February 2016 | Volume 16 Issue 2

EM:RAP
Emergency Medicine : Reviews and Perspectives

www.emrap.org

February Introduction

Rob Orman MD and Anand Swaminathan MD

Take Home Points

- **Intrascalene nerve blocks may result in phrenic nerve dysfunction with unilateral diaphragmatic paralysis and respiratory distress.**
- **We should start using the term massive PE without hypotension rather than hemodynamically stable.**
- **Oxygen saturation of 85% is concerning for a large decrease in the amount of oxygen bound to hemoglobin.**

CASE

A 70 year old female was brought in with a complaint of shortness of breath. Respiratory rate was 28 and oxygen saturation was 88% on room air. The patient presented three hours after a right shoulder arthroscopy. Surgery and shortness of breath? Should you do a d-dimer? CT angiogram?

- **This was an outpatient procedure and not performed under general anesthesia.** The patient would be unlikely to form a blood clot in such a short period of time. However, the patient had received an intrascalene nerve block.
- **There are two major complications associated with intrascalene nerve blocks; pneumothorax and unilateral phrenic nerve paralysis.**
- The patient likely had a transient phrenic nerve dysfunction causing unilateral diaphragmatic paralysis. This is not a big problem for young healthy patients who are able to compensate. However, it can be a problem in older patients with limited cardiopulmonary reserves.
- EKG was unremarkable. A chest x-ray showed an elevated right hemidiaphragm. Don't send a D-dimer or do a CT angiogram in this situation.
- The patient was placed on oxygen and had improvement in symptoms and oxygen saturation. The block had been performed with bupivacaine and was anticipated to last for 6-8 hours.

Pharmacology Rounds: Tramadol and Death from NSAIDS

Rob Orman MD and Bryan Hayes PharmD

Take Home Points

- **NSAIDs have the potential to increase the risk for heart attack and stroke. Patients with pre-existing disease are at increased risk.**
- **Naprosyn seems to be the safest of the NSAIDs.**
- **Tramadol is a synthetic codeine analog and very weak mu receptor agonist with an affinity 1/6000 of that of morphine.**
- **Tramadol use may result in seizures, abuse and withdrawal.**

- **Do non-aspirin NSAIDS increase the risk of stroke or MI?** Patients are asking about this. There were three COX-2 inhibitors marketed a while ago; celecoxib, valdecoxib and rofecoxib. The last two drugs were withdrawn from the market. There are randomized trials that show the COX-2 selective NSAIDS increase the risk for MI, stroke and thrombosis although they decrease the risk of gastrointestinal complications. The risk appears to extend to older NSAIDs that are more selective for COX-2 such diclofenac and meloxicam.
  - **Because of this data, regulatory agencies have concluded that NSAIDs have the potential to increase the risk for heart attack and stroke.**
  - Patients with pre-existing disease are most at risk for cardiovascular disease or thrombosis.
  - **There was a study published in Lancet in 2013 that found major vascular events were increased by about a third due to coxibs.** However, this was a small effect and chiefly due to an increase in major coronary events. Ibuprofen also significantly increased major coronary events but not major vascular events. Compared to a placebo, if you had 1000 patients taking a coxib or diclofenac for a year, three more patients had a major vascular events, one of which was fatal.
    - *Coxib and traditional NSAID Trialists' (CNT) Collaboration, et al. Vascular and upper gastrointestinal effects of non-steroidal anti-inflammatory drugs: meta-analysis of individual participant data from randomised trials. Lancet. 2013 Aug 31;382(9894):769-79.* PMID: 23726390.

- This was followed by an FDA warning. Naprosyn seems to be the safest out of all of the medications. It did not seem to increase the risk of vascular deaths in the meta-analysis.
- **Should patients stop taking non-aspirin NSAIDs? Is it a cumulative risk?** The increased risk of heart attack or stroke can occur as early as the first few weeks of using an NSAID. However, the risk is greater with longer use and higher doses. In general, patients with pre-existing heart disease or cardiac risk factors have a greater likelihood of heart attack following NSAIDs than patients without risk factors. Use NSAIDs in lower doses and for shorter time periods.
- **A 60 year old diabetic with a history of previous MI has sprained their ankle.** The patient does not want narcotics. Start the patient on acetaminophen first. If their pain is still not controlled, the patient could take an NSAID for a few days. Naprosyn was associated with the lowest risk. If the patient still needed a stronger pain medication, lower dose narcotics could be used.

- **How does tramadol work?** It has been used as an alternative to opiates but there is recent data suggesting that we should limit its use as well secondary to concerns about abuse and addiction potential. The FDA and DEA finally changed the schedule of tramadol to a schedule 4 controlled substance - similar to a hydrocodone product. There is a risk of addiction with this drug.
  - Tramadol is a synthetic codeine analog. It is a very weak mu receptor agonist (the affinity is 1/6000th of morphine). It also has norepinephrine and serotonin reuptake properties similar to the SNRIs. Some early studies show that it can be as effective as morphine in the treatment of mild to moderate pain. However, it is not very effective for severe or chronic pain. It has an active metabolite that adds to the analgesia property. It has its side effects, although respiratory depression is less than with other opiates. Tramadol can cause seizures or exacerbate seizures.
  - **Can patient withdraw from tramadol?** There is some data to support that patients can withdraw from tramadol. However, it seems to be less severe than the withdrawal associated with opiates.
  - **What is the abuse potential?** Tramadol can be abused. In 2013, there were 6500 exposure calls to the Poison Center related to tramadol. 4500 calls were in patients older than 13. There were 4 deaths and 200 major outcomes (for example, disability or ICU stays). This is probably an underestimate as many events go unreported.
  - **Tramadol isn't great at relieving pain and there is still a risk of dependence and abuse.**

Pharmacology Rounds: Antidepressants + NSAIDS = Bad?

Rob Orman MD and Bryan Hayes PharmD

Take Home Points

- **Although a study found an association between the use of NSAIDs with antidepressants and increased risk of intracranial hemorrhage, it does not show causation.**
- **An undetectable serum acetaminophen level drawn over an hour after the ingestion does not require additional work-up.**
- **Acetaminophen levels drawn within one hour post ingestion are unreliable.**
- **If you have an acetaminophen level between 0 and 100ug/mL drawn 1-4 hours post ingestion, you need to repeat the level at four hours and plot it on the nomogram.**

- **Do NSAIDs + antidepressants increase the risk of intracranial hemorrhage?**
  - *Shin, JY et al. Risk of intracranial haemorrhage in antidepressant users with concurrent use of non-steroidal anti-inflammatory drugs: nationwide propensity score matched study. BMJ. 2015 Jul 14;351:h3517.*
  - This was a retrospective, nationwide, propensity score matched study conducted in Korea. They took patients that were on the two drugs together and matched them with patients who weren't. The main outcome measure was time to first hospital admission with an intracranial hemorrhage within thirty days of drug use. They included 4 million patients in this study.
  - **They found the 30 day risk of intracranial hemorrhage was higher for the combined use of antidepressants and NSAIDs than for the use of antidepressants without NSAIDs.** The hazard ratio was 1.6 with a statistically significant confidence interval. They didn't find any meaningful differences between the different classes of antidepressant drugs.
  - They concluded that the combined use of antidepressants and NSAIDs was associated with a higher risk of ICH within 30 days of combining those drugs.
  - **However, the data doesn't show causation.** There is an issue of external validity as the study was published in a relatively uniform population in Korea. The rate of intracranial hemorrhage with the use of these drugs in isolation is unknown.
  - **There is no biologically plausible mechanism to explain why this combination is an issue.** The study did not have a control group of patients only on NSAIDs. Is the combination of medications or the use of an NSAID responsible?

- The rate of intracranial hemorrhage in patients taking antidepressants was 1.6/1000 patient years. When antidepressants were combined with NSAIDs, the risk rose to 4.1 hemorrhages/1000 patient years.
- If you start 3000 men on an NSAID while on an antidepressant, only 1 will have an avoidable ICH in the first 30 days.
- **We need additional data before changing practice.**

- **The utility of pre-4 hour acetaminophen levels in acute overdose.** We rarely know the exact time intervals regarding ingestion. Labs and toxicology panels are often sent on patient arrival. **What do you do with an elevated level obtained prior to four hours after ingestion?**
  - **Can acetaminophen concentrations of less than 100 mcg/mL obtained between 1-4 hours after an acute ingestion accurately predict a non-toxic concentration at 4 hours?** If the acetaminophen level is drawn after an hour post ingestion, an undetectable level does not require additional work-up. A level obtained prior to an hour post ingestion is not always accurate.
  - **What if the level is moderately elevated?**
    - 83 patients with a level of <100ug/mL between 1 and 4 hours post ingestion were included in a study. They found 2 cases that had a subtoxic level prior to 4 hours that was toxic after repeat at 4 hours. This was a negative predictive value of 98.8% with a false negative rate of 6.5%. They concluded that a 6.5% miss rate is unacceptable.
    - *Froberg, BA et al. Negative predictive value of acetaminophen concentrations within four hours of ingestion. Acad Emerg Med. 2013 Oct;20(10):1072-5.* PMID: 24127715.
    - A second study reported in abstract form found that the negative predictive value under an hour was 76%. This rose to 88% between 1-2 hours, 98% between 2-3 hours and 99% between 3-4 hours.
    - *Douglas, DR et al. APAP levels within 4 hours: are they useful? Vet Human Toxicol 1994;36:350[abstract].* Open Access Link
  - **If you have an acetaminophen level between 0 and 100ug/mL drawn 1-4 hours post ingestion, you need to repeat it at four hours and plot it on the nomogram.**
  - A study of 520 cases found that only 59% of patients had a second level drawn at 4 hours.
    - *Seifert, SA et al. Acetaminophen concentrations prior to 4 hours of ingestion: impact on diagnostic decision-making treatment. Clin Toxicol (Phila). 2015;53(7):618-23.* PMID: 26107627.
  - **Take-home points.** The Rumack Matthew nomogram needs to be utilized starting 4 hours after acute ingestion. Acetaminophen levels drawn prior to four hours can lead to unnecessary treatment, admissions and adverse effects if not repeated. If an acetaminophen level is drawn before four hours, a second level must be drawn at 4 hours unless the acetaminophen concentration is undetectable more than hour after ingestion. The current data supports waiting 4 hours to draw the level. If you are suspicious but the level won't be back until after 8 hours post ingestion, you can start the NAC and discontinue it if ultimately unnecessary.

