kg?** You could consider this. However, there was a study looking

at amnesia with propofol. *Miner JR et al. Assessment of the onset and persistence of amnesia during procedural sedation with propofol. Acad Emerg Med. 2005 Jun;12(6):491-6. PMID: 15930398.* They read patients words started about 2 minutes until about 4 minutes after the procedure. **1mg/kg was the cutoff for achieving amnesia.**

- **Dosing is based on ideal body weight.** Obese patients may only need a dose of 0.7-0.8 mg/kg and thin patients or children may need higher doses such as 1.5mg/kg.
- **Ketofol has great potential.** Ketamine is a great pain medicine. If it was developed today, it might be used as a pain medication at a dose of 0.1mg/kg rather than as a sedative. It is a good sedation drug in high risk airway patients. Ketamine at a ratio of 1:1 (propofol to ketamine) has less respiratory depression but it takes longer for them to wake up (about 10 minutes). Another option is 4:1 propofol to ketamine; this seems to have the same respiratory depression as propofol alone but less of the associated hypotension. Although studies seem to show that analgesia is not necessary with procedural sedation for brief procedures in the ED, there is less respiratory suppression with ketofol compared to opiates so there is little downside to using it. Ketamine potentiates the sedative effect of propofol without as much respiratory suppression and cardiovascular depression.
- **Downsides to using propofol?** A study of procedural sedation in critically ill patients found a higher degree of hypotension. *Miner JR et al. Procedural sedation of critically ill patients in the emergency department. Acad Emerg Med. 2005 Feb;12(2):124-8. PMID: 15692132.* In this study, etomidate was actually better than propofol in this patient population. However, Miner is now using ketamine in these patients.
- **There is no perfect drug or cocktail.** You have to match the agent to the patient and the situation.
- Propofol is best in a stable patient with a procedure that you know is going to go fast. For longer procedures, you can consider propofol with an infusion pump or combine it with ketamine.
- **You should be using capnography for deep sedation.** It is a much better monitor than O2 saturation. A patient on high flow O2 can have some pretty significant respiratory depression before the monitor will pick it up. It isn't better than someone standing there watching the patient's respiratory status but it is the next best thing.
- **What do you do when the end tidal CO2 monitor says apnea? Should you start bagging right away?** Some will use a rising CO2 level rather than apnea as an indication that there may be a problem. There are several reasons you might not need to bag the patient right away. If you have preoxygenated the patient or are using apneic oxygenation, you have some room for error before the patient becomes hypoxic. Hypoxia is what you are worried about, not the CO2. The chance of your patient developing a life-threatening acidosis from a few minutes of apnea is unlikely unless the patient has a coexisting acidosis or high ICP.
  - **If the apnea alarm goes off, it tells you to look at your patient's breathing.** Sometimes the stimulation from the procedure will increase the respiratory drive. If you are titrating your agent, you can ease up on the dosing. Maybe your patient is obstructed and will improve with airway maneuvers like a jaw thrust, head tilt, nasal trumpet or oral airway. The equipment may be dislodged or malfunctioning.
  - **What you do next depends on several things:** how well preoxygenated the patient is, how likely they are to desaturate, and how much longer until the sedation wears off. You can use the bag valve mask to deliver 1-2 breaths to ensure the patient is easy to ventilate. Then you can relax and consider watching and waiting depending on the sedation given and duration of the procedure. If not, open the airway, optimize oxygenation and consider airway adjuncts and LMA. Prepare to intubate. **Hypoxia is the big deal. The CO2 is a marker that something bad may be about to happen.**
  - **Does end-tidal CO2 monitoring decrease the incidence of hypoxic events?** Yes. *Deitch K et al. Does end tidal CO2 monitoring during emergency department procedural sedation and analgesia with propofol decrease the incidence of hypoxic events? A randomized, controlled trial. Ann Emerg Med. 2010 Mar;55(3):258-64. PMID: 19783324.* End-tidal CO2 monitoring decreased the incidence of hypoxic events by 17%.
- **Should you use an analgesic agent along with propofol when sedating an intubated patient?** Using propofol along with fentanyl is a good option. The study above was looking at brief episodes of pain associated with a procedure, not a sustained painful stimulus. The evidence showing harm due to a sustained painful stimulus is better than the evidence for short procedures.
- *Miner JR et al. Bispectral electroencephalogram analysis of pharmacologically paralyzed patients in the emergency department. Ann Emerg Med. 2004 Feb;43(2):293. PMID: 15083845.* They did bispectral monitoring on all intubated patients and found the best predictor of whether or not they were alert was heart rate.

Cardiology Corner
Unstable Angina – The New Definition

Amal Mattu MD

- *Braunwald E, Morrow DA. Unstable angina: is it time for a requiem? Circulation. 2013 Jun 18;127(24):2452-7. PMID: 23775194.*
- **Some have suggested that we should get rid of the term unstable angina due to highly sensitive troponin assays that are eliminating the diagnosis.** Highly sensitive troponins are picking up cases that we previously called unstable

angina and redefining them as NSTEMIs.

