Audio Companion for SESAP® 16
SURGICAL CRITICAL CARE — Category 9

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The following faculty report no relevant financial interests: Drs Jeremy W Cannon and Lena Marie Napolitano.
Drugs Commonly Used in Critical Care: Part I

**Norepinephrine:** I view norepinephrine as the bedrock first-line agent for most forms of shock in the context of actively resuscitating a patient to be sure his intravascular volume status is adequate. I reach for norepinephrine on a fairly routine basis as my first-line vasopressor therapy for patients in shock. **Complications:** The old saying, “Levophed equals leave ‘em dead” is probably overstating things. At the usual doses, I do not frequently see complications. Of course, there can be peripheral vasoconstriction-related complications, you can have some arrhythmias with norepinephrine, but those are actually fairly uncommon.

**Norepinephrine vs Dopamine:** For the septic patient, I find that norepinephrine is more effective than dopamine. I generally reserve dopamine for a younger patient with more reserve because it works indirectly through the release of native adrenergic reserves, plus it has sort of a mix of different end-receptors on which it is effective. I find — being more of a purist — norepinephrine is really my preferred agent.

**Dopamine:** As you escalate through the different doses, you transition your target receptor so at the low doses theoretically, it is more of a vasodilator. But in the last 5 to 7 years, I have decreased my use of dopamine pretty significantly. Its role in trying to preserve renal function is questionable, and its role as a vasopressor is pretty minimal. The one circumstance I will sometimes reach for dopamine is in a young trauma patient with a spinal cord injury. I want to get something on quickly, and I am less concerned about the arrhythmogenic effects of dopamine. In that young population with some catechol reserves, I find it can be effective, but in general I have steered away from using dopamine.

**Epinephrine:** Epinephrine is tried and true and really is a very effective vasopressor and inotrope. The context in which I typically use it is a septic patient or a patient in some form of shock with a depressed cardiac function. I have some evidence that tells me cardiac function is compromised, also I will add in a little epinephrine as typically a second- or a third-line agent.

**Dobutamine vs Milrinone:** Dobutamine is an agent I have used at times in the past. If I am going to try to pick a vasopressor or inotrope with some vasodilatory effects, I really prefer milrinone. I find that dobutamine is more arrhythmogenic, and I just have more comfort and more confidence in the effects of milrinone. Using milrinone, I generally will start at a fixed dose. I suppose that if I am not getting any sort of benefit early on I would add in a bolus, but generally it is a fixed dose. It is a phosphodiesterase inhibitor — its nickname is an inodilator — so you get some inotropic effects plus vasodilatory effects, especially in the pulmonary vascular bed, and even peripherally as well. It increases your contractility while decreasing your after load a little bit.

Drugs Commonly Used in Critical Care: Part II

**Phenylephrine or Ephedrine:** I see ephedrine as sort of a short-term bolus emergency intervention generally used by anesthesia colleagues, a kind of reactionary medication administration in someone who has a pretty precipitous decline in blood pressure after anesthetic induction. I really do not use ephedrine in my practice. I believe that recent evidence would support the use of norepinephrine in the setting of neurogenic shock, so I have steered away entirely from ephedrine and phenylephrine or Neo-Synephrine®. Theoretically, if you are going to use phenylephrine or Neo-Synephrine, you would want to use it in a patient with neurogenic shock, but I have found norepinephrine works quite well in that patient population too. It also decreases the number of drip amounts or drip doses you have to memorize.

**Vasopressin:** Typically I have used vasopressin as a second- or third-line agent. If I find I am escalating my dose of norepinephrine I will add it in — not to titrate, not to adjust the dose. I certainly do not bolus vasopressin, but I think of it as a background adjunct to my medication regimen. I usually use it in a fixed dose — typically 0.04 units per minute — and do not adjust it, and it will be the first agent I discontinue.
Fenoldopam Valuable in Hypertensive Emergencies

Fenoldopam: I have a few colleagues that have really embraced fenoldopam, but for the most part I have not encountered very many people that have used it routinely in their practice. This is a dopaminergic agonist. It acts as a vasodilator. Its primary indication really is for hypertensive emergencies, but in the surgical critical care realm, is often applied similar to how renal dose dopamine has been applied in the past. My experience has been that you actually do get a fairly significant drop in a patient’s blood pressure when you initiate fenoldopam, and for that reason I have tended to steer away from it in most patients. The one situation where I have seen it applied with some regularity was in our burn ICU in San Antonio. It was part of a sort of a toolbox of different therapies designed to address renal insufficiency in that population.

Pain: Be Vigilant to Control but Cautious With Opioids

The recommendations in the recent CDC paper on opioid use and the concern for opioid abuse by patients has not directly impacted my practice in the ICU and in the trauma bay, but certainly I have been aware that when patients come back to the trauma clinic seeking refills on their narcotic medications we need to be especially vigilant to ensure we are not only controlling pain but also working with patients to use non-opioid type medications when feasible and when it makes sense. Morphee: I use morphine occasionally. The one population where I find its use comes up is in our ECMO patients. Some of the narcotics are more lipophilic and seem to be cleared by the ECMO membrane, so you start having to escalate the dose to control whatever pain the patient might have. This often comes up on trauma patients that need ECMO support. You are still managing fairly significant analgesic requirements, and in those patients I tend to transition from Fentanyl, which is generally my go-to narcotic analgesic over to morphine.

Equianalgesic Dosing: The 3 most common opioids are morphine, Fentanyl, and Dilaudid®. While I am certainly familiar with the equianalgesic dosing, for me it is mostly a gestalt. My approach is to work with the bedside nurse to titrate the analgesic dose at the bedside for the individual patient, starting with a general ballpark range for the dose, taking into consideration any previous narcotic use that they may have recorded in their medical history and adjusting up or down based on the individual patient’s response. I use Fentanyl most commonly. I have seen Dilaudid used as a continuous infusion, but that is not generally my practice. My practice is to use that medication more as an intermittent, as-needed dose. Certainly in a PCA form, I find Dilaudid is a fairly reliable analgesic. Our medical practitioners, physician assistants, nurse practitioners, and residents are familiar with the dosing, so that is generally our go-to medication when a patient is transitioning to a PCA or intermittent dosing for their analgesic.

Stop Propofol Early to Avoid Side Effects

It seems pretty much all patients on a ventilator end up on propofol at some point during their ICU stay. For better or worse, I find it is a very effective agent for sedation. There are some side effects — hypertriglyceridemia is a potential problem and then the feared or dreaded propofol infusion syndrome is certainly a concern — but, yes, Propofol is absolutely a mainstay ICU drug. We have routine cautions or guidelines. You have to start checking triglycerides after 3-5 days on propofol. For propofol infusions, there is no requirement or defined guideline we use, but generally if a patient of mine is on propofol for 72 hours or more I will check a triglyceride level. Propofol infusion syndrome is a real syndrome. If you have ever seen it, you will never forget it. I have, unfortunately, seen a couple of patients that have had propofol infusion syndrome, and I truly believe it is a real phenomenon. You have a patient who, for all intents and purposes, is recovering with a very reassuring overall clinical
trajectory. Then, for whatever reason, he remains on propofol and really, almost like a lightning bolt, he becomes very unstable with elevated CKs and lactic acidosis and just starts circling the drain. You think maybe it is a septic type of process, and then you rule out any sort of septic source. You are basically left with the propofol being the culprit, so you remove that. Then, through supportive care, the patient gets better.

**Dexmedetomidine Is Invaluable Agent**

**Dexmedetomidine:** I think dexmedetomidine or Precedex® is an invaluable agent to have available on your formulary. Depending on what hospital you are in, you may find that it is a bit of an uphill battle getting it onto the formulary, but I do believe it is important to have as a second-line agent or an alternative agent for sedation in critically ill patients. And for those that either cannot tolerate propofol for whatever reason or have contraindications to propofol and developed complications, it has become my second-line go-to agent. In addition, I find it is useful in patients that are experiencing hyperactive delirium or alcohol withdrawal. Although this is an off-label use, I feel like it is actually quite useful in those patients. I generally do not administer a bolus dose which is an unnecessarily risky approach to using dexmedetomidine as hypotension can occur secondary to the beta cardiac side effects. I generally will start the infusion and then titrate up, and I generally do not titrate above a maximum dose of about 1.0 µg/kg/hour (usually range is 0.2 to 0.7 µg/kg/hour) and then will reassess. If the patient is still not adequately sedated on that dose, I start thinking about other adjuncts to combine with the dexmedetomidine. I escalate the dose roughly every 15 to 30 minutes. As far as the effect, I would say it has a variable response. Not every patient is going to respond, but in those who do, you generally get a sense they are responding favorably to it pretty early on. In my experience, it is not a silver bullet for all of these patients, but for some it is actually quite effective.