Medical Myths: The Loop Diuretic

Anand Swaminathan MD and Haney Mallemat MD

Take Home Points

- **There is no role for the administration of loop diuretics early in the resuscitation of patients with acute pulmonary edema.**
- **Immediate care should include non-invasive positive pressure ventilation and nitroglycerin.**
- **Patients with end-stage renal disease may need dialysis.**

- **Furosemide isn't necessarily good for all patients with acute pulmonary edema, especially at the beginning of care.**
- **Acute pulmonary edema is a heterogenous disease.** Much of our understanding is based on a cardiorenal model of acute pulmonary edema from the 1940s. Decreased blood flow to the kidneys leads to renal dysfunction. This causes retention of fluid and volume overload. In this situation, loop diuretics make sense.
  - **However, about 50% of the patients we see with acute pulmonary edema are euvolemic.** They don't have extra fluid but their fluid is shifted to the wrong place.
- **What is the neurohormonal activation model?** Decreased stroke volume and cardiac output leads to the release of a variety of substances such as norepinephrine (to increase the cardiac output and improve blood pressure and afterload) and renin-angiotensin-aldosterone (to improve salt retention from the kidneys and increase vascular tone).
- **Is there any evidence to support the neurohormonal activation model?**
  - While many patients with acute pulmonary edema have increased cardiac filling pressures, most did not have an increase in their dry weight on presentation. How can you be fluid overloaded if your dry weight is the same? *Zile, MR et al. Transition from chronic compensated to acute decompensated heart failure: pathophysiological insights obtained from continuous monitoring of intracardiac pressures. Circulation 2008 Sep 30;118(14):1433-41.* PMID: 18794390.

- **Where does the fluid in the lungs come from?** Probably the splanchnic circulation. This is a huge reservoir of blood that can rapidly release up to 800mL of blood into circulation in response to the release of neurohumoral mediators.
- **Loop diuretics are fairly harmless. We don't know who is volume overloaded and who is experiencing a shift of fluid, so why not give everyone a dose?** Furosemide can be harmful.
- **Consider the patient presenting in severe respiratory distress; diaphoretic, tripoding, B-lines and fluid overload on x-ray. This patient is trying to stay alive. What is happening with their physiology?** They have increased secretion of norepinephrine. The blood flow is going to their heart, brain and diaphragm. They have vasoconstriction of the blood supply leading to other organs such as the kidney.
  - **Furosemide won't work in this situation due to vasoconstriction.** We have been taught to increase the dose if there is no response. This will result in large amounts of furosemide in the circulation. Once they start to vasodilate, the furosemide will affect the kidney and cause diuresis. Now you are taking a patient who was euvolemic to start with and making them hypovolemic. This increases length of stay in the hospital and adverse complications.
  - **Furosemide decreases GFR, activates the renin-angiotensin-aldosterone system, decreases cardiac output and increases afterload early after administration.**
    - *Marik, PE et al. Narrative review: the management of acute decompensated heart failure. J Intensive Care Med. 2012 Nov-Dec;27(6):343-53. PMID: 21616957.*
- **This does not mean that you should never give furosemide to patients with CHF. However, there are some other therapies you should start first.** If the patient looks volume overloaded, it is ok to give them furosemide. Try to avoid front loading them with furosemide and doubling or tripling the dose. It doesn't work and can harm the patient.
- **The NAP mnemonic.**
- **Non-invasive positive pressure ventilation.**
  - **This is CPAP or BiPAP.** There is some evidence suggesting BiPAP is better for these patients. What does it do in acute pulmonary edema? Decreases the patient's work of breathing. Stents open collapsed alveoli and leads to better gas exchange. It decreases afterload on the heart and supports respiration. A number of papers have shown a reduction in ICU admissions (92% to 38%) and intubations.
  - **You need to start non-invasive ventilation as soon as the patient hits the door or even by EMS in the field.** Even if it doesn't stave off intubation, it will help you preoxygenate the patients so the patients don't crash during RSI.
- **Nitroglycerin.**
  - You want to start this as soon as the patient hits the door.
  - A study on high dose nitroglycerin found boluses up to 1 g of nitroglycerin did not result in many adverse events (only 1 patient or 3.4% developed symptomatic hypotension with high dose nitroglycerin). Most don't usually give boluses this large, but some will give a bolus of 300-400mcg. *Levy, P et al. Treatment of severe decompensated heart failure with high-dose intravenous nitroglycerin: a feasibility and outcome analysis. Ann Emerg Med. 2007 Aug;50(2):144-52. PMID: 17509731.*
  - **Nitroglycerin is recommended for all patients with acute pulmonary edema.** It reduces their preload and causes vasodilation. It takes the fluid away from the heart and improves performance of the left and right ventricle).
  - **Starting doses are 100mcg/min.** If the patient's pressure drops, you can turn off the drip and it is short-lived.
  - **It also decreases afterload.** These patients are clamped down with increase sympathetic tone and high blood pressure. The heart is trying to squeeze against this afterload.
  - **How do you titrate the nitroglycerin?** 50mcg at a time every 10-15 minutes while you are standing at the bedside. What is the maximum dose? Until the blood pressure drops. You are titrating to symptomatic relief. Some patients may start improving within 15-20 minutes.
- **ACE inhibitors.**
  - **These aren't used routinely but make sense.** They reduce the afterload and improve forward flow. ACE inhibitors also work on the kidney to vasodilate the afferent arteriole and allow improved perfusion. These are less studied in acute pulmonary edema but there is some available evidence.
  - Hamilton found that patients who received sublingual captopril in addition to standard therapy were more comfortable and had a decrease in respiratory failure that was not statistically significant.
    - *Hamilton, RJ et al. Rapid improvement of acute pulmonary edema with sublingual captopril. Acad Emerg Med. 1996 Mar;3(3):205-12. PMID: 8673775.*
  - Haude found that sublingual captopril improved cardiac index and stroke volume versus nitroglycerin in a small study.
    - *Haude, M et al. Sublingual administration of captopril versus nitroglycerin in patients with severe congestive heart failure. Int J Cardiol. 1990 Jun;27(3):351-9. PMID: 2112516.*
  - **If you are in place that has sublingual captopril, you can consider giving a small dose.** If you are in a place that has enalaprilat, you can give that. Mallemat will give a small dose if the patient remains hypertensive despite high doses of nitroglycerin (250-300mcg/min).

- **Many patients with acute pulmonary edema have end stage renal disease.** Most of these patients are volume overloaded. Furosemide won't be sufficient. These patients will need dialysis. Call your renal consultants early.

Bougie Every Intubation

Reuben Strayer MD

Take Home Points

- **The bougie is designed to pass through the vocal cords easily. The endotracheal tube is designed to be a conduit between the trachea and ventilator.**
- **Practice using the bougie in situations where you don't need it.**
- **The bougie can also be placed through an intubating LMA or cricothyrotomy.**

- **One of the scariest clinical scenarios is the anatomically difficult airway, where despite your best attempts at laryngoscopy, you still do not see cords.** There has been an explosion of devices and maneuvers that makes this situation less likely; video laryngoscopy, hyperangulated blades, ear-to-sternal-notch positioning, external laryngeal manipulation, etc.
- **There is a device that has been arounds for decades and is scientifically proven to improve your intubation success rate in difficult intubations.** It is cheap, available and easy to use. **It is the gum-elastic bougie.**
- This is a 60 cm long, 5 mm wide, semi-rigid tube with the distal tip bent at 30 degrees. It is a long flexible stylet that can be inserted into the trachea and an assistant can place an endotracheal tube over it. The bougie was invented over 50 years ago. **It is not routinely used in most emergency departments and this is a shame.**
- **Endotracheal tubes are designed to be a conduit between the trachea and the ventilator.** They are not designed to pass through the cords easily. The bougie is designed to be as easy as possible to pass through the cords. The literature unequivocally demonstrates that you are more likely to intubate successfully if you use a bougie, especially in difficult intubating conditions.
- **The bougie is a superior intubating device compared to a styleted endotracheal tube for three reasons.**
  - The bougie is half the diameter of the endotracheal tube.
  - It has a deflected coude tip engineered to get you where you need to go. You can slide it under the epiglottis or nudge it over the interarytenoid notch.
  - It can be molded and will hold its shape. If you are trying to advance the bougie and it is going too anterior or posteriorly, remove it and change the bend.
- **The bougie is an excellent adjunct to the video laryngoscopy with hyperangulated blades where the epiglottis is often easily visualized on the screen but hard to reach with the endotracheal tube.** Mold the bougie into the shape of the blade you are using.
- **The bougie gives you immediate feedback as to whether you are in the trachea or esophagus.** As you transmit the bougie through the cords, you often feel the coude tip slide over the tracheal rings. It will reach a stop point at the carina. If you are able to advance the bougie past the 50cm mark, you are in the esophagus. Put a lot of lube on the tip. You can also lube the cuff of the tip.
- **Don't take the laryngoscope out once the bougie is in the trachea.** This makes it harder to pass the endotracheal tube over the bougie. Leave it in place until the endotracheal tube is in place.
- **Sometimes the bougie may become caught at the laryngeal inlet and you can't advance it past the cords into the trachea.** It may often be overcome by rotating the coude tip 180 degrees so that it points posteriorly. This disengages the distal edge from whatever is blocking it and allows the bougie to proceed into the tracheal unimpeded.
- **The endotracheal tube can also get stuck on glottic structures as it is placed over the bougie.** Pull the tube back a few centimeters and rotate it, then re-advance. The tube will slide.
- **If you are good with the bougie, you can intubate a Cormack-Lehane Grade 3 view where you only see epiglottis and no glottic structures.** You don't get good at using the bougie if you only use it in a crisis.
- **Use a bougie for every intubation.** Get good at using the bougie in cases where you don't need it so you are ready for when you do need it. When you have done a few normal airways with the bougie, practice for grade 3 airways by getting the best view and then relaxing your lift on the laryngoscope so the epiglottis falls down and covers the glottis. This simulates a Grade 3 view. You can then attempt to intubate this with a bougie.
- **The bougie can also be used through an intubating LMA or cricothyrotomy.** There are case reports of using the bougie to facilitate blind digital intubation if you need to intubate someone sitting upright in the seat of a recently smashed car.