- **Disclosures.** The authors of this article have 18 disclosures, including some from the company that manufactures highly sensitive troponin assays.
- The World Health Organization's current definition of myocardial infarction still references unstable angina.
- **Some history.**
  - Heart disease was one of the big killers in the Western world. A lot of patients had underlying coronary disease and went on to have heart attacks and die. In many cases, the patients who initially had stable angina and later had a heart attack had an increase in the frequency or intensity of angina symptoms prior to the heart attack. In the 1930s-40s, physicians described what we later referred to as unstable angina.
  - **In the early 1970s, the term unstable angina was coined and began to be used on a regular basis.** This coincided with the first statement of the World Health Organization saying that to diagnose MI, you needed two of three criteria: typical symptoms, typical EKG pattern (development of Q waves), or an increase and subsequent decrease in serum enzymes attributed to myocardial necrosis.
  - **By the 1990s, unstable angina was recognized as the cause of 570,000 annual hospitalizations in the United States.** These patients were considered high risk for going on to develop myocardial infarction. It was also recognized that a lot of these patients didn't have myocardial infarction.
  - **Over the past 20 years, the definition of unstable angina has expanded with increasing ambiguity.** The number of negative work-ups started to skyrocket. The assays for cardiac enzymes were refined and improved.
  - **Acute coronary syndrome was redefined.** In the early 1990s, we used terms to describe a spectrum from stable angina to Q-wave myocardial infarction (now called a STEMI). The middle group was referred to as unstable angina and non-Q-wave MI. Non-Q-wave MI referred to patients who had positive enzymes but never developed Q waves. **Unstable angina referred to patients with negative cardiac enzymes; some had unremarkable EKGs and some had ischemic changes (clear cut ischemic T waves or ST segments).** Around this time period, the data clearly showed that if someone had positive enzymes, they had an increased risk of adverse outcomes, development of a full Q wave MI and death. The literature also showed that unstable angina with an ischemic looking EKG had a worse prognosis. Unstable angina with an unremarkable EKG was not studied well.
  - **In the past 10 years, the use of troponins has increased dramatically.** The World Health Organization uses troponins in its definition of myocardial infarction. Troponins were shown to have improved prognostic capability over CK-MB. Over the past 5-10 years, troponin assays have become more sensitive. The percentage of patients with non-Q-wave MIs (now referred to as non-STEMIs) has increased. As a result, the number of patients previously labeled unstable angina (with negative enzymes but concerning) has decreased. The troponin assays are now more likely to be positive when there is any cardiac event, even if it is a small event that used to have a negative CK-MB. **More recent data shows that if patients have negative troponins, their prognosis is not that bad even if they are diagnosed with unstable angina.**
- **The spectrum of coronary syndromes can be redefined.** At the far left are the patients with stable angina. At the far right are the patients with STEMI. In the middle group, moving from milder to more severe, we have a smaller group of patients previously called unstable angina (negative enzymes and unremarkable EKGs), a smaller group of patients previously called unstable angina (negative enzymes and ischemic EKGs) and a growing group of patients referred to as NSTEMI (positive troponins). **The data now shows if you have positive troponins, you have a worse prognosis. If you have unstable angina with an ischemic EKG, you also have a worse prognosis. We have now discovered that if you have unstable angina with an unremarkable EKG, your 30 day prognosis is pretty good.**
- **Many are questioning whether there is a need to categorize patients with unstable angina and unremarkable EKG.** Maybe these patients should be considered stable angina.
- **This article focused on troponins rather than EKG.** However, if you have patient with an ischemic EKG, you need to worry about them, even if they have negative enzymes. These patients were not mentioned in the article. They focused on positive versus negative enzymes. It is possible that we are going to get rid of concerns about patients with unstable angina and unremarkable EKGs because the 30 day risk of a bad outcome is less than 1%.
- **The groups of patients we need to worry about are:** patients who present with some type of concerning symptoms and have: a STEMI, a NSTEMI, or they have normal enzymes with an ischemic EKG. You still need to scrutinize the 12 lead EKG. Many of the medicolegal cases being brought against providers recently are for missed ACS with negative troponins and clearly abnormal EKGs.
- **Based on the current literature, it looks like it is reasonable to send troponins on your patients if you have some degree of concern.** If you do not have any concern, do not send troponins. Don't order tests that have high sensitivities and low specificities if your pretest probability is extremely low. You will get a lot of false positive troponins (this also was not discussed in the paper).
- If you look at the ACC/AHA guidelines, they have always said if you send a troponin you are obligated to do a full work-up and

take the patient all the way to stress testing. **This paper, among others, indicates it is reasonable to send a troponin in a patient in whom you are concerned has some risk and if serial troponins and EKG are negative, you do not have to go all the way to stress testing routinely.** Based on this literature, this strategy is reasonable as well as defensible.

- **This paper did not discuss if serial troponins are necessary or if a single highly-sensitive troponin is sufficient.** They also did not address the EKG. You need to take a good look at the EKG and if it is ischemic in appearance, it does not matter if the troponins are negative. They also did not address the high sensitivity and poor specificity of the newest generation troponin assays. There are many other medical conditions aside from ACS which can cause an increase in troponins. There is a concern that troponins may be ordered unnecessarily and this may lead to unnecessary admissions and work-ups.
- We are getting to the point where the next troponin assays may be positive in everyone. There is a baseline level of troponin in everyone that is normal and not due to cardiac injury. We are going to have to come up with cutoff values.