**Benzodiazepines Best Given in Low Doses to Select Patients**

I have tried to steer away from benzodiazepines, especially benzodiazepine infusions. Those have been found to be independently associated with poor outcomes including mortality in an ICU population, so I am typically very stingy with benzodiazepine dosing. I will give low-dose intermittent administration to select patients, especially those that have alcohol withdrawal or those for whom I need an additional adjunct, in the patient on dexmedetomidine for example. **Case 1:** You have a patient with traumatic brain injury in your ICU and certainly has what appears to be neurostorming. What do you use to control his hyperdynamic state that does not include benzodiazepine? **Recommendation:** I have found that, for those that can tolerate enteral medications, there is 1 benzodiazepine that seems to not interfere with their mental status and will take the edge off of that sort of storming physiology. It is also very useful in those patients that have alcohol withdrawal. The medication I use is oxazepam or Serax. Although it is still a benzodiazepine, it seems to have less of a sedating profile than all the others. I generally start with 15 mg and will escalate up as needed — 15 mg tid up toqid — but generally I start at 15 mg tid, then reassess. If the patient is still not controlled, I will double it to 30 mg tid.

**Seroquel, Haloperidol Used in Agitated Patients**

In the patient who is agitated, haloperidol has been our go-to drug, but now we have Seroquel available. I struggle with the management of patients with hyperactive or kinetic delirium. I think haloperidol and Seroquel are reasonable to use, although Seroquel and Haldol are being used off-label when it is for that
indication because they are antipsychotics and both of them have a pretty concerning risk profile when it comes to cardiac morbidity and even mortality. The traditional complication of Haldol is torsades. If you are treating a patient with Haldol, you need to follow his or her EKGs on a daily basis. That being said, for the patient that is a danger to himself and the staff in the ICU, I will dose Haldol, but I tend to use a lower dose than we might typically have used in the past. I would use 1 to 2 mg, assess the response, and take it from there. As far as which one is better or more used, I would say our intensivists have mixed feelings. Not infrequently, I will come on to the ICU service and see a few patients on Seroquel. This is becoming more and more common, but it is often difficult to sort out if it was started for a hyperactive delirium or for sleep hygiene or insomnia. Nonetheless, I do see that Seroquel and some of the other atypical antipsychotics and some of the benzodiazepines are being used as sleep adjuncts.

**Ketamine: A Wonder Drug in Some Patients**

Ketamine has crept into the world of critical care out of the operating room. It was an invaluable agent for my patient population in San Antonio where we had a number of combat casualties in particular that had these spirals of acute on chronic pain after severe multi-extremity, multi-cavitary injuries. For those patients, it was a godsend, a wonder drug in many ways. During a ketamine infusion, they would have evidence of disassociation. As they sort of emerged from that infusion therapy, they were dramatically improved from a chronic pain standpoint. It is sort of a multimodal pain regimen. Patients are given an infusion and then weaned off to some other pain regimen. We also use it in our trauma bay for our patients that do not have IV access that are a little rowdy and need some redirection. It allows them to be calm and not continue to be a danger to themselves and our staff, and it allows us to manage them more effectively, so I use a one-time IM dose in those trauma patients that do not have IV access.

**Etomidate Excellent Drug but Comes With Risks**

Etomidate is an excellent drug for rapid-sequence intubation. It is rapid onset and is very effective as a general anesthetic. However, it comes at the expense of very occasionally having an adrenal suppression effect. So, if you have a patient that you use etomidate for in your trauma bay or some other emergent airway situation in your hospital or in the operating room even, and several days later they have become hemodynamically unstable and just do not look right, consider adrenal insufficiency in those patients. I have seen 1 or 2 patients that may have had clinical adrenal suppression after 1 dose, but it does not come up often. However, it is good to know about this so, if you do have that 1 patient, you might make a difference.

**Cardiac Drugs Vary in Popularity, Use**

**Beta Blockade:** Beta blockade and metoprolol seem to be the workhorses these days. The most important patient population in which to use a beta blockade are those patients who come to you on a beta blocker as an outpatient. If you acutely withdraw that beta blocker, they will definitely have evidence of a withdrawal syndrome. **Labetalol:** I find that labetalol is an agent I prefer for patients with spikes in blood pressure. I prefer labetalol to hydralazine for example, but you do have to be very careful with the dosing. Like some of our other ICU base medications, you can sneak up on the dose. Do not just give a big bolus of the largest dose you can find; low ball the dose and sneak up on it. I actually really do find labetalol very helpful. **Calcium Channel Blockers:** Two calcium channel blockers that seem to crop up in the surgical population are diltiazem and verapamil. I really use diltiazem; I do not use verapamil as much.
Diltiazem is my second-line agent for rate control in patients with tachyarrhythmias, specifically atrial fibrillation, generally used as an infusion second-line after beta blockade has failed.

**Adenosine:** Adenosine is in our code carts, and I have used that occasionally — probably about once a year — in a patient that has a supraventricular tachycardia or tachyarrhythmia. You must be aware when you dose adenosine per the ACLS algorithms, your patient is going to go generally asystolic for a few seconds. You sort of hold your breath and hope that it comes back, and most of the time they do come back.

**Amiodarone Treatment Often Continue Too Long**

Amiodarone is probably in the water in most cities in the United States, but I do not feel like the weight of the evidence supports 1 strategy over another for a patient in an atrial tachycardia. I find that, for most of our postoperative surgical patients with a few days of rate control and aggressive attention to reversing the underlying cause — be it a fluid shift, a new infection, what have you — they generally will revert back to a sinus rhythm within that early period.

**Dr Weigelt’s Experience:** We found a very interesting phenomenon about the acute treatment of atrial fibrillation most commonly. At least 25% of the patients who get started on 1 of the treatments, choose your treatment, are continued on that treatment even after atrial fibrillation is resolved, as you said their primary problem is resolved. One of the reasons we fear amiodarone is because the long-term effect; the complications of amiodarone are not to be ignored, so we routinely try to get them under control and then stop the drug before patients get into the regular wards because it seems to always continue into their discharge medications.

**Digoxin:** I would have to go back a few years to the last time I used digoxin. I have continued it in patients that for whatever reason are on it as an outpatient. As far as using it acutely as a new therapeutic intervention, it is a distant third-line agent for me, probably in a patient I have discussed with cardiology and a unique clinical scenario in a patient that has some sort of cardiac-specific issue that warrants digoxin.

**Tips for Extubating an Agitated Patient**

**Case 1:** A trauma patient has been intubated for his decreased mental status and is found to have no injuries but found to have an elevated blood alcohol concentration. He is admitted to your ICU. How do you decide to extubate this pretty simple scenario in a patient with altered mental status and increased blood alcohol content?

**Recommendations:** This is a daily, nightly occurrence, and then we sort it out in the morning. This is a fairly straight-forward scenario. In order to begin to think about this patient, I start by listening to his story, looking back at his past medical and surgical history to see if he has had any underlying conditions that might predispose him to failing extubation. As far as my bedside patient evaluation, I want to be sure the effects of alcohol and any other substances that might have been on board have worn off, that the patient is awake and interactive and able to follow commands. Ideally he would be able to lift his head off of the bed and demonstrate he has good strength. As far as respiratory mechanics, on the ventilator I generally would do a spontaneous breathing trial as part of my assessment for ventilator liberation where I turn the patient to fairly minimal ventilator settings and then allow him to breathe spontaneously on the ventilator and assess his tidal volume and respiratory rate once I have set the ventilator to a minimal level of support. And then, provided he is awake, interactive, demonstrates he needs very little support, he has had no traumatic injuries, and his medical history suggests he does not have any medical problems that would predispose him to ventilator liberation failure, then I will go ahead and remove the tube.