Atrial Fibrillation ADP – Part 1: Cardioversion

Rob Orman MD and Cameron Berg MD

Take Home Points

- **An accelerated diagnostic pathway may be applied to adults presenting with new, symptomatic atrial fibrillation.**
- **Patients in atrial fibrillation less than 48 hours may be cardioverted and discharged from the ED with a low risk of complications.**
- **Patients with atrial fibrillation greater than 48 hours in duration may be rate controlled with metoprolol or diltiazem.**

- **This pathway applies to adults who present with new, symptomatic atrial fibrillation.** This includes patients with a recurrent abnormal rhythm. Ideally, these patients present with atrial fibrillation that is in isolation and is the cause of the symptoms. This is considered primary atrial fibrillation. Secondary atrial fibrillation occurs when the arrhythmia is the result of some underlying and predisposing medical condition such as alcohol withdrawal, decompensated COPD, thyrotoxicosis or sepsis.
- **Overall, we tend to see more secondary atrial fibrillation.** For these patients, the goal should be to treat and stabilize the underlying medical condition. If the atrial fibrillation persists, then you can consider some of the concepts in the diagnostic pathway.
- **What is the first decision point? Have they had symptoms for more than 48 hours or less than 48 hours?** Cardioversion is thought to be safe if the patient has been in the rhythm for less than 48 hours. If the patient has been in the rhythm for more than 48 hours, some additional diagnostics are indicated before cardioversion.
  - **This concept is controversial.** Previous literature suggested that thrombi were unlikely to form in the first 48 hours. This led to the hypothesis that cardioversion was safe in this window. This is wrong. Clots do form within 48 hours. However, all of the clinical data generated so far validates this practice. It indicates that while clots can form within 48 hours, patients treated within this window without obligatory pre-cardioversion anticoagulation appear to be stable for discharge with a low risk of complications.
- **What about the literature suggesting the risk of thromboembolic complications at 12 and 24 hours is higher than anticipated?**
  - *Nuotio, I et al. Time of cardioversion for acute atrial fibrillation and thromboembolic complications. JAMA. 2014 Au 13;312(6):647-9.* PMID: 25117135.
  - They did surveillance on a population of patients who was discharged from the ED after cardioversion for atrial fibrillation and followed them for 30 days. They found that the incidence of thrombotic events was higher than previously quoted and may start at 12 hours.
  - However, most of the ED literature has shown lower rates of thrombotic complications. Patients will have bad outcomes whether or not they are cardioverted. Atrial fibrillation is a risk factor for strokes. Some believe the patients in the study would have had complications whether or not they were cardioverted.
- *Weigner, MJ et al. Risk for clinical thromboembolism associated with conversion to sinus rhythm in patients with atrial fibrillation lasting less than 48 hours. Ann Intern Med. 1997 Apr 15;126(8):615-20.* PMID:9103128.
  - This study included 357 hospitalized patients converted to sinus rhythm within 48 hours after symptom onset. Some patients converted spontaneously and others were cardioverted. Only 3 patients had thromboembolic events after conversion to sinus rhythm. All three of these patients were in their 80s and had converted spontaneously.
- *von Besser, K et al. Is discharge to home after emergency department cardioversion safe for the treatment of recent-onset atrial fibrillation? Ann Emerg Med. 2011 Dec;58(6):517-20.* PMID: 22098994.
  - This was a best available evidence review that looked at five papers examining the safety of ED cardioversion. The authors' synopsis of the five reviewed papers was that most of the complications related to cardioversion come from procedural sedation. They found zero reported thromboembolic events post cardioversion after ED discharge with follow-up periods ranging from 7 to 30 days.
- **If the symptoms of atrial fibrillation have been going on for longer than 48 hours, control the rate. If the symptoms are under 48 hours, the patient goes straight to cardioversion.**
  - **Why not use pharmacologic attempts to cardiovert?** This is a reasonable approach. Procainamide has been identified as safe and effective by the Ottawa research group. Others have looked at similar strategies with flecainide or ibutilide. About 50% of patients receiving the drug will convert during their ED stay.
    - *Stiell, IG et al. Association of the Ottawa Aggressive Protocol with rapid discharge of emergency department patients with recent-onset atrial fibrillation or flutter. CJEM. 2010 May;12(3):181-91.* PMID: 20522282.
  - Procedural sedation and electrical cardioversion is likely safer, easier and quicker than pharmacologic treatment. It requires little monitoring. The results are much faster. It is more effective when compared head to head with drugs alone.
  - Whether you use electrical or pharmacologic cardioversion will depend on your practice environment and resource constraints.

- **What is the recommended starting energy level?** There is no solid evidence. Berg places the pads on the front and back and shocks them at 200J biphasic.
- **Can you offer a watch-and-wait option?** Some may offer the patient rate control with a plan to reassess in the morning. About 50% of patients will spontaneously convert back into sinus rhythm within 24 hours. Some rate control may help with certain patients but this is controversial. Several cardiology studies have found a weird association between acute rate control and persistent atrial fibrillation. However, this data may be confounded by spectrum bias (i.e. sicker patients received rate control).
- **We are experts in sedating patients. Electrical cardioversion, when delivered in a monitored ED environment is exceedingly safe.**
- **What happens if the cardioversion is unsuccessful?**
  - These patients are given urgent rate control when needed. They are risk-stratified according to risk of thromboembolic disease and anticoagulated as needed.
  - Most will use up to 3 or 4 successive shock attempts before terminating the effort. Berg's group found that approximately 90% of patients will be successfully cardioverted using the protocol. When it doesn't work, there is usually some secondary cause. For example, a patient with atrial fibrillation who didn't respond to cardioversion was later found to have a PE.
- **What work-up is indicated in these patients?** The one obligatory test is an electrocardiogram. Everything else should be determined based on their clinical risk factors and symptoms. Many will get an electrolyte panel on these patients. You can consider hemoglobin or chest x-ray. You do not need to routinely get a troponin. ACS is a very unlikely cause of acute atrial fibrillation.
- **Do patients need post-cardioversion anticoagulation to potentially decrease the rate of thromboembolic events?** There is no evidence that acute post-cardioversion anticoagulation for all-comers has an effect on thromboembolic risk. However, patients receiving anticoagulation and completing short term follow-up were associated with good outcomes.
  - **Sicker patients at baseline are likely to experience complications.**
  - **Berg recommends use of the CHA_2DS_2-VASc risk stratification score with a cut-off of 2.** Patients with a cutoff of 2 are recommended to start anticoagulation. ED physicians are encouraged to initiate anticoagulation at the time of the ED visit. They recommend considering anticoagulation for a score of 1.
- **CHA_2DS_2-VASc** – *See Chart*

Modifed from Lip, GY et al. Refining clinical risk stratification for predicting stroke and thromboembolism in atrial fibrillation using a novel risk factor-based approach: the euro heart survey on atrial fibrillation. Chest. 2010 Feb; 127(2):263-72. PMID: 19762550.

CHA_2DS_2-VASc

Criteria	Value	Points
Age	65-74 years old	+1
	> 75 years old	+2
Sex	Male	0
	Female	+1
Congestive Heart Failure History	Y/N	+1
Hypertension History	Y/N	+1
Stroke/TIA/Thromboembolism History	Y/N	+2
Vascular Disease History	Y/N	+1
Diabetes Mellitus History	Y/N	+1

Atrial Fibrillation ADP: Part 2 Rate Control and Anticoagulation

Rob Orman MD and Cameron Berg MD

Take Home Points

- **Patients may be rate controlled with metoprolol or diltiazem.**
- **Metoprolol is contraindicated in patients with asthma or COPD. Do not use if the patient is wheezing.**
- **Diltiazem is contraindicated in patients with a low ejection fraction.**

- **Berg's group has collected data on 422 patients in their cohort.** They have had zero short term thrombotic events.
- **Do patients with a heart rate greater than 160 get rate control prior to cardioversion?** No. They proceed directly to cardioversion. There is an association between acute rate control and failure to cardiovert and although this may not be valid, it doesn't make sense to complicate things.
- **Get diagnostics if it makes sense (often, it doesn't).** Consent, sedate and cardiovert the patient. If the cardioversion fails, you can do rate control. Most patients don't need rate control.
- **What do you do with patients who have duration greater than 48 hours or the duration is unclear?** If you don't know, err on the side of rate control. Most of these patients will not arrive with therapeutic anticoagulation. If they do, you can enter them into the pathway. Understand that cardioversion is unlikely to be effective if they are in the rhythm for a longer time.
  - You can confirm therapeutic anticoagulation with warfarin by checking the INR. You may be unable to do so if the patient is taking a newer agent. You need to find out exactly how compliant they have been. Are they taking the medication correctly? Rivaroxaban needs to be taken with food. Dabigatran needs to be taken twice daily. If they deviate from appropriate dosing, don't cardiovert.