Paper Chase 2: Etomidate, Sepsis, RSI

Sanjay Arora MD and Mike Menchine MD

- *McPhee LC et al. Single-dose etomidate is not associated with increased mortality in ICU patients with sepsis: analysis of a large electronic ICU database. Crit Care Med. 2013 Mar;41(3):774-83.* PMID: 23318491.
- **This controversy started in the 1980s with a series of letters to the editor published in Lancet describing patients intubated in the ICU with increased mortality.** This was followed by some prospective studies that showed a transient decrease in the cosyntropin stimulation test. It does suppress adrenal function and this can last up to 72 hours. The question is if this affects the patient.
- Subgroup analysis in a number of randomized controlled trials including CORTICUS and KETASED appeared to show worse outcomes with etomidate.
  - *Cuthbertson BH et al. The effects of etomidate on adrenal responsiveness and mortality in patients with septic shock. Intensive Care Med. 2009 Nov;35(11):1868-76.* PMID: 19652948.
  - *Jabre P et al. Etomidate versus ketamine for rapid sequence intubation in acutely ill patients: a multicenter randomised controlled trial. Lancet. 2009 Jul 25;374(9686):293-300.* PMID: 19573904.
- **Is a patient oriented outcome or a disease oriented outcome affected?** The number on the lab test may decrease, but does this translate into increased mortality for patients? We know that patients with poor adrenal function tend to do worse. CORTICUS showed that when they are low and you give them replacement steroids, it does not help.
- **The use of etomidate in sepsis has been controversial as there was concern that suppressing adrenal function further would result in worse outcomes.** There have been some small prospective studies showing that supplementation with steroids in patients receiving etomidate had no effect.
- **This is a retrospective database study utilizing the Philips eICU Research Database including almost 20,000 sites and almost 750,000 patients.** They used a strict rule set to identify clearly septic patients who were intubated within 4-96 hours after admission. 2,014 patients met criteria and were included in the study. These patients were not intubated in the ED but the findings likely apply to the ED. 1,102 patients received etomidate and 912 received other induction agents for intubation. They looked at different features including age, gender, APACHE score and comorbid conditions and found that the two groups were essentially the same. The etomidate group was a little sicker.
- **What did they find?** Hospital mortality was similar between the two groups; 37.2% versus 37.8%. ICU length of stay was 8.7 days versus 8.9 days. Some patients did receive supplemental steroids due to their sepsis but this was similar in the two groups. They did some fancy math with regression analysis and found that patients with increased age, high APACHE score and comorbid conditions did worse. **However, when they did subanalysis looking at etomidate in the sickest patients, there was no impact.**
- **There has been one recent observational study and two randomized controlled trials looking at this in ED patients.**
  - *Tekwani KL et al. A comparison of the effects of etomidate and midazolam on hospital length of stay in patients with suspected sepsis: a prospective, randomized study. Ann Emerg Med. 2010 Nov;56(5):481-9.* PMID: 20828877.
  - *Tekwani KL et al. A prospective observational study of the effect of etomidate on septic patient mortality and length of stay. Acad Emerg Med. 2009 Jan;16(1):11-4.* PMID: 19055676.
  - *Paven JF et al. Corticosteroid after etomidate in critically ill patients: a randomized controlled trial. Crit Care Med. 2012 Jan;40(1):29-35.* PMID: 21926601.
  - **What did they find?** These studies included about 100 patients in each study. Mortality and length of stay were about the same in all of them. Giving corticosteroids showed no benefit.
- **In summary.** We are familiar with etomidate. It is hemodynamically stable in these sick patients. This is the biggest and best study to date and shows no difference although it still remains controversial as there is counterevidence available.

Note: This applies to single dose etomidate; the numbers are worse with repeat dosing.

- **It's ok to use etomidate for RSI. You are not going to kill your septic patient.** If you are still afraid to use it, there are plenty of other agents available.