**Case 2:** As you are going through these steps, the patient becomes agitated. The patient starts swinging at people. Do you get all of those things done on all patients, or do you ever just extubate these patients?
**Recommendations:** About half of these patients are calm and wake up very nicely just like you would want them to. The other half turn into what I call a *honey badger* — they are swinging all over the place and are difficult to manage. In that setting, sometimes you do have to do what I call a *rapid sequence extubation*. You have to make your best judgment. Is this patient swinging and agitated because of the intubation or because of the mechanical ventilation, or is there some underlying other problem that would make extubation unsafe? For those who are simply irritated by having an endotracheal tube in and otherwise their mechanics look good and they have no issues with their airway that would preclude extubation, I will go ahead and pull the tube. For most patients in the surgical or trauma ICU on a ventilator who might now be at a point where they no longer need the ventilator, this is the general framework I use as well. As far as rapid shallow breathing index, lower is better. If it is up around 110, I may be nervous about extubating. If they are in the midrange — 45 to 80 — then I am thinking about noninvasive ventilator adjuncts as something I would use immediately upon extubation because that middle range rapid shallow breathing index value would suggest they are a little tenuous or have some risk for needing reintubation.

**Tips for Extubating a Patient With Rib Fractures**

**Case:** You have a young patient, about 35 years old, who was in a motor vehicle collision with multiple rib fractures. He had a VATS with a rib fixation procedure on postinjury day 3, but low and behold, he came back from the operating room and failed extubation. You reintubated him. Now we are postinjury day 5. Does this patient get any different approach to trying to see if he can be extubated? **Recommendations:** My framework in approaching him is generally the same, but for patients with trauma and especially those with rib fractures, I really want to be sure that they have superb ideal optimized pain control, be that with IV analgesics or probably even better an epidural catheter. I would want to assess the patient to see if I can determine why he failed extubation. Theoretically, after rib stabilization, his pulmonary mechanics should be much better. So why was it he failed extubation? Was it a pain control issue? Was there some other upper airway or pulmonary parenchymal process that led to the reintubation? I would want to get that sorted out. But then, assuming that all of those were not fractures, I would want to be sure that his pain control is optimized. Pain control is as important as any kind of assessment of the pulmonary mechanics.

**Pay Attention to Pulmonary Mechanics in Elderly Patients With Rib Fractures**

**Case:** You have an elderly patient who has been intubated after she fell down the stairs. She was intoxicated at the time, but now she is pretty cooperative. She had 3 rib fractures on the right and 1 on the left. She has developed an effusion on the right side, and you have tapped it off. However, it looks like it is not blood; it is serous. How does that patient get extubated in your unit? **Recommendations:** In patients with rib fractures in the ICU, I really do believe optimized analgesia is wildly important. Given her age and her underlying injuries, I would want to pay careful attention to her pulmonary mechanics. I would want to assess her first for pain control but then also for her ability to sustain her respiratory function without the ventilator. I would assess her rapid shallow breathing index. This is a patient where I might want to also determine if she has a strong cough. It is a bit difficult on a ventilator, but you could deflate the cuff and see if she can breathe around the endotracheal tube or cough around the tube and see if she has enough strength and adequate pain control to do that. I think some additional precautions in this older patient are warranted. As far as the effusion goes, I have occasionally seen a reactive effusion in these patients, but more typically it is a hemothorax, which makes me wonder if maybe they did not fully assess the effusion and she needs a chest tube prior to extubation.
Noninvasive Ventilation Delaying the Inevitable

The only ideal candidate for noninvasive ventilation is someone who uses BiPAP or CPAP at home. In all others, it is an adjunct. In some cases, it is forestalling the inevitable — reintubation. If you delay reintubation too long because you have tried noninvasive ventilation as a postextubation bridge excessively, then those patients can have poor outcomes. In the patients who use BiPAP or CPAP at home, I want to be sure they have their family or they have brought their own mask from home so that we get a good fit. I would be a little leery of using noninvasive ventilation, particularly in patients who have had upper GI or esophageal surgery. They could swallow some of the air. They could get distention of their esophageal conduit. For those patients, it is perhaps not the ideal therapy. In those with a nasogastric tube in, you sometimes are not able to get a good seal around the face. There are full-face type noninvasive ventilation masks that some patients find work better, but in general it is those patients with a nasogastric tube or those that have had esophageal or foregut surgery for whom I am a little concerned.

Airway Management and the Use of a Difficult Airway Cart

We have laryngoscopes available in our difficult airway tackle box, but we routinely have the videoscopes available as our first-line intubating tool. For me, the transition from the laryngoscope to videoscope is very akin to laparoscopy — once you have that sort of eye-hand coordination, looking at a video image and then coordinating what your hand is doing, it is fairly straight forward. With videolaryngoscopy, the challenge is you can often see things with the scope that you cannot necessarily get to with your tube. So, you have to bend your tube in a slightly different way and use the dedicated stylets if it comes with the videolaryngoscope. Otherwise, you will get into a situation where you can see the airway and you can see what is going on, but you cannot do anything about it. You cannot get the endotracheal tube down to that location.

**Difficult Airway Cart:** We have a difficult airway cart in each one of our ICUs. It is arranged on the same principles as a code cart. At the top are tools for less invasive measures, and as you work your way down to the bottom drawers of the cart, you get into more invasive measures. We have all the potential tools and adjuncts we might need to manage a difficult airway — starting with a suite of different endotracheal tube size options, laryngoscopy blades, handles with charged batteries, and the video laryngoscopy equipment is on the cart. And then as you work your way down, you have other adjuncts such as an intubating LMA. You have a Cook catheter that you can use to exchange tubes over. For an awake bronchoscopic or video laryngoscopy with a fiber optic scope, we would have to bring that scope separately. We do not keep it on our difficult airway cart, but the respiratory therapist has ready access and will bring it to the bedside if needed. The bottom drawers are where you have your surgical airway options — the tracheostomy, the cricothyroidotomy to include both the surgical kits, and a percutaneous option. We definitely do not exhaust every option before we get down to the surgical airway. Most of the time, it is an overall gestalt and a discussion between the surgeon who has responded to the rapid respond and either the anesthesia or emergency medicine practitioner that might be there as to what we think the best, first approach is. Usually, your first shot is your best shot, and you want to make it a high likelihood attempt that you are going end up with an airway in place. So, in a patient who has a massive angioedema, for example, your first shot may actually be a surgical airway. We have a gentleman’s agreement that once we start looking with whatever device we want to look at it through the mouth, 3 attempts and then the surgeon enters the field to do the emergent surgical airway.
Percutaneous Tracheostomy a Tremendous Advance in Airway Management

Percutaneous tracheostomy is a tremendous advance in airway management over the past decade, and I would really commend to the listeners the article by Kornblith et al from Denver that was published in The Journal of the American College of Surgeons in 2011, where they document their experience with 1,000 bedside percutaneous tracheostomies in the surgical ICU. They demonstrated this procedure could be done safely and at the bedside with a very low complication rate in the hands of experienced surgeons and intensivist. I do perform this procedure at the bedside and will make a case-by-case determination as far as whether the patient would be better served in the operating room versus the ICU, but generally, I feel like most patients safely undergo a bedside percutaneous tracheostomy in the Surgical ICU. **Steps of Percutaneous Tracheostomy:** Anytime I am approaching a patient that needs an airway, I start with positioning and lighting. I will use a transverse shoulder roll to bring the patient’s neck forward into the field, and then I get my right spotlight right on the field. As far as the actual insertion steps, I have an airway or an endotracheal team that is dealing with the endotracheal tube, and then I have the tracheostomy team that is inserting the tracheostomy. The endotracheal team starts by using what our colleagues in Denver document in their manuscript — they will withdraw the tube to the level of the vocal cords under direct visualization using a laryngoscope. The very step is to pull back the endotracheal tube just beneath the level of the cords. Then, while holding that tube in place, I insert a bronchoscope and do the actual insertion under bronchoscopic guidance. As far as the surgical part of the procedure, down at the neck, I will usually make a transverse incision if it is a non-emergent airway, approximately 2 fingerbreadths above the sternal notch and 1 fingerbreadth beneath cricoid cartilage, a small transverse incision, dissect to the tracheal ring. Generally you are able to feel the tracheal ring with just a couple of spreads. Sometimes you may have to go north or south a little bit to get around the thyroid isthmus, but usually that is not too difficult. Once you are at the level of the rings, you access the airway using an access needle, and then you can usually see the entry of the needle on your bronchoscope. So, you are accessing the airway using a syringe with a little bit of fluid in it, you are pulling back on that syringe as you are advancing your needle, and then — when you get into the airway — you will both see it on the screen with the video bronchoscope as well you will see air bubbles in your syringe. Once you have your access, you advance a guide wire through the access needle, and then withdraw the needles. Now, you are able to use that access point essentially like a Seldinger technique to dilate up the track and ultimately advance a tracheostomy tube into the opening. My approach is to dilate up to an 8 Shiley tracheostomy tube. I use the Blue Rhino dilator to dilate up to the luminal diameter and then advance the 8 Shiley tracheostomy tube into the airway once I have dilated to that diameter using the Blue Rhino.