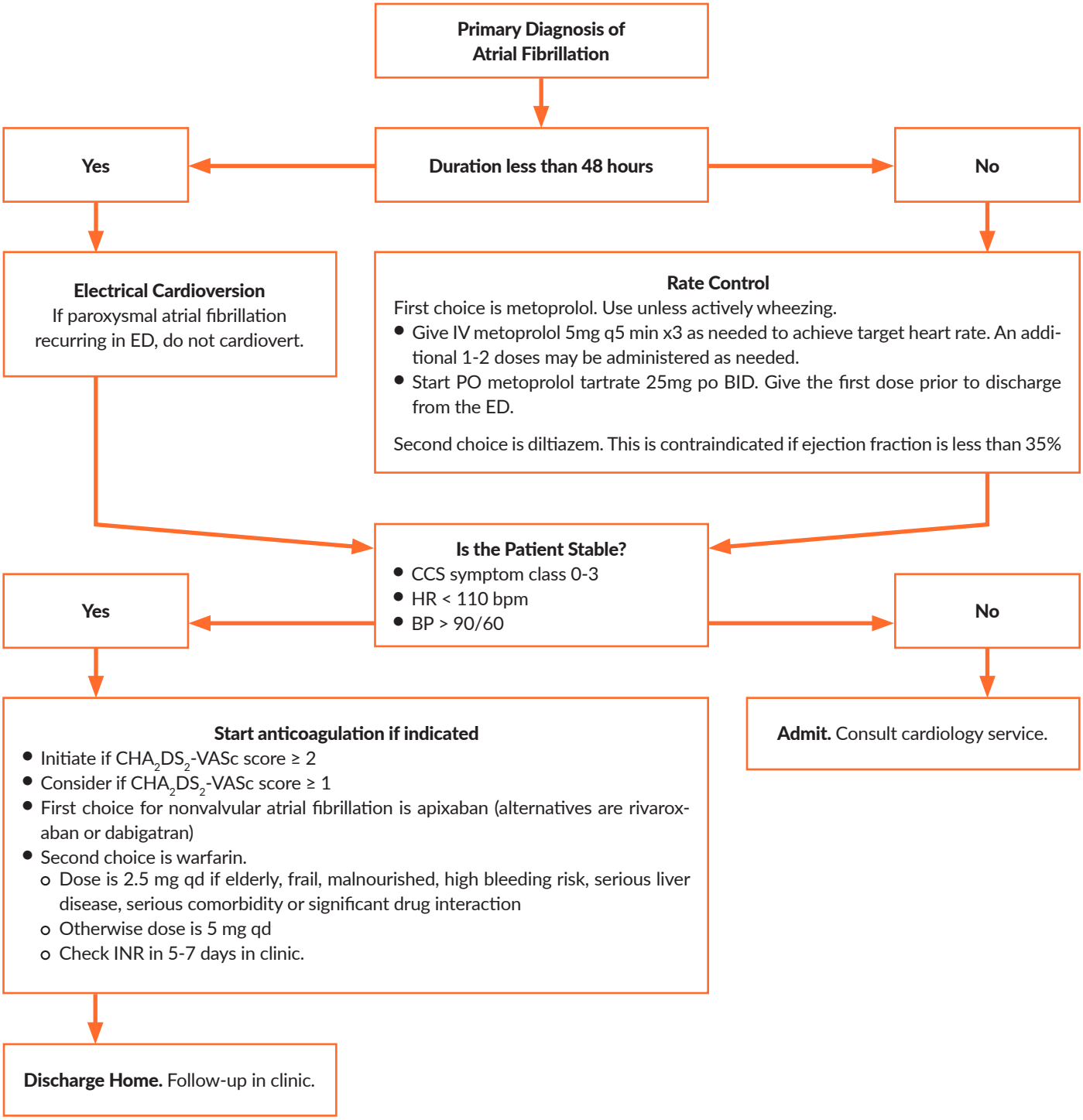
- **A rate of less than 110 at rest is targeted for rate control.** Berg recommends IV metoprolol. They give 5mg boluses with a maximum of 15mg and assess responsiveness. Diltiazem is their second-line agent with a weight-based dose of 0.35mg/kg. This is often higher than the reflexive 10-20mg.
  - **This drug selection is controversial.** There is some research available but no large randomized controlled trial. Berg views diltiazem as slightly more effective in terms of acute rate control. However, metoprolol is a better baseline drug for maintenance. It is easy to convert from IV to PO formulations; 5mg of IV metoprolol is equivalent to 25 mg of PO metoprolol. Metoprolol has less impact on ejection fraction which may be safer in undifferentiated ED patients.
  - Give 25 mg PO metoprolol prior to discharge from the ED.
  - Metoprolol is contraindicated if allergy and may impact asthma or COPD. Don't use it if the patient is actively wheezing.
  - Diltiazem is contraindicated if allergy or a low baseline ejection fraction. The literature indicates that number is probably 35%. If you do not have a baseline ejection fraction, you should be cautious if there is a history of pre-existing heart failure.
- **You should stick to one medication until maxed out rather than jumping to another therapy.** Beta-blockers with calcium channel blockers may be safe in a limited cohort of patients.
- ***See Flow Chart – Next Page***
- **Most patients with atrial fibrillation are admitted to the hospital.** However, most of these patients can probably be discharged home.
  - For the patient who remains in atrial fibrillation, the heart rate should be as near normal as possible (<110 bpm). The systolic blood pressure should be greater than 90.
  - The patient should be feeling well. How can you quantify this? The Canadian Cardiology Atrial Fibrillation scoring system.
- ***See "Canadian Cardiovascular Society SAF Scale" Below***

Modified from Dorian, P et al. A novel, simple scale for assessing the symptom severity of atrial fibrillation at the bedside: The CCS-SAF Scale. Can J Cardiol. 2006 Apr;22(5): 383-386. PMID: 16639472.

  - In the ADP, patients with a score of 0-3 are eligible for discharge.
- **If the patient is stable, do you initiate anticoagulation in the ED and what do you use?** They prescribe anticoagulation in the ED. They do not usually administer it prior to discharge as the thrombotic risks are long term. Patients with a CHA2DS2-VASc score of 2 should be given anticoagulation. You can determine bleeding risk using the HASBLED score.

The Canadian Cardiovascular Society SAF Scale	
Class 0	Asymptomatic with respect to atrial fibrillation
Class 1	Symptoms attributable to AF have ***minimal*** effect on patients general quality of life • Minimal and/or infrequent symptoms (palpitation, dyspnea, dizziness, presyncope or syncope, chest pain, weakness or fatigue • Single episode of AF without syncope or heart failure
Class 2	Symptoms attributable to AF have ***minor*** effect of patient's general quality of life • Mild awareness of symptoms in patients with persistent/permanent atrial fibrillation or • Rare episodes (less than a few per year) in patients with paroxysmal or intermittent AF
Class 3	Symptoms attributable to AF have a ***moderate*** effect on patient's general quality of life • Moderate awareness of symptoms on most days in patients with persistent/permanent AF or • More common episodes (> every few months) or more severe symptoms in patients with paroxysmal/intermitted AF
Class 4	Symptoms attributable to AF have a ***severe*** effect on patient's general quality of life • Very unpleasant symptoms in patients with persistent/paroxysmal AF and/or • Frequent and highly symptomatic episodes in patients with paroxysmal or intermittent AF and/or • Syncope thought to be due to AF and/or • Congestive heart failure secondary to AF

PRIMARY DIAGNOSIS OF ATRIAL FIBRILLATION



HAS-BLED Score		
	Clinical Characteristics	**Points**
H	Hypertension	+1
A	Abnormal renal and liver failure	+1 each
S	Stroke	+1
B	Bleeding	+1
L	Labile INRs	+1
E	Elderly	+1
D	Drugs or alcohol	+1 each

Modified from Pisters, R et al. A novel user-friendly score (HAS-BLED) to assess 1-year risk of major bleeding in patients with atrial fibrillation: the Euro Heart Survey. Chest. 2010 Nov;138(5):1093-100. PMID: 20299623.

- **There is a lot of thought that goes into the decision to start oral anti-coagulation.** Our job is to start the conversation. It is fine if the patient wants to follow-up with their primary care doctor or cardiologist, but most patients with a higher score will end up on anticoagulation and there is no reason to delay.
- **Berg prefers the newer oral anticoagulants.** They have fewer medication interactions. Although the existing data is skewed by industry bias, it does suggest a lower incidence of dangerous hemorrhagic events. None of the trials were powered or configured to make a definitive conclusion. We have apixaban, dabigatran, edoxaban and rivaroxaban available.
  - **The drugs that end in –aban are oral Xa inhibitors like enoxaparin in a pill.**
  - **Dabigatran is different and is a direct thrombin inhibitor.**
- **What do you do for patients that are unstable?** Truly unstable atrial fibrillation can be difficult to manage. Berg typically gives more medication and sometimes multiple agents for rate control. If the rhythm is dangerous and life-threatening, cardiovert them. Berg avoids diltiazem drips. Diltiazem is not short-acting and boluses are simpler and easier for nurses. It is easy to assess for response after boluses. Berg sometimes uses esmolol.