Pulse Point App for Cardiac Arrest

Zack Shinar MD and Steve Brooks MD

- **The Pulse Point smartphone application was developed by Richard Price, the chief of a fire district in San Ramon, California.** He was previously a software developer. He was sitting in a café with his paramedic chief when a person in the restaurant next door experienced cardiac arrest. No bystander CPR was initiated. Unfortunately, Price was unable to assist as he was unaware of the situation until he saw a fire truck arrive. In response, they designed this application.
- **What does the application do?** When calls are placed to 911 reporting cardiac arrest, the program alerts CPR-trained citizens who have downloaded the application to their smartphones and provides a map with the location of the patient and nearby AEDs.
- **What cities are using this application?** There are approximately 10-12 communities currently utilizing this and it is growing every day. A randomized controlled trial is ongoing in Toronto, Ontario using the Resuscitation Outcomes Consortium database to determine if it is effective.
- **There are potential problems with this technology:** for example, news vans using the app and showing up for some dramatic footage. However, it is interesting.
- **Where should AEDs be placed?** They have done some research in Toronto using the Resuscitation Outcomes Consortium database to determine where cardiac arrests happen in the community and what type of buildings have an increased frequency of cardiac arrest. They found that AEDs tend to go to schools and other publicly funded buildings. However, cardiac arrests were more likely to happen at race tracks, casinos, hotels, hostels and transport hubs. Many of these higher risk sites were not covered with AEDs. They are trying to encourage data driven placement of AEDs.
- **Chest compressions.** We have all heard the recommendations for push hard and fast by the AHA. This is based on data showing that if we push too slow and not hard enough, it is directly associated with worse outcomes. The next time you go to a code in your hospital, count the rate of the compressions being delivered. They are seeing rates of compression that are as high as 140-160 beats per minute.
  - *Idris AH et al. Relationship between chest compression rates and outcomes from cardiac arrest. Circulation. 2012 Jun 19;125(24):3004-12.* PMID: 22623717. They looked at chest compression rates used by EMS and outcomes published in the Resuscitation Outcomes Consortium. **They were able to show that the best chance of ROSC occurs when the chest compression rate is 125 per minute.** If the rates are faster, the chance of ROSC decreases.
  - **Chest compressions can cause harm and if we are doing them too fast:** there is not as much output and worse outcome.
- **How long should resuscitative efforts last?**
  - *Goldberger ZD et al. Duration of resuscitation efforts and survival after in-hospital cardiac arrest: an observational study. Lancet. 2012 Oct 27;380(9852):1473-81.* PMID: 22958912.
  - This used data from the Get with the Guidelines registry. This includes data from 435 US hospitals on thousands of in-hospital cardiac arrests. They found that hospitals that tended to perform longer resuscitations had associated higher rates of ROSC and longer term survival to discharge and survival at 1 month.
  - **This study had many limitations and was a retrospective study. However, the take-home point is that a lot of arrests (>25%) achieve ROSC at durations greater than 20 minutes. Consider going a little longer with your resuscitation, taking the patient's status and comorbidities into consideration.**
- **Neuroprognostication is the idea that we could determine when someone is not going to achieve neurologic return.** We need to better assess if someone will have neurologic return. We have all heard stories of patients improving after being in a coma for 3 weeks; however, the literature on this is dismal. We do not have good data to say that a patient has no chance of neurologic return.
  - We do have BIS scores, NSE scores and neurologic exam. None of this prognosticates well.
  - Recent literature is examining the use of NIRS (near infrared spectroscopy) to evaluate regional cerebral oxygenation. This is essentially a pulse-ox of the brain. Instead of placing it on a finger, you place it on the skull base.
  - Adequate cerebral hemispheric oxygenation can be estimated by sampling the oxygen content in blood from the jugular vein. However, this requires an invasive procedure and may not demonstrate regional malperfusion despite adequate global cerebral oxygenation. **Commercially available cerebral oximeters estimate regional tissue oxygenation via transcutaneous measurement of areas most vulnerable to changes in oxygen supply and demand like the frontal cortex.**
  - There has been recent research from Japan showing patients with regional cerebral oxygenation saturation threshold values greater than 40, measured intra-arrest, had good neu-

rologic outcome. If you had a value less than 15, only 0.6% of patients had good neurologic return. *Ito N et al. Noninvasive Regional Cerebral Oxygen Saturation is a Reliable and Readily Available Neurologic Prognostic Indicator in Out-of-Hospital Cardiac Arrest Patients: A Multicenter Prospective Cohort Study. In Resuscitation Science Symposium, Los Angeles, CA November 3-4, 2012. Page 40. Abstract number 343.*

- **If we could use this to decide which patients were likely to have good neurologic outcome, we could better assess who would benefit from novel therapies such as ECMO.** A patient with a low NIRS score might not benefit from aggressive resuscitation such as ECMO but a patient with a high score might.
- **This is not ready for primetime and needs more research, but is interesting.**

Paper Chase 3: CPAP or BiPAP in CHF

Sanjay Arora MD and Mike Menchine MD

- *Li H et al. A comparison of bilevel and continuous positive airway pressure noninvasive ventilation in acute cardiogenic pulmonary edema. Am J Emerg Med. 2013 Sep;31(9):1322-7.* PMID: 23928327.
- Noninvasive positive pressure ventilation has emerged as an incredibly valuable tool for management of a large range of medical conditions in the ED such as COPD, pulmonary edema and ARDS. In acute pulmonary edema, it can improve hemodynamic function, avoid the need for intubation, and improve mortality.
- **BiPAP** delivers pressurized air (for example, at 15mmHg) into the patient's nose or mouth during inhalation. During exhalation, the pressure decreases to 5mmHg. CPAP has continuous pressure at either 5mmHg or 10mmHg. Both may be delivered via nasal masks or face masks.
- **Theoretical advantages of BiPAP.** It can aid you if respiratory muscle fatigue is an issue. This may be more relevant in COPD patients. The pressures are higher than CPAP during the inhalation phase (blowing air in) and lower than CPAP during the exhalation phase. If you are having difficulty expelling air, such as a COPD patient, it may be better to have lower pressures during the exhalation phase.
- **What is the best way to deliver noninvasive positive airway pressure: CPAP or BiPAP?** This study was a meta-analysis including 12 randomized controlled trials with 1433 patients. **They found there was no difference: the need for intubation, mortality, length of hospital stay, and myocardial infarction rates were all the same.** There has been concern that increased pressure in patients with acute pulmonary edema puts strain on the heart and might lead to increased rates of myocardial infarction.
- **However, all of the relative risk ratios tended to slightly favor the CPAP group although these were not statistically significant.**
- **One of the disadvantages of BiPAP is that patients have to synchronize with it.** If you have a patient who is really struggling with it, you can consider trying CPAP for the patient's comfort.
- **Contraindications.** Respiratory or cardiac arrest (they have to be breathing). Unconsciousness or severely impaired consciousness (it does not provide airway support). Be very cautious in patients who are high aspiration risk or unable to clear secretions.
- **Take-home points.** They both work. If patients aren't tolerating BiPAP, consider switching to CPAP or vice versa.