Ultrasound-Guided Radial A-Line Is First-Line Approach to Monitoring

Early in our patient’s ICU course, if they are on a ventilator and I need to monitor their blood gas and laboratory values with some frequency, more than say once a day, then I will go ahead and place an arterial line. Ultrasound-guided placement of a radial A-line is a pretty routine standard procedure that ICU physicians should be facile with as well as advanced practitioners depending on your location. Ultrasound-guided radial A-line is our first-line approach. **Case:** You have a difference of 20 mm Hg between your invasive and non-invasive blood pressure values. How would you adjudicate that? You are also giving the patient something for hypotension on top of it. Do you always follow the lower one just to make yourself feel good? **Recommendations:** It depends on the waveform from the invasive monitor. After a while, you get a sense for what an arterial waveform should look like when the catheter and the system are working correctly. But if you are getting artifact, big spiking waveform patterns and that sort of thing, then maybe your values from the arterial line are not that reliable. In general, I take all the numbers into
consideration and then come up with some determination as to what I think, together with the patient’s clinical picture, is the most accurate approach — or most accurate value. Just from a monitoring standpoint, it is important to realize the non-invasive pressures you are seeing on the monitoring — the systolic and diastolic pressures — are derived numbers. The blood pressure cuff is actually measuring the mean pressure just through the way that the blood pressure cuff works, so your mean is probably going to be your most accurate number on a non-invasive measurement. For the arterial line, it is directly measuring the maximal and minimal amplitude of that waveform, so your systolic and diastolic pressures will be more accurate, whereas the mean pressure on that A-line is derived from those directly measured systolic and diastolic numbers.

**Pressure Monitoring Guidelines:** When writing guidelines for pressure monitoring, our tendency has been to go with the mean pressure, even though for an invasive monitor your mean pressure is a derived number. Most of the guidelines that deal with patients in shock do recommend targeting a mean pressure. Occasionally, if I am not quite sure that I am getting a reliable number, I will assess the insertion site. Especially if it is a radial insertion site and if the patients on vasopressors for example, I tend to like a femoral arterial line as opposed to a radial A-line. In those patients, I will frequently get a non-invasive pressure as well to get that directly measured mean pressure.

**Machines for Cardiac Output Helpful but Not Without Pitfalls**

Arterial wave contour analysis for a cardiac output is another great innovation in critical care over the last several years. Those that have been monitoring arterial lines for a while probably feel like it is an unnecessary luxury to have a machine tell you what a seasoned ICU physician can tell you by just looking at the waveform, but these new devices automatically interpret the change in the arterial waveform with mechanical ventilation breaths. At each mechanically delivered tidal volume, you will change both the pre-load and the after load. As a result, you will change the waveform of the arterial line. These monitors are able to pick up those subtle variations and from those variations get some sense of the patient’s intravascular volume and some other hemodynamic parameters.

**Pitfalls:** These waveform analysis devices really rely on a good waveform. If you have a poor-quality waveform or a patient that is in an arrhythmia, the reliability of quality of your interpretive measurements — these waveform analysis measurements — will be suspect. Also, it requires an adequate tidal volume. At a minimum, 8 cc per kilo has been found to be necessary to actually give you that reliable fluctuation, and the patient needs to be sedated enough so that they are entirely mechanically ventilated. For the awake, spontaneously breathing patient, these monitors are not very reliable.

**Pulmonary Artery Catheters:** There are select patients in whom I really do think pulmonary artery catheters are invaluable, especially the patient with an arrhythmia. They could be fully mechanically ventilated, and the waveform analysis will be completely invalid because they got an arrhythmia. The patients in whom I tend to start thinking about using a PA catheter are the elderly with some degree of cardiac dysfunction and some degree of renal dysfunction. It is very difficult to sort out their intravascular volume status with these other newer approaches we have. That is when I still reach for the tried and true PA catheter. Two other challenges I find with PA catheters are finding a nurse that has actually hooked up the equipment and finding a monitoring box that is still functional.

**Know How to Perform a Straight Leg Raise**

For the awake, spontaneously breathing patient that may have had a slight decline in clinical status — perhaps she is a little on the oliguric side and you want to sort out what her volume status is — a straight leg raise is an important adjunct for an important technique to know how to perform. I do not think a lot of us in the ICU realm really understand how to perform it, and there are a couple of nice reviews that
have gone through the literature on how a straight leg raise should be performed and what maneuvers make it most reliable. For me, the patient starts out in the typical head-up position, then you lie the patient down flat and bring both her legs up to at least 45° at the same time either with a wedge or with the foot of the bed. Typically, you have to get a wedge to get the legs up high enough. The parameters you are monitoring are not so much the blood pressure or the CVP but the cardiac output. And for me, that is where one of these other non-invasive measures — like the NICOM bioreactance monitor that will give a non-invasive cardiac output — is useful. Others have described using a transthoracic echo while you are doing this to look at the cardiac output and measure the aortic arch blows to see what effect your straight leg raise has had.

**Ultrasounds Useful in ICU**

I think we should have ultrasounds on our ICU rounds with us. This is an important technique to have. It is user dependent and patient dependent. Even in the most challenging of body habitus patients, you can still get those great windows to see the vena cava. You also need some pretty good equipment. I prefer to use the cardiac probe or the phased array probe — the square one — when I am assessing the IVC. It gives you a sense for the patient’s intravascular volume. In some ways, though, it is like the rapid shallow breathing index where there is a range and spectrum. You cannot say, “Okay, this patient has an IVC that is 1.5 cm in diameter,” and know exactly what that means. You have to stay in there and watch it. You have to see what the IVC does during respirations, either spontaneous or mechanical. If you still cannot quite tell, then give the patient a fluid bolus and reassess the IVC. It is really a dynamic monitoring device rather than one in which you put the probe on, get a measurement, and then make a decision.

**When Is Nutritional Support Necessary?**

There is a great website that is a compilation of guidelines on nutrition. There is so much in the literature these days that it is sometimes difficult to stay abreast of these guidelines, even for those who eat, drink and breathe this every day. The website is criticalcarenutrition.com. The site has a nice layout with all the most current guidelines with regards to nutrition. With that in mind, the patient that has a high-risk nutritional status — specifically if he is malnourished with evidence of temporal wasting and wasted hypothenar eminence or thenar eminence — really benefits from early nutritional support. In those who are nutritionally replete, you have time to sort this out. They do not need nutrition in their first 24 to 48 hours of ICU stay.

**Case:** You have a patient who is nutritionally replete and is on one of those pressors.

**Recommendations:** Your concern is outstripping the blood supply of the small bowel which might cause intestinal ischemia. Historically that is the teaching. But in the patient that is well resuscitated with an adequate intravascular volume and is on a stable dose of vasopressors, I would still consider using enteral nutrition support if they have evidence of being a high-risk nutritional patient.

**Parenteral vs Enteral Nutritional Support: Which Is Better**

As long as there are IV catheters and nasoenteric tubes, there will be a debate about which modality is better. I think the debate is alive and well and continues. There is certainly a little bit of a religious element here — the place you trained and the organization you grew up in really can influence your practice, as it does in other areas. I would say the weight of the evidence nowadays is probably as equivocal as ever, but in general it is felt that enteral nutrition has a lower risk of infectious
complications whereas parenteral has a higher risk of not just complications from the central access but also a higher rate of infectious problems down the road. If you use enteral nutrition, use of a post pyloric tube is a patient-by-patient decision. If the patient has a functional GI tract and there is no reason not to use the GI tract, some form of enteral access is important. If the patient has a pneumonia and has respiratory compromise from that, I do try to make some effort to get the enteral feeding access not just post pyloric but actually post ligament of Treitz. This is not clearly demonstrated in the literature, but I think feeding — even beyond the ligament of Treitz — may be better than feeding the proximal GI tract. As far as gastric versus post pyloric, I think it has been difficult to tell if one is superior to the other. In some respects, with a transpyloric tube, you are actually stenting the pylorus open, so you might get reflux of duodenal content into the stomach. This may be part of why we have not been able to demonstrate a difference between the two. If I have a high-risk patient with respiratory issues so that I am concerned about aspiration or concerned about a pneumonia, I will make the effort to try to get it beyond the ligament of Treitz. I have seen all different techniques to try to get a tube beyond the ligament of Treitz. The one I have settled on and like the best is an electromagnet tracking system in which it tracks the tip of the feeding tube as it goes down through the stomach, and it is displayed on a screen. There is no magnetic device or anything that sort of drags it into place. It is still a push-type technique, but you can at least see a graphic of where the tip of the tube is. For me, this has been a pretty reliable approach. I then confirm the location or placement of the tube with an x-ray at the end just to be sure I am in the right spot. Some of the nurses get to be really good at this technique as well.