EM:RAP
Emergency Medicine:
Reviews and Perspectives

LIN Session: NEXUS Chest CT

Michelle Lin MD and Robert Rodriguez MD

Take Home Points

- **The use of CT scans in blunt trauma has increased dramatically but the incidence of injuries remains the same.**
- **A chest CT scan is 5-7 mSv and has a risk of cancer of 1/300 for young women.**
- **Patients do not need a CT scan in the absence of abnormal CXR, distracting injury, chest wall tenderness, sternum tenderness, thoracic spine tenderness, scapula tenderness or rapid deceleration mechanism.**

- *Rodriguez, RM et al. Derivation and validation of two decision instruments for selective chest CT in blunt trauma: a multicenter prospective observational study (NEXUS Chest CT). PLoS Med. 2015 Oct 6;12(10):e1001883. Open Access Link*
- **The use of CT scans to evaluate blunt trauma has increased dramatically over the last decade even though the incidence of injuries has remained the same.** A chest CT scan is about 5-7 mSv. This is about 300-400 chest x-rays. This radiation increases the risk of cancer. Trauma patients are usually young and more susceptible to adverse outcome from radiation. The risk of cancer for a young woman receiving a chest CT is about 1/300.
- **The yield of chest CTs are low, especially in the identification of injuries that would change management.** There is a lot of concern about missed injuries and malpractice.
- **Patients were enrolled from eight US, urban level 1 trauma centers.** They included patients with blunt trauma who received either chest x-ray or CT. They derived two decision rules; Chest CT-Major and Chest CT-All.
- **Why did they develop two different decision rules?** There is a wide spectrum of viewpoints and risk tolerance regarding the need to diagnose minor injuries in trauma. Everyone agrees that we need to identify aortic injuries, pneumothorax or diaphragmatic injury. Our surgical colleagues are often risk averse and want to diagnose all minor injuries.
- **Chest CT-All has a high sensitivity for both major and minor injuries.** It is 99% sensitive for major injuries and 95% sensitive for minor injuries.
  - **What are examples of minor injuries?** For example, 1-2 rib fractures or a minor pulmonary contusion that doesn't cause hypoxia or require ventilation, small pneumothoraces, etc.
- **Chest CT-Major has a sensitivity of 90% for minor injuries.**
- **Both decision instruments had 99% sensitivity for major injuries.**

The decision rules missed one patient who was an elderly man with a pneumothorax that required a chest tube and subarachnoid hemorrhage.

- **Chest CT-Major has a higher specificity of 37%.** It will allow you to forgo imaging in a greater number of patients than with Chest CT-All.

Chest CT-All	Chest CT-Major
1. Abnormal CXR 2. Distracting injury 3. Chest wall tenderness 4. Sternum tenderness 5. Thoracic spine tenderness 6. Scapula tenderness 7. Rapid deceleration mechanism	1. Abnormal CXR 2. Distracting injury 3. Chest wall tenderness 4. Sternum tenderness 5. Thoracic spine tenderness 6. Scapula tenderness
May forego CT if all criteria absent. One or more criteria present cannot exclude thoracic injury but presence of criteria does not indicate need for CT.	

  - Abnormal chest x-ray includes any thoracic injury, including clavicle fracture or widened mediastinum.
  - Distracting injuries are any condition thought by the clinician to produce significant pain to distract the patient from a second injury. Examples include but are not limited to long bone fractures, visceral injuries requiring surgical consult, large lacerations or degloving injuries, crush injuries, large burns , spine fractures, spinal cord injuries or any other injury producing acute functional impairment.
  - Rapid deceleration mechanism is defined as a motorized vehicle accident greater than 40 mph or a fall greater than 20 feet.
- **These are meant to be used in conjunction with the NEXUS C-spine criteria.**
- **The negative likelihood ratio for any injury is 0.18.** The likelihood ratio for major injury is 0.04.
  - **What does this mean?** If you have low suspicion or pretest probability for major thoracic injury and the CT-Major instrument is negative, it is highly unlikely that the patient has a major injury.
  - If your pretest probability was moderate and the CT major instrument is negative, it is unlikely the patient has an injury.
  - If you have a high pretest probability for major injury, you should still obtain a CT chest even if the decision rule is negative.
  - For a more in depth discussion of likelihood ratios, you can revisit the EM:RAP segment by David Newman in March 2012.
- **What if the patient has a motor vehicle crash and is intubated for head injury?** These decision instruments are intended for use in the hemodynamically stable, non-intubated patient. This is the vast majority of patients.
- **The presence of one or more criteria does not mandate a CT of the chest.**

NEXUS C-SPINE CRITERIA

MAJOR INJURIES	MINOR INJURIES
• Aortic or great vessel injury • Ruptured diaphragm • Pneumothorax: received evacuation procedure • Hemothorax: received drainage procedure • Sternal fracture: received surgical intervention • Multiple rib fracture: received surgery or epidural block • Pulmonary contusion: received ventilation for respiratory failure within 24 hours • Thoracic spine fracture: received surgical intervention • Scapular fracture: received surgical intervention • Mediastinal or pericardial hematoma: received drainage • Esophageal injury: received surgical intervention • Tracheal or bronchial injury: received surgical intervention	• Pneumothorax: no evacuation but observed >24h • Hemothorax: no drainage but observed >24h • Sternal fracture: no surgical intervention • Multiple rib fracture: no surgery or nerve block • Thoracic spine fracture: no surgical intervention • Scapular fracture: no surgical intervention • Pulmonary contusion/laceration: no ventilation but observed >24h • Mediastinal or pericardial hematoma: no surgery • Mediastinal or pericardial hematoma: no surgery • Esophageal injury: no surgical intervention • Tracheal or bronchial injury: no surgical intervention

Paper Chase 1: Is Droperidol Safe?

Sanjay Arora MD and Michael Menchine MD

Take Home Points

- **IV and IM droperidol are safe and effective for the management of acute agitation with minimal risk of prolonged QT.**
- **In this study, the median time to sedation was 20 minutes and 70% of patients were effectively sedated after the first dose.**
- **No patients had torsades, dysrhythmia or cardiac arrest.**

- *Calver, L et al. The safety and effectiveness of droperidol for sedation of acute behavioral disturbance in the emergency department. Ann Emerg Med. 2015 Sep;66(3):230-238.* PMID: 25890395
- **IV and IM droperidol are safe and effective for the management of acute agitation.** The risk of prolonged QT was minimal.
- **Acutely agitated patients are common in the ED.** An ideal drug for management would be rapid onset and offset with a good safety profile. Benzodiazepine and neuroleptics either alone or in combination are the mainstay but there are some problems with these such as dystonic reaction, hypotension and respiratory depression.
- **Droperidol is a first generation anti-psychotic of the butyrophenone class.** It was used for decades to manage agitation and nausea. In 2001, the FDA issued a black box warning stating that it caused QT prolongation and torsades de pointes. They recommended using alternatives when available and advised cardiac monitoring. This was very controversial. Critics have asserted that droperidol is safe and the incidence of QT prolongation and torsades is probably no more than any other anti-psychotic. The black box warning caused the use of droperidol to drop dramatically.
- **The authors of this study looked for QT prolongation with the administration of high dose parenteral droperidol.** The secondary objective was to look at the risk of other adverse events and success of sedation.
- **The study was performed at 6 large hospitals in Australia.** By protocol, they gave 10mg of IV or IM droperidol as a bolus. The dose was repeated in 15 minutes if the patient was still agitated. After 20mg, the management was deferred to the treating physician. Patients were monitored closely with ECG obtained as soon as feasible. There was no control group.
- There were 1009 patients with an ECG performed within 2 hours of droperidol administration. 13 patients had QT prolongation. Most of the time it was mild. 7 of these 13 patients had obvious other reasons for QT prolongation including some who had documented QT prolongation from medications. 0.6% of the analyzed sample had QT prolongation that could not be attributed to another cause. This is not very much. No patients had torsades, dysrhythmia or cardiac arrest. The QT prolongation in the 6 patients was minimal.
- **The median time to sedation was 20 minutes and 70% of patients were effectively sedated after the first dose.**
- **The most common adverse event was oversedation which happened in 7%.** This was more common in patients who received droperidol with a benzodiazepine. There were 34 staff injuries; some kicked, some punched and a needle stick.
- **The study was limited as it was not a randomized controlled trial.** The adverse events can't be directly compared across other agents such as haloperidol, benzodiazepines or ketamine. However, the risk of prolonged QT in this large cohort was very small. The QT prolongation when present was mild and there were no dysrhythmias.

This is fair evidence that droperidol has a good safety profile and is highly effective at sedating agitated patients, even as a single agent.

Paper Chase 2: Ketamine for Pain

Sanjay Arora MD and Michael Menchine MD

Take Home Points

- **IV ketamine is just as effective as IV morphine in treating acute moderate to severe pain.**
- **Ketamine is associated with increased minor adverse events such as dizziness and disorientation.**

- *Motov, S et al. Intravenous subdissociative-dose ketamine versus morphine for analgesia in the emergency department: a randomized controlled trial. Ann Emerg Med. 2015 Sep;66(3):222-229.* PMID: 25817884
- **IV ketamine is just as effective as IV morphine in treating acute moderate to severe pain but with increased minor adverse events such as dizziness and disorientation.**
- **Relieving pain is an important part of our jobs.** Although we often use opiates for moderate to severe pain, some have suggested ketamine as an effective opiate sparing agent. In low or subdissociative doses, ketamine works as an NMDA receptor antagonist to decrease the wind-up phenomenon that occurs when more pain receptors are recruited and decrease pain memory. It has been used in a variety of settings including rural and prehospital settings. It has also been used in a variety of

patient populations such as cancer, post-operative and patients with sickle cell disease. Most of the available data is retrospective. The few prospective randomized controlled trials compare ketamine and morphine to morphine alone.