Mizuho Reviews Exercise Induced Syncope

Mizuho Spangler DO and Matt Baird MD

- This is focused on teenagers and patients up to forty years old but can be extrapolated to other ages.
- **Exercise related syncope is loss of consciousness associated with exercise, either during or following.** It is a type of exercise associated collapse. There are other types of exercise associated collapse that do not involve loss of consciousness. It makes up about 3-20% of syncopal cases.
- **The vast majority occurs following exercise and these are almost always benign.** These are due to neurocardiogenic causes. These usually do not have sequelae and have good outcomes.
  - This is a typical vasovagal episode with a brisk decrease in venous return. This is due to reflex bradycardia and vasodilation secondary to increased vagal tone. Although you might think you would be ramped up after an athletic event, you can become bradycardic with vasodilation. This often occurs just after the finish line in an endurance event.
  - **Vasovagal episodes can recur.**
- **Syncope during exercise should be considered a four alarm fire.** It is more indicative of an ominous cardiac cause. Patients with syncope during exercise should not be allowed to return to their sport until a full work-up has been obtained.
  - **These patients should all receive an EKG.**
  - **An echocardiogram with a cardiologist or in the ED is usually obtained.**
  - Some will undergo 24 hour Holter monitoring or electrophysiology studies.
  - Tilt table testing is sometimes used for neurocardiogenic causes.

- **Causes of true exertional syncope.**
  - Heatstroke.
  - Hypoglycemia.
  - Anaphylaxis can cause syncope although it is unusual.
  - Hyponatremia. Endurance events can lead to a loss of sodium through perspiration and dilution with free water. However, hyponatremia can also result from an underlying ion channelopathy. This is less common but does occur. These patients should have cardiac causes ruled out and may benefit from referral to a nephrologist. These are less likely to have adverse outcomes like death.
  - **Hypertrophic cardiomyopathy.** If you are lucky, they will have a murmur on exam that increases with valsalva. The EKG will have high voltage and deep narrow Q waves in the lateral leads. Occasionally there will be septal T wave inversions.
  - **Arrhythmogenic right ventricular dysplasia (ARVD).** Physical exam will be normal. EKG can be relatively nonspecific but may typically show T wave inversions in the septal leads V1-V3, a right bundle branch block and an Epsilon wave which is a terminal notch after the QRS complex due to delay in ventricular conduction.
  - **Long QT syndrome.**
  - **Brugada syndrome.**
  - **Coronary artery disease.** More underconditioned people are participating in endurance events. This is typically the most common cause of death during exercise in people older than 40 years.
  - **Myocarditis.** This is a rare cause. These patients may have evidence of heart failure on exam. Ultrasound might show global wall abnormalities. EKG may show diffuse repolarization.
  - **Valvular disease.**
  - **Wolff-Parkinson-White** and other similar conditions.
  - **Marfan's** or connective tissue disorders.
- **The syncopal episodes during exercise are essentially temporary sudden cardiac death and should be treated as such until proven otherwise.**
- **What are we responsible for in the ED?** In the emergent situation where the patient is not in extremis, the required workup is very limited. Take a careful history and physical. The most important aspect of the history is if the event happened during the exercise or afterwards. Vital signs may be helpful if they are hypotensive or significantly tachycardic. Blood glucose is important if they are altered in any way as well as sodium or a chemistry panel. Physical exam is often unhelpful; most of these young adults will look good when they get to us. Listen for a murmur. Check a neuro exam if they seem altered. **Everyone should get an EKG looking for ischemia or sequelae of the above conditions.**
- **What about energy drinks?** They are so high in caffeine and sugar that they can cause a burst followed by a drop in susceptible people. The amount of caffeine may be associated with increased risk of heat illness but there is not great evidence available.
- **What are concerning cases?** Collapse or syncope during exertion. They need to be kept out of their activity until they are seen by a cardiologist. This doesn't have to happen in the ED if they look well, are feeling well and have normal vital signs.
- **There is not a lot of literature on this topic.**
  - *Holzhausen LM et al. Clinical and biochemical characteristics of collapsed ultra-marathon runners. Med Sci Sports Exerc. 1994 Sep;26(9):1095-101.* PMID: 7808242. They studied 46 male marathoners with collapse during or after an event. 85% collapsed after the completion of event. 15% were during the event; of these 15% they found a much higher incidence of organic pathology (hyperthermia, hyponatremia or cardiac disease) on further work-up.
  - *Colivicchi F et al. Epidemiology and prognostic implications of syncope in young competing athletes. Eur Heart J. 2004 Oct;25(19):1749-53.* PMID: 15451154. This was a large screening group of 7568 athletes around the age of 16. They were doing pre-participation physical exams. 474 reported a history of syncope that was not necessarily exercise related. Only 63 (0.83%) had syncope related with exercise. Of these, syncope was post exertional in 57% and exertional in 6 patients. These 6 patients were worked up: one had hypertrophic cardiomyopathy, one had arrhythmogenic right ventricular dysplasia and four had neurocardiogenic syncope. The patients (excluding the 2 diagnosed with cardiac disease) were allowed to participate in unrestricted activity and followed for 6 years on average; there were some recurrences of syncope but no significant adverse events.
- **Syncope during exertion is rare, especially in young patients.** If it happens during exercise, it is more likely to have an ominous cardiac cause. If it is not due to a cardiac etiology, there may be recurrence but there is almost never a significant adverse event.
- **Remember that syncope rules do not take exercise induced syncope into account.** For example, the CHESS mnemonic; C=history of CHF, H=hematocrit less than 30, E=abnormal EKG, S=shortness of breath and S=systolic blood pressure less than 90. A syncope patient with any of these is considered high risk. The specificity and sensitivity are not great.