The Value of Hypocaloric Feeding

Hypocaloric feeding is a really interesting concept, but I would say it is probably not without some risk — the risk of misinterpretation. I absolutely agree with the concept of hypocaloric feeding. You do not need to ramp up the patient’s carbohydrate and fat content to absolute normal levels immediately or throughout his early ICU stay, but for all patients who are getting nutritional support in the ICU, it is vitality important to be sure they get adequate amounts of protein. You want that patient to be in a positive nitrogen balance so that he will have the building blocks to recover from his surgery or heal his traumatic injuries or recover in general. It is pretty clearly demonstrated protein is the most important part of that equation. I think hypocaloric feeding, especially in the low nutritional risk patient population, is absolutely acceptable and probably the approach we should all adopt so long as the patient gets enough protein.

Adult Respiratory Distress Syndrome Redefined

New Definitions: The Berlin definition is the new definition for adult respiratory distress syndrome (ARDS). It changed the category of acute lung injury (ALI) to instead a definition of mild ARDS. Previously, acute lung injury was a PaO\textsubscript{2} to FIO\textsubscript{2}, a PF ratio of 200 to 300. Now, ALI is dead and instead has been re-categorized as mild ARDS. The other 2 categories are moderate ARDS and severe ARDS. Moderate is defined as a PF ratio 200 to 300 and severe is defined as ARDS <100. This is a very important new definition of ARDS because it now separates the syndrome into severity of hypoxemia. The severity of hypoxemia impacts outcomes in ARDS — the more severe the hypoxemia, the higher the mortality rate. When it was initially published, the new Berlin definition really reviewed the mortality rate in each cohort of mild, moderate to severe. It did show a higher mortality in the severe cohort.

Implementation: The most important aspect of the new definition is how it will be implemented. For severe ARDS, our practice is going to move to rescue strategies more quickly because the death rate is higher and you want to get patients out of the severe hypoxemia category. The rescue strategies of prone positioning, nitric oxide, ECMO, and recruitment maneuvers are all valid in that category. We would propose that we use the new categories and new definitions of ARDS and strongly
recommend severe ARDS — again PF ratio <100 — should go to an ARDS referral center, particularly where they are expert in the implementation of rescue strategies including the use of ECMO. I think we are moving in a very good direction, in part related to preserving the high-risk and high-cost therapies to the severe ARDS cohort.

Strategies for Everyone on Mechanical Ventilation

Mild ARDS: I believe the strategies for treatment of mild ARDS — low tidal volume and low plateau pressures — should be used in everyone on mechanical ventilation. It is a strategy of trying to not cause lung injury. The question becomes whether or not it invokes a change in patient outcome. This patient cohort was included in the ARDSNet trials and will be included in the new PETAL Network trials.

Severe ARDS: At the University of Michigan, we have a severe ARDS algorithm we use. It is an evidence-based algorithm we established with our pulmonary critical care colleagues, and we use it institution-wide both in the Medical ICU and in the Surgical ICU. We really only get severe ARDS patients referred to us because of our ECMO program. When a patient presents with severe ARDS — again defined as a PaO\textsubscript{2} to FIO\textsubscript{2} ratio <100 — they have striking disease. Most of them are in some degree of shock, and many of them are in renal failure as well. For hypoxemia treatment, our algorithm first is low tidal volume ventilation with high PEEP and keeping plateau pressures low. Our next step in the algorithm is a consideration of recruitment maneuvers if we think they have recruitable lung, and there we are fond of using high plateau pressure for a defined period of time, such as a plateau pressure of 40 on CPAP for 40 seconds. We will remain at the bedside because the major complication is hypotension. The next step in our strategy is really to optimize PEEP based on oxygenation and compliance. Our subsequent rescue strategies are related to cost, simplicity, risk, and the team being advanced in those strategies. So, the next 2 rescue strategies that we consider for patients who do not respond to the baseline approach are prone positioning, which in the PROSERVA trial had a very large effect size, and inhaled nitric oxide. If there is no response to these strategies, then we moved to consideration for ECMO.

Two Strategies for Recruitment Maneuvers Based on Breathing

Patients Not Spontaneously Breathing: Most of our patients on transfer are neuromuscularly blocked already from the outside hospital and are not spontaneously breathing. There we usually put them in a CPAP mode with an inspiratory plateau pressure of 40 and hold it for 40 seconds. Then we can go higher. Clearly,Gattinoni has identified that not all patients will respond to 40, and he has shown, by repeat CT imaging, that alveolar recruitment and lung recruitment of de-recruited lungs sometimes require higher pressures of 50 or 55 and even up to 60. But again these are transient — 40- or 50-second maneuvers.

Spontaneously Breathing Patients: These patients will have a hard time holding their breath for a minute. We put them in bi-level ventilation and use a rate of 20, an I:E ratio of 1:1. We do the initial recruitment maneuver with a mean airway pressure of 30. It is 40/20 — so, a high PEEP of 40, a low PEEP of 20 — that gives a mean airway pressure of 30. The advantage to this approach is you can hold that for much longer because it is not just an inspiratory pause. We can do it for 2 minutes, but if the patient is continuing to improve oxygenation, we could hold it for 5 minutes or 10 minutes at that level and then step down. Then we try to determine optimal PEEP. We try not to step down too quickly to de-recruit again. We are monitoring to try to identify optimal PEEP; we are looking at pulmonary compliance and trying to identify the PEEP level in which we have optimal pulmonary compliance.
Recruitment’s Relationship With Pressure Controversial

Some believe recruitment is related to the peak inspiratory pressure, while others think it is related to mean airway pressure. There are controversial data supporting both camps.

**Mean Airway Pressure:** I really think its related to the mean airway pressure, but I could not tell you that for certain I am correct. I find I get better results standing by the bedside and using a peak inspiratory pressure to recruit.

**Peak Inspiratory Pressure:** Our respiratory therapists firmly believe you need to keep increasing the PEEP for a protracted period of time. But to each his own, I guess. Clearly, the older I get, the more I do not want to paralyze the neuromuscularly blocked patients. We try to bring them up and let them spontaneously breathe as quickly as possible, because then you can use high PEEP. And most of our patients with ARDS, again it is mostly hypoxemia, you can get to breathing even on pressure support with high PEEP. They do not need much support for ventilation.

Steps to Safely Prone Patients

We have a very simplistic approach to prone patients. We use 4 staff members, usually 1 or 2 physicians and 1 or 2 staff. We basically pull the patient over to the side of 1 bed. We tuck a half-folded sheet under him and go half way, so 90°, and then we pull him a little bit more to the side and flip him. It is basically like flipping a pancake. If he is a critically ill patient, we do the 90° step and then put a wedge under 1 side. This is a very important thing that we do not teach enough, but then we determine which lung is better and put that lung down. On the chest x-ray, there is usually 1 lung that has denser infiltrate. We put the good lung down for 2 reasons. We put the more lucent lung, less infiltrate, down, meaning dependent, so that it gets better blood flow and therefore improved gas exchange. The other reason to do this is that the PEEP will go preferentially to the up lung which has the greater infiltrate and in greater need of recruitment. Basically, we go to the bedside, look at the x-ray, and discuss which lung is better. Then we are careful about how we position the endotracheal tube. A very good thing we have determined with this approach is that the backside is fully available, so you can clean his backside. We protect the face and the eyes with 2L saline bags in a pillowcase. It is basically like a water bath for his face. And then we put 1 arm up and 1 down by the sides, so a swimmer. We believe in the PROSERVA trial results, and so we do 16-hour prone positioning. This is standardized in our ICU; everybody knows our prone positioning goes from 4 PM to 10 AM. We have chosen those times in part because it does not invade into shift change. It is important to protocolize what you do and do it very safely. Every time you prone a patient, dysrhythmias can occur, hypotension can occur. We want the whole ICU team there, so we do it before evening rounds and after morning rounds.