- **In this paper, ketamine alone was compared to morphine alone with a primary outcome of comparative reduction in pain at thirty minutes.** This was a prospective, double-blinded, randomized controlled trial. They looked at the safety and efficacy of the medications. They enrolled a convenience sample over a one year period. Patients were aged 18 to 55 with acute abdominal, flank or musculoskeletal pain rated at least 5 out of 10 on the visual analog scale. Patients who were hemodynamically unstable or had contraindication to ketamine or morphine were excluded.
- **Patients were randomized to 0.3 mg/kg of ketamine or 0.1 mg/kg of morphine.** The pain scores, vital signs and adverse effects were recorded at 15, 30, 60, 90 and 120 minutes. They looked at the need for rescue medications at 30 and 60 minutes.
- **90 patients were randomized with 45 in each group.** The groups were similar at baseline.
- **Both medications worked equally well in reduction in pain scores at 30 minutes (8.5 to 4 in both groups).** At 15 minutes, more patients in the ketamine group reported complete resolution of the pain (49% compared to 13%). However, this evened out by 30 minutes.
    - **Patients in the ketamine group did need more rescue fentanyl at two hours.**
- **There were no serious or life-threatening side effects in either group.** Ketamine had more associated minor adverse effects such as dizziness or disorientation.
- **This is a small but well-conducted controlled trial reminding us that ketamine is available for pain control in the ED.** It is not just for musculoskeletal pain. It is nice in trauma and burn patients although they were excluded in this study. Does your hospital consider this to be procedural sedation? It depends on the hospital. Check before you use it. Don't use more than 0.5mg/kg IV.
- **For more on dosing of ketamine, check out Ruben Strayer's post on this topic:** *http://emupdates.com/2013/12/25/the-ketamine-brain-continuum/*

Paper Chase 3: No Need To Irrigate Abscesses

Sanjay Arora MD and Michael Menchine MD

Take Home Point

- **Irrigation of cutaneous abscesses does not improve treatment success.**

- *Chinnock, B et al. Irrigation of cutaneous abscesses does not improve treatment success. Ann Emerg Med. 2015 Sep 10. Link*
- **Irrigation of abscesses after I and D did not improve outcomes.**
- Irrigation is described in most emergency medicine and surgical textbooks as part of the standard approach to incision and drainage. However, it is not validated in studies.
- **This was a non-blinded, randomized controlled trial of irrigation versus no irrigation for the treatment of abscesses.** The group without irrigation had a standard incision and drainage while the other group received irrigation. However, the physicians were free to irrigate however they wanted. Physicians in both groups were allowed to give antibiotics and determine antibiotic selection.
- The primary outcome was need for further intervention such as repeat incision and drainage or change of antibiotics within 30 days.
- They had relatively broad inclusion criteria. Patient with comorbidities such as diabetes were included. 209 patients were enrolled and randomized over 4 years. 187 patients had complete follow-up data.
- **There was no difference in need for repeat procedures across the treatment groups.** 15% of patients in the irrigation had repeat incision and drainage or antibiotic change compared to 13% in the control group. Procedural pain was the same in the treatment groups.
- **The study was limited by a lack of blinding.** The assessors were blinded. The physicians had a lot of discretion in management of these patients. Irrigation may have been minimal.
- **This was a small study from a single site with some significant limitations.** However, the key finding that 1 in 8 cutaneous abscesses will require further treatment is probably correct. Irrigation does not appear to change this.

Paper Chase 4: Who Should Get a Foley

Sanjay Arora MD and Michael Menchine MD

Take Home Points

- **Implementation of a protocol to decrease urinary catheter placement in the ED improved practitioner knowledge and slightly decreased the rate of catheter insertion.**
- **Don't place catheters in patients for staff convenience.**

- *Mulcare, MR et al. A novel clinical protocol for placement and management of indwelling urinary catheters in older adults in the emergency department. Acad Emerg Med. 2015 Sep 22(9):1056-66. PMID: 26336037.*
- **Implementation of a protocol to decrease urinary catheter placement in the ED improved practitioner knowledge and slightly decreased the rate of catheter insertion.**
- Decreasing catheter use in the emergency department is a positive thing for patients. Foleys are often placed reflexively in elderly patients for convenience. This is not a benign event. It causes pain, discomfort and places the patient at risk for catheter associated infections. Research suggests that this comprises 1 in 10 health-care associated infections. This is bad for patients and Medicare reimbursement.
- **The authors went to a lot of effort to develop and implement a clinical protocol designed to reduce inappropriate catheter placement and encourage early reassessment and removal when possible.** They did a very thorough literature review. They developed focus groups with physicians and nurses and brought together local experts. They developed a protocol utilizing green, yellow and red schema for the placement of catheters for a variety of diagnosis (ventilated and spinal cord injury are green, mild CHF exacerbation is yellow, convenience for care is red). The protocol was distributed to the doctors and nurses with a presentation, pocket card and posters.
- **Providers were given surveys immediately before and after implementation of the protocol as well as 6 months after assessing knowledge.** They had an 86% response rate.
- **They showed that knowledge about who should and shouldn't get a catheter did increase.** However, most providers still were willing to place a catheter despite the protocol. They looked at the rate of catheter placement in elderly ED patients in the 6 month pre- and post- period. They saw a drop from 19% to 15%. We don't know if the drop was appropriate or inappropriate. We don't know if it changed practice for physicians, nurses or mid-level providers.
- **It is a positive thing that you can change your groups practice.** The best method is unclear. We should think twice about placing foley catheter and consider removing them when no longer needed.

Paper Chase 5: Workload and Work Quality

Sanjay Arora MD and Michael Menchine MD

Take Home Points

- **ED providers are interrupted on average, 5.5 times per hour and spend over 30% of their time multitasking.**
- **There was a significant negative association between the ED providers' self-assessed mental workload and patients' perceived quality of care.**
- **Interruptions and multitasking were positively associated with patient satisfaction and the quality of handoffs.**

- *Weigl, M et al. Work conditions, mental workload and patient care quality: a multisource study in the emergency department. BMJ Qual Saf. 2015 Sep 8. Open Access Link*
- **This study looked at the prevalence of work flow interruptions and multitasking and impact on patient perceived quality of care and handoffs in the hospital.** We have a lot of interruptions and frequently multitask. When workload was higher, perceived quality of care was lower. Interruptions had a positive association with quality of handoffs.
- **Workflow interruptions, multitasking, high work demands are as central to emergency medicine as the ABCs.** Some studies have estimated the number of interruptions to be 1 to 30 times per hour. There are some studies that show when an ED provider is interrupted, they fail to return to original activity about 20% of the time. Multitasking represents a lot of our work activity. These issues have been demonstrated to cause mental stress for emergency care providers. The ED is a mentally tasking place to work. Can we handle it? Does it affect the quality of care and patient safety?
- **This paper reports on a complex study of a single, community ED in Germany.** They had two questions. What is the prevalence of interruption and multitasking? Are these events associated with lower patient perceived quality of care or care transitions (handoff)?
- **They had direct observation of ED MDs and nurses.** They observed and clicked every time they were interrupted. They then asked the ED providers to complete an index of mental workload. This involved 5 items to gauge how stressed the provider felt. They asked the patients seen by the provider to complete a patient satisfaction questionnaire immediately at the end of

their ED care. If the patient was admitted, they asked the receiving doctor to fill out a survey regarding the quality of the handoff.

- **Overall, they had twenty observation periods during which 565 patients received care.**
- **Providers were interrupted on average, 5.5 times per hour.** They spent over 30% of their time multitasking.
- **There was a significant negative association between the ED providers' self-assessed mental workload and patients' perceived quality of care.** However, the interruptions and multitasking were positively associated with patient satisfaction and the quality of handoffs to other providers. This is what we do.
- **We are good at multitasking and have good coping skills but when we get stressed, our patients feel it.** If you feel stressed, try to give yourself a break and decompress.

Annals *of* Emergency Medicine
An International Journal

Annals of Emergency Medicine: Why The Limp?

Paul Jhun MD and Ryan Raam MD

Take Home Points

- **Limp in children can be evaluated with the mnemonic LIMPSS; Legg-Calve-Perthes disease, Infection/inflammation, Malignancies, Pain from trauma, Slipped capital femoral epiphysis or a Source somewhere else.**
- **Obtain an ultrasound of the hip when ruling out septic arthritis; a joint effusion of 2mm is concerning.**
- **Some studies show ESR> 40 and CRP> 2 to be sensitive for osteomyelitis.**

CASE

An 11-month-old female presented to the pediatric emergency department with a one day history of refusal to bear weight on the right lower extremity after a two week progression of pain and limping. There was no history of trauma or fever. Her vital signs were within normal limits. The right ankle was noted to be edematous, warm to the touch, diffusely tender to palpation and she had diminished creases when compared to the left. Her exam was otherwise unremarkable. Serum laboratory studies were all within normal limits with the exception of an elevated white blood cell count of 16,400 cells/mm3. Plain films of the ankle revealed a lucency in the posterior aspect of the talus and soft tissue swelling of the ankle joint. MRI was subsequently completed and revealed a 7mm abscess in the lateral subcutaneous soft tissues. Diagnosis? Talar osteomyelitis.