High Humidity High Flow Nasal O2

Al Sacchetti MD

- **There has been a lot of talk about high flow oxygen via nasal cannula.** There are two things that fall under this category.
  - The first is what Scott Weingart and Richard Levitan talk about when performing intubation: you use a regular nasal cannula, crank up the oxygen as high as it will go and it provides a reservoir to buy you some time during your intubation. This works phenomenally well.
  - **The second is the high humidity, high flow nasal cannula (HHFNC).** It is important to differentiate between the two.
- **What is high humidity, high flow nasal cannula?** This is a specific device that delivers oxygen at very high flow. It is not your everyday nasal cannula. The device is able to go up to 40L/min. They are able to add humidity to increase patient comfort. Heating the gas allows for increased humidity. How hot? Body temperature.
- **What patients are good candidates?** HHFNC can be used on a variety of patients. Consider it in a patient who is extremely hypoxic with increased oxygen demand.
- **The high flow helps keep alveoli open to increase oxygenation.** You may be able to use less oxygen than a non-rebreather, by turning the FiO2 down to 60-70% but keeping the flow cranked up. You can adjust the FiO2, which may be beneficial in a COPD patient. You can crank up the FiO2 in patients when you are preparing for intubation.
- **Other benefits.** It goes in the patient's nose rather than via facemask and allows the patient to eat, drink and talk. You can double-up with a non-rebreather mask. It is easier to titrate down. It does not need to occlude the nares. It provides some element of positive airway pressure.
- **What age groups can use this?** All. It is used in neonates with a lower flow. You can use this in a 3 month old with bronchiolitis who is close to intubation. It can be used in CHF. Older patients with pneumonia. Trauma patients with pulmonary contusion.
- **We should be using it more often.** How do you ask for it? Ask for the high humidity, high flow nasal cannula.
- **This does not deliver breaths and is not useful in respiratory failure.**
- In general, the literature says it is well-tolerated and effective in hypoxic patients.
  - *El-Khatib MF et al. High-flow nasal cannula oxygen therapy during hypoxemic respiratory failure. Respir Care. 2012 Oct;57(10):1696-8. PMID: 23013907.*
  - *Ojha S et al. Use of heated humidified high-flow nasal cannula oxygen in neonates: a UK wide survey. Acta Paediatr. 2013 Mar;102(3):249-53. PMID: 23167445.*

Paper Chase 4: Intranasal Ketamine for Analgesia

Sanjay Arora MD and Mike Menchine MD

- *Andolfatto G et al. Intranasal ketamine for analgesia in the emergency department: a prospective observational series. Acad Emerg Med. 2013 Oct;20(10):1050-4. PMID: 24127709.*
- **We know that ketamine is a dissociative anesthetic and can be given via multiple routes (IV, IM, PR and PO).** They looked at the intranasal route. Ketamine is frequently used for procedural sedation and we often forget that it can be used for straight analgesia at lower doses.
- **This was a prospective observational study that included both kids and adults with moderate to severe pain, defined as a score of 50 or greater on the visual analog scale.** Although there is a fear that ketamine will result in emergence reactions when used in adults, this is not based in science. Some people do have bad experiences, but this is a medication that is used worldwide, often as the only anesthesia for surgery in undeveloped countries.
- **Patients received somewhere between 0.5-0.75 mg/kg of intranasal ketamine.** Normal sedative dose is 1-1.5mg/kg.
- The idea for this research stemmed from the military, which was looking for an easy way for medics in the field to give analgesia to injured soldiers but still keep them awake enough to assist in extrication. The goal of this study was to improve the door to analgesia time. Providing ketamine intranasally for pain control may be easier and faster than waiting for a bed, placement of an IV and administration of morphine.
- **The pain scores were reassessed every 5 minutes for 30 minutes and then every 10 minutes for a total of an hour.** They looked for a clinically significant reduction in the visual analog pain score defined as a drop of 13.
- **The mean age of the patients was 47 years and they ranged from 11 years to 79 years old.** They found 88% of the patients had a clinically significant reduction in pain within 30 minutes and the median change in pain scores was 34. The median time to reduction of 13mm was 9.5 minutes.
- **They found no serious adverse events that required intervention.** There were some minor adverse events such as mood changes, a few people had hallucinations (these weren't well described but seemed to be minor and not major freak-outs) and one person had "trouble hearing."
- **This is preliminary. It was a small sample size and there was no placebo or comparison drug but it clearly worked.** It is easy to see the advantages over waiting for a gurney, IV and morphine. The authors are planning a randomized controlled trial. It also may be something to consider in an opiate dependent patient who has acute pain and requires high doses of narcotics for pain relief.

- **How was the ketamine actually administered?** There are different commercial devices. They used the MAD (mucosal atomization device). The medication is aspirated into the syringe with a little extra (about 0.1cc) due to some dead space. The syringe, with the atomizer on the tip, is put in the nostril and aimed slightly up and out (towards the ear). You want to get it on the mucosa, not the septum. The dose of the medication is split between each nostril. About 1/3 cc up each nostril is a perfect dose. 1cc per nostril is the max dose. It reaches therapeutic levels in the bloodstream relatively quickly.