Cannula Use Has Both Advantages and Disadvantages

We have transitioned to using ECMO in a way that provides the ability for the patient to be awake, alert, breathing spontaneously and sitting up. We promote the use of a dual lumen bicaval cannula. A right IJ Avalon cannula is a cannula where the tip is in the inferior vena cava below the diaphragm for removal of blood to the circuit for oxygenation and then the inflow is in a medial port in the right atrium adjacent to the tricuspid valve.

**Advantages:** The advantage to that is it is a single cannula for both blood removal and blood return, and in those patients we can sit them up, we can walk them, they can stay awake and alert, we can extubate them. This approach has been a huge advance. Some evidence suggests this method produces less re-circulation because outflow to the machine comes from the tip of the cannula that is in the
inferior vena cava. There is another port in the superior vena cava, so that you get deoxygenated blood from those areas. Oxygenated blood goes into the medial port that is in the middle of the right atrium.

**Disadvantages:** The disadvantage of the use of that cannula is that it has to be placed with fluoroscopy because of a high risk for right ventricular rupture if you do not. So, that is a challenge. You need to have a stable patient to be able to move them to fluoroscopy. On occasion, we have a patient that really is on death’s door — his PaO₂ is 30 or 40, he is hemodynamically unstable on multiple pressors — and then we still resort to the traditional right IJ, right femoral approach. This is an approach where we remove blood from the right femoral — it is really a long line in the inferior vena cava — and then we infuse oxygenated blood into the right IJ. The disadvantage is the patient really cannot get up out of bed, cannot ambulate — these are very large lines. The Avalon cannula is a 31 French; it is the size of a chest tube. It is a bit challenging, but circuits have gotten smaller, more efficient. Overall, it is a far simpler and simplistic system than we have had in the past.

**Management:** When I first arrived in 2005, we still had ECMO specialists at the bedside; we now just have 2 ICU nurses. We have 1 ICU nurse who does all the charting and takes care of the circuit and the other does the patient care. So, it has transitioned to an extracorporeal approach that can be managed by routine ICU nurses with specialized training, just like we do for CRRT. In the future, I think we are going to see more and more extracorporeal support because of the new studies for extracorporeal future removal for COPD patients so that they never need to go on the ventilator because those patients really have a problem with hypercarbia and not hypoxemia.

**Standard Protocol to Heparin Bolus**

We have a standardized approach to a heparin bolus with implantation of the ECMO line. The patient in whom we are not concerned about anticoagulation at all will get 100 units/kilo, but in many of our patients we modify that dose to 50 units/kilo or an even smaller dose if we are worried about anticoagulation. Because the flow is so high — 4.0 L/minute or 4.5 L/minute — we usually do not have a problem with no anticoagulation. Once the line is in place, we promote continuous heparin infusion to keep the PTT 45 to 50. Bleeding is a complication on VV ECMO. We are worried about bleeding, so we keep the heparin drips at a lower level. If a patient develops bleeding on ECMO, we stop the heparin infusion. Our standardized protocol is to stop it for 4 hours, restart it in 4 hours if we want to. We can keep it ceased for a day or 2 if we need. For the trauma patient, this is very helpful. I think we monitor for bleeding now at the bedside very easily. If you had concern for someone who had intra-abdominal risk for bleeding, you can just do serial fast exams. I had 1 trauma patient in whom I put a little DPL catheter in the umbilicus, and we just aspirated it once a day to make sure we were not bleeding because he had a bad splenic injury.

**Mortality Rates Decreasing, but Cause Uncertain**

**Brain-Injured Patient:** The brain-injured patient is a high-risk patient population. Many of our TBI patients are elderly patients on oral anticoagulants to start with, and in those patients we are reversing their anticoagulation. There are less data, and there is clearly risk, in part, because on ECMO you lower your platelet count and fibrinogen levels. The challenge is getting repeat CT scans on those patients. So, though brain injury is a relative contraindication, it is no longer an absolute contraindication. Every case is very different. In all the studies done to date on ECMO, there is still is about a 5% to 10% spontaneous intracranial bleed rate with anticoagulation for ECMO, so it is not trivial.

**Patients With Severe ARDS:** If you look at outcomes by mortality — 30-day mortality, 90-day mortality, 1-year mortality, over time, over decades — mortality rates are decreasing. As to whether this reduction in mortality is related to these rescue strategies or really related to other things that are very
difficult to capture — such as not such aggressive fluid resuscitation, as we were doing in the past; more appropriate antibiotic management because many of these patients have pneumonia; better sepsis management — it is very hard to tell. Overall, we can say that mortality is significantly reduced over the decades, but the cause is very challenging to discern. Prevention has come a long way as well.

TRALI and TACO Important Area Still Underreported

TRALI: About a decade ago, transfusion-related acute lung injury (TRALI) was better defined, although I will remind you that we just got rid of the term acute lung injury in the new ARDS definition. Basically, TRALI is ARDS temporarily related to blood transfusion. It has to be within 6 hours of transfusion. TRALI is under recognized and underreported. It is the leading cause of death from transfusion complications, and the current incidence is estimated between 1:1,000 and 1:2,000. Again, many of us feel it is much, much higher.

TACO: TACO is a bit different, but in some ways similar. Remember that the ARDS definition has to have no evidence of congestive heart failure or ventricular failure. TACO is hydrostatic pulmonary edema due to a transfusion, so there is some degree of pulmonary edema related to cardiac dysfunction. It is the second leading cause of death related to transfusion reaction, and 1 of the biggest risk factors for TACO is that the patient is in positive fluid balance prior to receiving the blood transfusion. Basically, you have a patient who is already fluid resuscitated and now is getting a unit of blood, or 2 units of blood, or 2 units of FFP and they get hydrostatic pulmonary edema. An important issue for both of these is the rapidity with which blood is transfused.

Mandated Transfusion Time: Clearly, if somebody has hemorrhagic shock and is bleeding, you are going to give blood quickly, but many times if we are giving transfusion for just severe anemia, as is common in many patients prolonged in the ICU, we have now mandated those transfusions go in over 3 hours. In someone who is not hypotensive, blood cannot be pushed in. It essentially has to be hung by gravity, and that is easy to understand for everybody. Sometimes, it takes educational effort at explaining why we need to have it go in by gravity over 3 hours. We actually write that as part order now. When we are writing a unit of blood for anemia, we write over how much time to make certain it takes.

Treatment Approach for PE

As our imaging gets better, we are now seeing segmental PEs or subsegmental PEs. Many of the patients have minimal to no hypoxemia.

Case 1: A single segmental PE in 1 of the lungs was done because the patient had some shortness of breath and tachycardia, and you now have a CT that shows this 1 small segmental PE.

Approach: As soon as we get a CT imaging report that says PE, patients get anticoagulated as long as they do not have a contraindication to anticoagulation. It is pretty much that simplistic. I am not certain those patients really need anticoagulation for 6 months, and we do not really have any studies to guide us.

Case 2: This time, the CT shows 1 small subsegmental PE.

Approach: As soon as the report basically says PE, they get anticoagulated unless they have a contraindication.

Concern: Anticoagulation is not benign, and the risk of major bleeding is significant. Yet, most physicians fall on the side of “but they have a PE and it can become bigger. If there is not a contraindication to anticoagulation, we should move forward.”

Length of Treatment: The vast majority of us would still anticoagulate for 6 months. There are not good data to guide, but as soon as that report says PE of any type, most clinicians are going with the traditional 6 months. I wish there was some new information, but I think most people are concerned more that there are still risk factors to all these patients even though they are low risk.
What Happens to PE Patients?

We have a series where 25% of our initial CT scans showed pulmonary emboli in asymptomatic patients in our trauma population, so they are not hypoxic. CT scan with our 64 slicer shows clot in the lung, and the first response is to anticoagulate. It would be a great longitudinal study to do. In particular, if there are patients that have contraindications to anticoagulation, it would be informative to follow those patients to see what happens over time. There are also new devices or approaches.