- **There is an easy mnemonic for that: LIMPSS.**
- **L is for Legg-Calve-Perthes Disease.**
  - This is a progressive idiopathic avascular necrosis of the femoral head.
  - It is typically seen in kids between 3 and 12. Males more than females (4:1). Caucasians more than African Americans.
  - The classic presentation is unilateral pain with difficulty internally rotating and abducting the hip. However, up to 1 in 6 kids will have bilateral presentation.
  - The diagnosis is made on x-ray of the hips. **Remember to get the "frog leg lateral" to get the best views.** X-rays may be unremarkable early in disease. Findings may range from crescent sign (subcortical lucency) early on to complete bony destruction. The patient may need an MRI which is considered the gold standard in imaging.
  - **Why do we care?** A significant portion will develop arthritis later in life.
  - Once diagnosed in the ED, these kids typically can go home with instructions for limited activity, non-steroidal anti-inflammatory drugs for pain and orthopedic surgery follow-up within the week.
- **I is for infection or inflammation.**
  - **The lower extremity is the most common site for pediatric cellulitis.** Keep it on your differential. Undress the kid and get a good exam.
  - **Osteomyelitis is a common presentation in a limping child.** This affects boys more than girls. The most common bones are the femur, tibia and fibula (these 3 sites make up more than 50% of osteomyelitis in kids). Staph aureus is the most common organism.
    - **Some studies have shown serum erythrocyte sedimentation rate and C-reactive protein to be sensitive in evaluating for osteomyelitis.**
    - **Don't be tricked by normal x-rays.** The classic "rat bite" finding of bony cortex destruction doesn't appear until 2-3 weeks.
  - **How can you differentiate between transient synovitis and septic arthritis of the hip?** Patients with transient synovitis can go home with NSAIDs and expectant management and patients with septic arthritis need surgical washout in the operating room. Both can present with fever, pain with passive movement of the hip and refusal to bear weight.
    - **The best decision rule is Kocher's criteria.** This decision rule has 4 criteria; refusal to bear weight, fever >38.6, WBC count > 12,000 cells/mm3 and ESR >40mm/h. The study

found that the more criteria present, the greater likelihood of septic arthritis. If the patient had 0 out of 4 criteria, the likelihood of septic arthritis was 0.2%. In patients with four predictors was 99.6%.

- *Kocher, MS et al. Differentiating between septic arthritis and transient synovitis of the hip in children: an evidence-based clinical prediction algorithm. J Bone Joint Surg Am. 1999 Dec;81(12):1662-70. PMID: 10608376.*

- **Another study found that CRP>2.0 mg/dL and refusal to bear weight might be the best clinical predictor.**

- *Caird, MS et al. Factors distinguishing septic arthritis from transient synovitis of the hip in children: A prospective study. J Bone Joint Surg Am. 2006 Jun;88(6):1251-7. PMID: 16757758.*

- **Don't forget to get an ultrasound of the hip.** A hip effusion greater than 2mm in diameter is septic arthritis until proven otherwise. Get your orthopedic surgeon on board.

- **M is for malignancies.**
  - **There are a lot of bone tumors.** You don't need to know all of them but you should know about Ewing's sarcoma and osteosarcoma. These make up 90% of all bone cancers in kids.
  - **Classically, kids complain of constitutional symptoms like fever and weight loss with a subacute, insidious leg pain that might be worse at night.** Local trauma to the area might be the inciting event that draws attention to the lesion.
  - **They are usually found in the long bones of the lower extremity such as the proximal tibia and distal femur.** Ewing's sarcoma likes to hide in the pelvis.
  - **On x-ray you may see characteristic "onion-skinning" and "sunburst" appearance of the pelvis.** This is a reactive periosteal change that occurs because of the rapidly growing tumor at the margins of the bone.
  - **Patients may have fever, swelling, redness, an elevated WBC and ESR with periosteal changes on x-ray similar to osteomyelitis.** Beware.
  - **Acute lymphoblastic leukemia is the most common pediatric cancer and children may present with long bone pain and limping.** If you have a kid with constitutional symptoms, some abnormalities on the CBC and insidious pain in the legs, keep ALL on your differential.
- **P is for pain from trauma.**
  - **The most obvious cause of pain we encounter is from fractures.** These are the most commonly missed pediatric orthopedic emergency diagnoses.
  - **Toddler's fracture is a nondisplaced oblique fracture of the distal tibia.** It is usually found in toddlers between 1 and 4 years old and caused by normal toddler activities like tripping, falling from low heights, etc.
  - **Toddler's fractures are difficult to identify on x-rays.** The AP view is your best bet and you may just see a subtle oblique lucency through the distal tibia that ends medially. There may not be a break in the cortex.
  - **Always consider child abuse or non-accidental trauma.** Up to 20% of fractures are the result of non-accidental trauma and 80% occur in children less than 18 months old. Be aware of the developmental stage of the patient, the clinical history, the mechanism and the type of fracture. Don't mistake a spiral fracture of the midshaft of the tibia for a Toddler's fracture (which occurs in the distal half to third of the tibia). When in doubt, get child protective services involved.
- **S is for SCFE (slipped capital femoral epiphysis).**
  - **In this disease, the epiphysis is falling off the metaphysis at the physis ("the ice cream is falling off the cone").** SCFE tends to affect overweight tweens, boys more than girls and African Americans and Hispanics more than Caucasians.
  - **In 25% of cases, patients will complain of knee or thigh pain and not hip pain.**
  - **This is second most missed pediatric orthopedic emergency diagnosis.**
  - SCFE can present bilaterally 10% of the time. X-rays are used to diagnose this condition. Get the frog-leg view. Klein's line on the AP view misses this diagnosis 60% of the time.
  - **SCFE leads to avascular necrosis of the hip.** The child should be placed in non-weight bearing status with immediate orthopedic consultation for admission and inpatient surgical management.
- **S stands for somewhere else.**
  - **If you do a complete work-up of the lower extremity and don't find anything, consider sources above the legs** such as psoas abscess, appendicitis, discitis, urinary tract infection, ectopic pregnancy, torsion and pelvic inflammatory disease. Most the diseases may cause referred pain through irritation of the iliopsoas or obturator internus muscles or obturator nerve.
- **Back to the case.** The patient with a talar osteomyelitis was managed operatively with incision and drainage of the abscess as well as irrigation and debridement of the talus. She received antibiotics and recovered well.

Do We Still Need the Cervical Collar?

Rob Orman MD and Chris Colwell MD

Take Home Points

- **There is no evidence that C-collars restrict harmful movement.**
- **C-collar use may result in harm to patients and pain.**

- **Most literature on the cervical collar discusses the non-utility of the cervical collar. How did every trauma patient end up in a C-collar?** There has never been any evidence that suggests that the C-collar benefits our patients in any way.
- **We use C-collars because trauma patients may have an unstable C-spine injury.** If we move the injury, the patient could be paralyzed. We make every effort to not extend the neck, including during intubation. However, it is not motion that causes harm but energy. This terror of causing any mobility is unfounded and goes against reason.
- **What is the harm of placing patients in a collar?** Taking patients out of a position of comfort and placing them into a rigid cervical collar that extends their neck does not make them safer. There is evidence that C-collars reduce venous return and increase intracranial pressure.
    - *An article by Gaither on failed airways found that C-spine immobilization was a primary reason that we struggle with airways in the field. Gaither, JB et al. Prevalence of difficult airway predictors in cases of failed prehospital endotracheal intubation. J Emerg Med. 2014 Sep;47(3):294-300. PMID: 24906900.*
- **This something that was never based on evidence, causes harm and pain and we have allowed it to become our standard and culture.**
- A study by Hauswald on emergency immobilization on neurologic outcome of patients with spinal injuries comparing the US to Malaysia where spinal immobilization is not performed found patients that were immobilized did worse with similar injuries.
    - *Hauswald, M et al. Out-of-hospital spinal immobilization: its effect on neurologic injury. Acad Emerg Med. 1998 Mar;5(3):214-9. PMID: 9523928.*
- **Culture is difficult to change.**
- Forcing immobilization on a combative and resistant patient could increase the energy and potential for damage. Sedate the patient so they don't move around so much.
- **What about penetrating trauma?** The literature shows the mortality doubles with immobilization. Immobilization is not indicated in penetrating trauma.
- **Does the C-collar restrict movement?** We have no evidence that C-collars restrict movements that could be harmful.

CASE

A drunk patient found down gets placed in a cervical collar until sober. There are repeated battles; the patient sits up with the collar askew. Once they take off the collar, it rarely goes back on.

- **Sedate these patients so they are not thrashing about.** We don't have to intubate and paralyze all these patients.

CASE

A drunk patient arrives in a collar. You don't know what happened to them. Do you leave the collar on?

- Practice varies. Colwell will remove the collar when they are sedated or cooperative. Sometimes the agitation is due to the collar. Sedation to keep the collar on can lead to respiratory compromise. Some of these patients will just fall asleep when the collar comes off.

CASE

An 80 year old with fall and large hematoma with 8cm occipital scalp lac. They deny neck pain. You know they have a C1 fracture.

- **These patients are very risk for high cervical spine injuries.** These are the most concerning injuries and the reason we immobilize. However, we may be causing more harm.
- **Rigid cervical collars can stretch the spinal cord in unstable high cervical fractures and reduce the blood flow to the spinal cord.**

CASE

A patient in a motor vehicle accident walks into triage with neck pain and tingling in the arms.

- **The patient has proved to you that movement will not paralyze them.** Putting them in a less comfortable position won't benefit them.