Notes from the Community Palliative Care

Rob Orman MD and Jean Abbott MD

Case

You are working a busy shift on Friday night in the ED, when the medics bring a 90 year old female with fever and altered mental status. Within a few minutes, it is apparent she is in septic shock. She has no protective airway reflexes. She has a POLST form that requests comfort measures only. The family arrives and reports that she has been demented and bedridden for several years and they would like her to die peacefully. You are about to start a morphine drip for her comfort when the nurse tells you, "Doctor, there is a phone call for you". It is the patient's daughter from California. You explain the situation, the grave prognosis and the plan for comfort measures only. **"You are just going to let my mother die? I want everything done!" This scenario is never easy.**

- **When it is clear that the patient is going to die, how do you start off?** The patient may be obviously dying: they are not responsive, they have stopped eating and their systems are shutting down. They may be less obviously dying; you may be sitting there thinking, "This does not look good." **Start off by listening to the family and hearing what they think is going on.** Some will be aware of what is going on – "Oh, we were expecting this." Others may be puzzled and need you to take the lead.
- **Advanced directives come in lot of flavors.** They are helpful as they describe what the patient's values are and what the patient would want in certain circumstances. However, there are a lot of ways that people can die and usually these are not addressed in the advanced directives. Advanced directives can serve as a guide, but a lot of times they aren't pertinent to the situation at hand.
  - **The POLST (Physician Orders for Life-Sustaining Treatment) or MOLST (Medical Orders for Life-Sustaining Treatment) is a set of orders written by a physician and signed by the patient or a surrogate.** These need to be followed. You are legally protected if you follow these orders. These are different than advanced directives that describe the kind of treatment they want done and can be nebulous.
- **With the clearly dying demented, bed-ridden patient, most physicians would opt for comfort care.** Often times, the family is not on the same page. We can use language to suggest that making the patient more comfortable is more appropriate than critical care or "doing everything". **Can you make a decision that overrides the family without their consent?** Not unless you happen to have the POLST/MOLST orders sitting in front of you.
  - **Advanced directives give us guidance and can help open the conversation.** You can say to the family, "It seems from their advanced directive, they want to be kept comfortable or they want to have heroics. Help me determine how this will play out." In general, your patient is the most important and you need to do the best you can to assist the family in respecting their wishes.
- **The do-everything-trap is an interesting one and we often bring it upon ourselves.** We ask the family, "Do you want us to do everything?" For the family, "doing everything" is equal to love. We need to break this linkage for them. Don't ask it. If the family asks you to do everything, ask them "What do you mean by do everything?" We need to have a more goal-focused discussion than procedure-focused discussion.
  - Rather than running through a menu of treatment options ("Would you like CPR, intubations, antibiotics, transfusions, etc.), we need to find out what was important to the patient, their values and what they were most afraid of. Were they afraid of living on a ventilator in a nursing home? Then you as the emergency physician can assist in determining what procedures, if any, will achieve those goals and what treatments will not be beneficial.
- **When you come to a point where a decision needs to made and you do not have a POLST form, is this something you ask the patient or tell the patient (in the form of asking)?** Tell the patient in the form of asking. Families don't know what they want. They do not understand a lot of the technology we have available. In the ER, we can go full court press very easily. Sometimes it is difficult to not go full court press; it is fun for us and it is what we do best. Things that you can say:
  - **"She seems to be actively dying. I'm not sure that we have any treatments that will be able to pull her out of this. Is this something that you were expecting or is it unexpected for you?"** This helps you understand where the family is coming from and it also makes it permissible to do less than everything. Often the family does not realize that they have choices about what to do.

- **Saying something like, "In this situation, a lot of families might want us to focus on making her comfortable and avoid invasive treatments that are likely to cause more suffering than benefit."** This tells them it can be normal and loving to do less than full-court press.
- **Sometimes the family may react badly to suggestions of doing less than the full court press: "Why wouldn't you treat her or give her antibiotics?"** You can say, "Because in my experience, it is unlikely to be of benefit." "Based on her prior instructions, it sounds like she would have wanted to be comfortable."
- It is nice to explore why people want everything done: they may not be ready, they may be afraid, sometimes they have suspicion of the healthcare system due to cultural or economic basis. This can take a lot of time. Don't expect to arrive at a decision in the ER. However, you can encourage them and you can give them recommendations based on your experience.