Case: A patient is ready for discharge, gets up in bed to transfer to the wheelchair to be wheeled out of the hospital, and collapses on the floor. He has a massive PE.

Approach: Recently, 1 of our colorectal surgeons had done a laparoscopic colostomy for a patient who had a colovesical fistula. When he got home, he collapsed in his bathroom. When they got him in, he was in full arrest for a while. They got him back and had severe hypoxemia, and clearly had a massive PE. Another recent incident was a patient who had no surgery. We do the traditional immediate hyper and anticoagulation, as long as there is not a contraindication. The question will then come forward, very quickly, of whether we should do systemic thrombolysis. This is quick, easy, and clearly many studies have shown to reduce the death rate by about 50%. So, in patients who have had surgery, even if surgery is within a recent time frame, we will consider thrombolysis, and we will usually give half dose. So, this is systemic thrombolysis, not local. In the patient I just described with the laparoscopic colostomy, we gave him half dose of thrombolytic therapy because, again, it is immediate and can be done in the ED. We recognize there are bleeding complications related to that. He did have a bleeding complication that we were able to take care of, so that is the next step for us.

New Catheter Is Multi-Functional

The third step is considered for percutaneous catheter embolectomy and percutaneous catheter thrombolysis. In the gentleman I just described, per newer protocol, rather than just catheter embolectomy, they did embolectomy and thrombolysis. The catheter placed for thrombolysis also does ultrasonic disintegration, and it basically accelerates the rate of thrombolysis because it allows the thrombolytic agent to better penetrate the embolus. This is a special catheter that does this ultrasonic disintegration and can deliver the thrombolytic. If a patient can wait that long and go to Interventional Radiology directly from the ED, this is our approach. If the patient arrested in the ED, has a massive PE, and has both hypoxemia and right heart failure, he is a patient I would get called to cannulate for ECMO. If the patients are in severe shock, they will get on VA ECMO. If they just have severe hypoxemia, they will get VV ECMO. And then everybody can take a breath. The cardiac surgeon can decide whether we should do a catheter embolectomy or a surgical embolectomy. Many times, once they are on ECMO for 5 to 7 days and we redo the pulmonary angiogram, the clot is already gone. We are fortunate we have that modality we can bring to the bedside, and it allows everyone to not have to do emergent cardiac surgery, in particular.

Ventilator-Associated Pneumonia: Definition Still Not Clear

The patients on prolonged mechanical ventilation are clearly the patients that in all studies are shown to be high risk. When we look across our institution, the patient populations in that cohort are trauma and burn patients in our Surgical ICUs because they have long lengths of intubation. In the Medical ICU, the patient population is those with COPD. The CDC did a very good thing in changing to a surveillance definition of VAC and IVAC because it is objective, but it has really made VAP monitoring problematic. Now we have great objective surveillance definitions, but we have no idea how they relate to ventilator-associated pneumonia. So, I think this is a very challenging area, and we still do not have a
good definition for VAP. It is still a problem area. Another thing the CDC definitions have done is decreased the furor in the quality department of our hospital about the VAP patients because it is hard to identify them now. Every month in our ICU QA meetings, we look at our VAC, IVAC, VAP rates, and nobody can make any sense out of them, and the benchmarks are poor because most of the earlier benchmarks that we have are medical ICUs.

Three-Phase Strategy for VAP Prevention

Endotracheal tubes are impregnated with silver or they have these special suction ports. Obviously, there are proponents and there are opponents. We have used a 3-phase strategy for VAP prevention in our adult ICUs. In terms of the specialized tube, we currently use the continuous aspiration of subglottic secretions (CASS) tubes. So, there are multiple studies in meta-analyses that support the CASS tubes prevent VAP in the prolonged ventilator population — so, in patients who are intubated for greater than 7 days. The hard thing is trying to figure who should get those tubes because we are not very good at predicting who is going to be on the ventilator for how long. This is a challenging issue. In ARDS patient population, we find a lot of patients come to us with CASS tubes in place. I still have concern about how we use the continuous suction on those tubes. In 1 of our ECMO patients, we had a severe bleeding complication related to an ulceration that occurred at that site of the posterior subglottic suction port. We do not use the silver impregnated tubes because, in the studies that have been done, the effect size is small and the cost is high. We never change the tube if they had a standardized tube. For patients that get an emergent intubation such as one who had a respiratory arrest on the floor, they are all going to get CASS tubes. Patients that go to the Operating Room for an elective surgery do not get them. That is pretty much how we have standardized it. The second strategy we use is chlorhexidine in all intubated patients, and the third strategy is head of the bed elevated, which nobody is quite sure if it works or not.

Diagnosing and Treating VAP

Pathogens: The national evidence of surgical and medical patients is still that staph aureus and pseudomonas the 2 most common pathogens, but in any specific unit, that is all local. We have a separate Trauma/Burn ICU and Surgical ICU, and the microbiology of VAP in each of those units is quite different. I would say in our institution in general, we are seeing many more gram-negative VAPs than gram-positives. So, our microbiology has changed over the last couple of decades. Pseudomonas and enterobacter are our most problematic pathogens. Local politics does rule. For us, our Surgical ICU is our ARDS ICU as well, so many of those patients are medical patients. Pathogen prominence is local within an institution and within the country in general.

VAP Diagnosis: We favor bronchoalveolar lavage (BAL) diagnostics. We manage to use either a bronchoscopic BAL where we do a formal bronchoscopy if we want to sample both sides or in particular if the patient has a left lower lobe infiltrate. We have standardized our approaches to how we do that, but the Fellows and Residents are very used to doing BALs very quickly, and our respiratory therapists are very use to getting a bronch cart quickly available for that. We use a step off of >10,000 organisms as the final indication to continue antimicrobial management.

Mini-BAL: For patients in whom we are not so worried we need to sample both sides or they only have a right lower lobe infiltrate and if we are really busy, we do not feel like we need to do a bronchoscopic BAL, we will do a mini-BAL. We have taught our respiratory therapists to do a mini-BAL. It is quick to do because it is a single lumen catheter, it goes down the right side and samples. This is pretty much always the process. We never get sputum cultures. We never get any other kind of cultures.
New Guidelines for HAP/VAP

The new 2016 HAP/VAP guidelines from Multisociety from IDSA, ATS, SHEA, SCCM, will be presented at the upcoming ATS meeting and published shortly. I was a part of the guideline panel. It will come out in CID initially, but this guideline is quite a bit different than the prior HAP/VAP guideline in 2 ways. First, it uses the standardized grade approach, which was not used in the prior guideline, and uses a PICO question (population (P), intervention (I), comparison (C) and outcome(s) (O. For instance, one of the questions is this: What diagnostic strategy should you use to diagnose VAP? You know the answer is that there really is no evidence that BAL or mini-BAL is better than a quantitative sputum culture. It is very important in that it is a very different guideline than in the past and hopefully will be of better use to clinicians at the bedside. There are 2 sections in the guidelines. One is the empiric treatment of HAP/VAP, if you do not know the bacteria. And then there are specific questions for specific pathogens: What antibiotics should be used for MRSA HAP/VAP? What antibiotics should be use for pseudomonas HAP/VAP? Then, the evidence is delineated below it. I think the new guidelines will be of greater use to clinicians at the bedside in care of the patient who has a pseudomonas HAP/VAP or a MRSA HAP/VAP. We will be interested to hear the controversy that always comes out of new guideline presentations.

Ventilation Weaning Techniques

After successfully treating all these significantly ill patients with respiratory problems, there is 1 more step — getting them off of mechanical ventilation, weaning, liberating. I will go from bi-level ventilation and go directly to pressure support and just let them breathe. As soon as I start to see a little bit of spontaneous ventilation effort, I immediately make them change over.

Use of Tracheostomy Not Obsolete

With these new endotracheal tubes and all the other interventions we plan to do, a tracheostomy is still definitely not obsolete. A trach is a wonderful airway for a number of patients. Now, we probably have the best data that we have ever had as to when to do it and how to do it. We have much better randomized trials to guide our management. I put patients into 2 camps. 

Early Trach Patients: There are the patients who need trachs early. Those are the patients that — particularly in trauma and burn — are really on the ventilators solely because they need an airway. Patients with TBI, patients with significant burn inhalation injuries, patients with neurologic disease of some sort — those are patients that get trachs early. They really do not need the ventilator, so you can liberate them from the ventilator. We do not have enough studies in that patient cohort to really define exactly when, but normally once they are not on high FIO2 or high PEEP, we move to early trach.