- **We can't do this in isolation.** We need to have this conversation with the entire team including orthopedics, trauma and neurosurgery.
- **For more, check out** *http://www.scancrit.com/2013/10/10/cervical-collar/*

Doc In The Bay: Stop Bleeding Without a Hospital

Howie Mel MD and Roy Alson MD

Take Home Points

- **Properly applied tourniquets are life-saving in exsanguinating hemorrhage and have a low rate of complications.**
- **Direct pressure should be applied with one finger for a long time.**
- **Israeli bandages with clotting agents or sanitary napkins may be used to control bleeding.**

- **A true arterial bleed is an emergency lasting a few minutes unless you can get control.** If the artery is completely transected, it may retract and the bleeding won't be apparent until the patient receives crystalloid fluids. Much of the arterial bleeding in extremity wounds is due to small surface arterioles and arteries. These may have pumping blood but rarely exsanguinate. We rarely deal with exsanguinating bleeding in the civilian sector aside from the tactical environments.
- **There are a lot of misconceptions regarding tourniquets.** There is a perception that using a tourniquet will result in the loss of the limb. This dates to the Civil War when patients experienced cannonball and artillery injuries. Patients would be placed in a tourniquet and receive amputation. The tourniquet was a marker of an injury requiring amputation rather than a cause. In more recent conflicts, prolonged evacuation times led to limb ischemia.
- **Our orthopedic colleagues perform surgeries with a tourniquet in place for several hours with no damage.** Abdominal aortic aneurysm repair also does not result in limb loss.
- **In reality, the literature shows that properly applied tourniquets in exsanguinating hemorrhage are life-saving.** The tourniquet registry showed three cases of permanent damage. All had the tourniquet in place for more than eight hours and the complications were numb fingertips.
- **We need to stop this dogma that the patient will lose their limb if you place a tourniquet. They will not. Use the tourniquet.** If you have a brisk arterial bleed that you can't control with direct pressure, place a tourniquet and note the exact time of placement.
- **How long can the tourniquet remain in place?** 1-2 hours seems like a relatively safe recommendation. Hopefully, you will be able to get the patient to definitive care before this.
- **Are there other options?** The Israeli bandage. This is an elastic bandage with a gauze pouch impregnated with a clotting agent. It can be wrapped around like a tourniquet with direct pressure. It can also be balled up and placed into a body cavity with injury.
    - **The clotting bandages do not work alone and must be accompanied with direct pressure.** We are bad at continuing direct pressure to control bleeding. It may take several minutes of direct pressure. If the patient is anticoagulated, it may take much longer.
    - **Apply pressure with one finger to the site of bleeding.** Hold firm pressure. Don't let go for a while. Don't peek.
- **Sanitary napkins are another option.** They are designed to soak up a large amount of blood.
- **Start with direct pressure unless it is a life-threatening bleed.**

Airway Corner: Out of Hospital Cardiac Arrest

Darren Braude MD and Brent Myers MD

Take Home Points

- **If the patient with cardiac arrest has been in asystole for 20 minutes, the chance of neurologically intact survival is less than 1%.**
- **Approximately 5% of patients with PEA arrest with increasing end-tidal CO2 or end-tidal CO2>20 will have ROSC and good neurologic outcome after prolonged resuscitation for 40-60 minutes.**
- **Patients with refractory ventricular fibrillation may benefit from alternate pad placement and double sequential defibrillation.**

CASE

EMS arrives on scene to a witnessed cardiac arrest with rescuer CPR in progress. The patient is 60 years old. EMS places an airway. CPR is continued. The patient has received two doses of epinephrine and the resuscitation has been in progress for 25 minutes. The patient is 15 minutes away from the hospital. EMS calls to request termination of the resuscitation.

- **What is the role of the rhythm, end-tidal CO_2, age and length of resuscitation?** How do we make the best evidence based decision?
- **Scenario 1. The patient is in asystole.**
    - **If the patient is in asystole for the duration of the resuscitation, can you stop?**
    - If the patient has been in asystole for twenty minutes, the chances of neurologically intact survival are less than 1%. This is medically futile.

- An initial rhythm of asystole without an obvious sign of death and unwitnessed arrest deserves some effort at resuscitation but they do not need prolonged resuscitation.
- **What if the end-tidal CO_2 is high?** It doesn't matter. If the end-tidal CO_2 is high with persistent asystole, you must be perfusing with compressions but you are not getting return of rhythm. With good chest compressions, minimal interruptions and controlled ventilations, we can create end-tidal CO_2 values that reflect high quality CPR but are unrelated to survivability.
- **The trend of the end-tidal CO_2 is far more important than any single value.**

- **Scenario 2. The patient is in PEA. There is no ultrasound on scene.**
  - **PEA is a survivable rhythm.**
  - **Rate is important.** A rate above 40-60 beats per minute is workable.
  - **The trend in the end-tidal CO_2 is important.** A trend upward above 20 or a current value above 20 should have consideration of prolonged resuscitation.
  - **Prolonged resuscitations of 40-60 minutes have decreased survival but patients who survive have a good chance of neurologically intact survival.**
  - If the patient has point-of-care ultrasound (POCUS) that shows no cardiac activity, the resuscitation may be terminated earlier. Most EMS will not have this capability.
  - History obtained on scene can be used to make the decision to stop resuscitation.
  - **Age decreases your odds of survival by 0.03 per year of life but is not a sole reason to end resuscitation.**
  - **If the initial end-tidal CO_2 was 30 but dropped to 5 or was at 5 throughout the resuscitation despite good CPR, do you continue the resuscitation for 40-60 minutes?** Most would not continue a resuscitation of a PEA arrest with an end-tidal CO_2 of 5 after 20-25 minutes of resuscitation.
  - **What percentage of PEA patients with prolonged resuscitation survive?** Most neurologically intact survivors with PEA arrest are attained by 40 minutes of resuscitation. The rate of neurologically intact survival with resuscitation between 40-45 minutes is 8%, 6% between 45-50 minutes and 5% at 50 minutes. At an hour of resuscitation, the rate of neurologically intact survival is 2% and this is consistent with medical futility. The rate of neurologically intact survival is 20% at 25 minutes of resuscitation.
  - **Does prolonged resuscitation increase the likelihood of persistent vegetative state?** The chance of survival decreases but the proportion with persistent vegetative state remains consistent until 40-60 minutes of resuscitation.

- **Scenario 3. The patient is in ventricular fibrillation.**
  - **These codes need to be managed aggressively.** We need to minimize interruption in compressions and deliver a shock as soon as possible.
  - **Pad placement.** These are placed on the anterior chest and apex to allow chest compressions to continue while the pads are placed. The original pads are left in place for 3-4 defibrillations. If the patient remains in fibrillation after the ACLS algorithm is completed, Myers then places a second set of pads in the opposite pad placement configuration which is usually anteriorly and posteriorly. They have found anecdotally that changing the pad placement makes a difference and the patient is more likely to convert.
  - **If the patient remains in ventricular fibrillation, Myers will charge both defibrillators and perform a double sequential external defibrillation with the maximum Joules.** This is only performed in the setting of ventricular fibrillation that is refractory to the ACLS algorithm. Attempt to deliver the shocks simultaneously although a small delay might result between shocks. Make sure the team is on the same page; "3, 2, 1, shock".
  - **Is this safe for the patient?** There is some data but this is primarily in refractory perfusing rhythms such as atrial fibrillation. Some feel that leaving the patient in ventricular fibrillation does more myocardial damage than the theoretical damage due to double sequential defibrillation.
  - **What are the chances of triggering an R on T phenomenon with double sequential defibrillation?** At this point, the patient is pretty much dead. You are unlikely to make things worse.
  - **Will this damage the machines or void the warranty?** The defibrillators are designed to prevent electricity from going back up into the machine. Myers uses the Lifepak defibrillator. It is unclear if this is true for all devices.

- **Resuscitation is a prehospital science.** We need to make sure that all of our EMS responders have the best knowledge available to conduct resuscitations where they find them.

EM:RAP
Emergency Medicine: Reviews and Perspectives

February Summary

Rob Orman MD and Anand Swaminathan MD

The EMRAP Mail Bag.

- **From Ash Mukherjee. Can we change the terminology of 'hemodynamically stable' when talking about submassive PE?** Someone with a heart rate of 110 to 130 would never be called hemodynamically stable in any other circumstance. It should be called massive PE without hypotension.
  - **Even this statement is suboptimal.** A person with a normal BP of 150 should be considered hypotensive with a blood pressure of 95. Too many clinicians take the conservative route because the systolic is above 90. You have to get an echocardiogram to grasp the actual situation.
  - **It is up to emergency physicians to make this call.** The moment you ask others to assist in the decision, thrombolysis goes out the window.
  - **A 44 year old patient with no comorbidities presented with a massive PE without hypotension.** The heart rate was 110 and bedside ultrasound showed an estimated right ventricular systolic pressure of nearly 70mmHg. The pulmonary and critical care physicians did not do anything. The emergency physician had a long discussion with the patient and his wife. Within 15 minutes of receiving thrombolysis, the tricuspid regurgitation had disappeared.
- **From Chris. What are your thoughts regarding prescribing over the counter medications?** Some patients have a difficult time just paying for food. It depends on the situation.
- **Does the slope of the hemoglobin dissociation curve really fall off at 85% rather than 90%?** Some of this has to do with the percentage error of the oxygen saturation monitor. Previously, we were worried at an oxygen saturation of 90% because the monitor had accuracy of +/- 5%. Newer generations are probably more accurate with an error rate of 1-2%. An oxygen saturation of 88% is more likely to be accurate.
  - **Temperature, pH and 2,3-DPG also affect the hemoglobin dissociation curve.**