- **What should you do if there is a serious disagreement between paperwork requesting comfort care and the family at the bedside?** "I'm sorry. This must be very hard. My job is to honor your mother's wishes. She seems to be actively dying right now and she has told us what is important to her. I would like to respect that and not cause her more suffering."
- **Everyone is in agreement, but there are a lot of relatives who are out of town and they request that you keep her alive until they can come say goodbye; this may mean intubation, pressors, antibiotics and ICU admission.** Sometimes you can finesse it: for example, using CPAP, fluids, antibiotics. This is an individual negotiation of the situation you are in. "I don't want to cause her suffering. Why don't we try some conservative measures? It is important to respect her. Let's try to keep things going for a little longer as you are also my patient." If it is a reasonable family who really wants to be there to say goodbye, it is not wrong to admit the patient, do CPAP or even intubate with a plan to extubate in the ED or in the ICU.
- **Everyone is in agreement until the daughter from California calls (the metaphorical seagull that flies in from the coast and craps all over everything).** It is usually the person with a lot of guilt who hasn't seen the patient for 10 years and has a lot of baggage. It is important to take leadership. "Wow, this must be hard. I am obligated to honor your mother's wishes. I'm sorry to have to give you information over the phone."
- **How can you describe comfort care to the family?**
  - Don't use phrases like, "There is nothing more we can do." There is always something we can do.
  - Don't say, "We are going to withdraw care". We withdraw treatments that are unwanted or not beneficial.
  - Stress the positives. "I think it is important we put your mother in the hospital because we can aggressively treat her symptoms. We can make her comfortable and allow you some last hours to be with her."
- **How can you describe a morphine drip?** Families may think that this is what is killing the patient. It takes very small doses of morphine (1-2mg) to relieve respiratory distress at the end of life. Your goal is to keep the patient from suffering. Enlist the family to give you feedback as to whether it is too much or if they need more. "This is to relieve her suffering. I am giving her small doses. This will not hasten her death. It will make her more comfortable in her last minutes or hours." The doses used for respiratory distress do not depress mentation. If it does, you can titrate it down.
  - **You can start with 1-2mg IV and see how it works.** If you are able to start a drip in the ED, you can consider doing so.
- **The art of code status discussions.** It is important to have an informed discussion. **Offering a patient a choice between "If your heart stops, do you want us to put a tube down your throat, pump on your chest and give you a bunch of drugs to bring you back … or would you rather die peacefully?" is threatening.**
  - "We have you stable in the ER right now. When you go upstairs, if your heart should stop, what would you want people to do? CPR is an option but it is rarely successful, particularly in a patient with severe disease like you, and it is uncomfortable. I would recommend that you not have CPR because at your stage of illness, when your heart stops, it really says that things are over."
  - It can help our colleagues on the floor to have the paperwork done in the event that there is an arrest. However, this is not really our job in the ED.
  - The term DNR can be very emotional for the family and can derail your discussions with them about things that are more important for us like goals of treatment.
  - **DNR orders are often interpreted as do not treat.** Patients with DNR orders have been shown to receive different care in the hospital. Families may be afraid that the patient will not get the desired care if they sign a DNR form. Beware of pushing the DNR order.
  - **We can prime the patient and the family even if we can't get them to understand that DNR orders would be appropriate.** "Somebody upstairs is going to ask you about this and you need to think about it. My recommendation is that CPR would not be appropriate. While we are doing comfort care and other important things, once her heart stops, she has died and I would not try to restart it by doing CPR. It is unlikely to be successful."

- **Can a young suicidal patient say they want to be no code?** No. We think that suicidal ideation is a transient state and is treatable. When somebody attempts suicide, even if they have advanced directives, we resuscitate them in the ED. The law says that you are not allowed to use advanced directives as a mode for killing yourself.
- **We need the humility to realize that we may not know if a trial of treatment will be appropriate or not because we are not good at prognosticating with absolute certainty.** If you encounter resistance from the family, you can suggest, "Well, let's give a trial of treatment. The doctor upstairs will revisit this subject with you if this doesn't work." We don't have all the answers.
- *Bailey C et al. Trajectories of end-of-life care in the emergency department. Ann Emerg Med. 2011 Apr;57(4):362-9. PMID: 21131103.* He notes that we love spectacular deaths in the ED but we aren't really good at "subtacular" deaths. These "subtacular" deaths are difficult and ambiguous. Our job is to not admit to the ICU unnecessarily and overutilize resources, but also to determine if someone may benefit from a floor bed and palliative care if they are dying.
- **The trial of treatment is a good option to have.** Often these discussions can feel like it is all or nothing. The trial of treatment says, "Let's meet on the middle ground and see how she does in a few hours or a few days." You can help prepare the family, "Let's do a trial of treatment and see if this pneumonia is something her body will be able to overcome. If, after a few days, she is not improving, we should consider extubating her and making her comfortable." This can help families have appropriate expectations.
- **"Do you want us to allow a natural death?"**

Bonus Section

Paper Chase 5: Ultrasound for Soft Tissue Infections

Sanjay Arora MD and Mike Menchine MD

- *Marin JR el al. Emergency ultrasound-assisted examination of skin and soft tissue infections in the pediatric emergency department. Acad Emerg Med. 2013 Jun;20(6):545-53. PMID: 23758300.*
- This article highlights a convergence of two major trends in emergency medicine: 1 – Skin and soft tissue infections are increasing in frequency and MRSA is often the culprit with its associated abscesses; 2 – People are looking for procedures to do with ultrasounds.
- **Given the high prevalence of MRSA, there may be an underlying abscess in patients presenting with red, swollen skin infections that is not clinically evident. Can ultrasound help us determine if there is an abscess?**
- This was a prospective study comparing clinical exam with bedside ultrasonography and clinical exam in a single pediatric ED. The average age was 7 years. These were normal kids and their abscesses were in a normal distribution.
- The treating physician examined the patient and determined if there was an abscess. They decided if there definitely was an abscess, definitely wasn't an abscess, or they weren't sure. Then an ultrasonographer examined the patient and used the ultrasound to determine the presence of an abscess (yes, no, or maybe).
- They looked at 378 lesions. Overall, the clinical examination did very well: 87% sensitive and 71% specific. They did miss 13% of cases. The doctors weren't sure about 20% of the time. The ultrasound did exactly the same overall: 87% sensitive and 72% specific. **Ultrasound didn't add to the sensitivity and specificity when used in all comers.**
- **In cases where the clinician was unsure (85 cases), the ultrasound was only 77% sensitive and only 64% specific.** Ultrasound appears to add very little in the majority of cases.
- If you think there is an abscess there, just do it. You don't need to use ultrasound. If you don't think there is an abscess there, don't do it. **If you are on the fence, you can use ultrasound but realize that it isn't as good as you think it is. It will be wrong about a third of the time.**
- **This contradicts the findings of another paper.** *Squire BT et al. ABSCESS: applied bedside sonography for convenient evaluation of superficial soft tissue infections. Acad Emerg Med. 2005 Jul;12(7):601-6. PMID: 15995090.* They found 98% sensitivity of ultrasound and 88% specificity.