Late Trach Patients: Then there is the other camp. They really need the ventilator, and then the question becomes can we really predict how long a patient will need mechanical ventilation? So, the best trial we have to guide our management is the TracMan trial, and this guides my management of when I do a trach in the rest of the patients who require mechanical ventilation. I usually wait about 10 days. The TracMan trial was a >900-patient trial done in the UK, and they randomized patients to early trach (within 4 days) or late trach (after 10 days). The most important finding of that study was that in the >10-day group, the late trach group, only 45% of patients required tracheostomy. By 10 days, 55% of patients had been extubated. So, I wait 10 days because our endotracheal tubes are better, the balloons are low volume, low pressure and the patients sort themselves out. They are either extubated or they are going to be long-term ventilation, and then they are going need a trach. The TracMan trial has shown us that clinicians are not able to determine who will be extubated. Flexibility is needed here.
In all of the newer randomized controlled trials that have been done about tracheostomy, initially there was the thought there was going to be a signal for a lower rate of ventilator-associated pneumonia. This did not occur. In the TracMan trial, they did not even measure VAP; they just measured antibiotic use. So, there are really no data to push us to an earlier trach for VAP prevention.

**Percutaneous vs. Open Tracheostomies**

We do both percutaneous and open tracheostomies based on the patient.

**Percutaneous:** For trauma and burn patients, percutaneous trach is the routine, as long as their anatomy is favorable. We just had an elderly trauma patient who had multi-system injuries who had his inferior cricoid cartilage just below his sternum. And so, even with his head extended, I could not find a safe place to put a needle, so we did an open and pulled his trachea up a bit with a trach hook.

**Open:** But we have a whole other covert of ARDS patients, many of whom are on anticoagulation, and many of them are also on high PEEP. They are occluding the airway with a dilator, which is very problematic. In these patients, we do an open trach, but we do them at the bedside as well. The other patient population I have a little concern about occluding for any period of time with a dilator are the severe TBI patients — meaning $PO_2$ will drop, $PCO_2$ will rise — and whether or not the patients can tolerate that has to be considered.

**Common Terms Redefined**

There have been some recent articles about definitions of sepsis. In the new definitions, the comment has come forward that systemic inflammatory response syndrome (SIRS) is dead. There have been pictures on the Internet of a gravestone with SIRS on it.

**Previous Definitions:** To review, the way we defined sepsis in the past was 2 part: The patient had to have 2 or more SIRS criteria — temperature, heart rate, respirations, and white cell count — and then there had to be concern for either a presumed or a confirmed infectious process. Then we had the 2 other categories of severe sepsis and septic shock. Severe sepsis was defined as sepsis with 1 or more organ failure, and septic shock was defined as patients who required vasopressors. It was pretty simplistic.

**New Definitions:** The study that has moved forward in redefining sepsis was a study done by the Australian/New Zealand Critical Care Trials group with over 100,000 patients. They identified that, of all of their severe sepsis patients, 12% did not have SIRS. So, there was concern that SIRS would miss 1:8 patients. They published a wonderful article in *The New England Journal of Medicine* by Kaukonen and colleagues and reviewed that SIRS will not pick up all patients with sepsis. On the other hand, they did show evidence that the higher the SIRS score, the higher the mortality rate in the patients that did have SIRS — sort of some confounding data, right? So, a certain population did have it, but of those who did have SIRS, the higher the SIRS score, the higher the mortality rate. Suffice it to say that, after reviewing all of the data, the definitions were changed by our leaders in critical care, and now SIRS is no longer. And there are only 2 definitions: the definition for sepsis is life-threatening organ dysfunction with suspected or documented infection; organ dysfunction is defined by using a SOFA score, and the true definition that is proposed is an acute increase of 2 or more SOFA points. My problem with this definition is I do not keep this SOFA score in my brain. It is a big table of multiple organ systems with different scores for each organ system dysfunction, and it is quite complicated. It is very difficult to actually do. What has persisted is the definition for septic shock, and that is sepsis with persistent hypotension requiring vasopressors.
Criteria of Quick SOFA Score

The SOFA score is the organ failure assessment score used to define severity of illness in patients in the ICU. It includes respiratory, coagulation, liver, cardiovascular, renal, etc., but it is not a score that you do quickly at the bedside. Because we do want to be able to define sepsis at the bedside when evaluating a patient, the leaders came up with the qSOFA score, a quick SOFA score.

Criteria: The qSOFA score has 3 criteria. One is altered mental status with a GCS ≤13. The second is tachypnea, a fast respiratory rate, a respiratory rate >22. And the third is a low blood pressure, a systolic pressure of <100. If you have 2 or more of these qSOFA points, that predicts sepsis similar to a full SOFA score. But this is all done in medical patients. So, if I walk around my Surgical or Trauma or Burn ICU, this is just like SIRS. All my patients have this because they are on pain medication. They have pain, so they are tachypneic. They are a little altered in their mental status, not just from sepsis but in part because of narcotics for pain control. So, to me, qSOFA has not yet been validated in the surgical patient population or in the trauma or burn patient population. I think it is a little bit of a slippery slope, and I do not think SIRS is dead. The 12% of patients that do not have SIRS and have sepsis is an interesting group. That patient population probably has altered inflammatory response, and that is probably all it means. So, I would just ask everyone who looks at the new sepsis definitions to consider them carefully and determine how they apply to your patient population. I am concerned in that even in our very advanced critical care system, I do not have an electronic way to measure SOFA. SOFA is very complicated.

New Sepsis Guidelines Being Challenged

Significant change is taking place in the guidelines for surviving sepsis. In particular, there has been a significant change in the sepsis bundle, which is really how we implement the guidelines. In April 2015 the sepsis bundles were updated in response to new evidence from the 3 large randomized, controlled trials.

Trials: The Protocolized Care for Early Septic Shock (ProCESS) trial in the United States compared the Rivers protocol, early goal-directed therapy to standardized care versus routine care. A group from Australia and New Zealand did the ARISE trial. They compared the Rivers early goal-directed therapy protocol to usual care. In the United Kingdom, researchers did the protocolized management in sepsis (ProMISe) trial, which was early goal-directed therapy versus usual care. All of these studies showed no difference between early goal-directed therapy with the Rivers protocol using ScvO₂ titration compared to usual care.

Guidelines: We were very pleased to see the change because it is based on very high-quality evidence. The 3-hour bundle did not change. It is still very basic: Measure a lactate, get cultures, start broad-spectrum antibiotics, and give fluid resuscitation. The change took place in the 6-hour bundle. It used to be as follows: Check CVP, look at ScvO₂, consider placement of a catheter to measure ScvO₂ continuously, and now all of that has been removed. The 6-hour bundle still includes use of vasopressors if needed, consideration of additional fluid resuscitation, and re-measuring lactate. Fluid resuscitation is now guided by a clinical examination. Additionally, we have moved to using bedside cardiovascular ultrasound, a bedside ultrasound assessment of cardiac function, IVC filling, IVC compressibility. A dynamic assessment of fluid responsiveness with passive leg raise or a fluid challenge is also used. This allows the clinician to provide fluid resuscitation in a more minute-by-minute, hour-by-hour manner not just to a normalization of ScvO₂ alone which I think has the same problems of trying to fully normalize lactate. Many times you end up with over resuscitation.

Clinical Interpretation: I worry a bit about the use of ultrasound in terms of clinician interpretation. It is sort of like the old pulmonary artery catheter. Really, the catheter was not the problem — it was how we as clinicians interpreted it. This is really true for ultrasound because not all physicians have the same skill in ultrasound as each other, and we have not standardized education in this realm.
Drugs Used for Patients at Risk of Drug Resistance

When considering multiple drug-resistant organisms, I think it is important to know about the patients who potentially have multidrug-resistant organisms and the risk factors. Most surgeons know patients who are exposed to antibiotics who have had prior infections, patients who have been in and out of the hospital, and patients who have had prior surgery are at a higher risk. When considering antimicrobial management, these are the patients who need more broad coverage for resistant organisms than the patient who just comes in from the community and is otherwise healthy and does not have risk factors for multidrug-resistant organisms. Many opt for extended beta lactamase producers as well as the carbapenem-resistant organisms, but we are using colistin more and more. It is an old drug that has come back to be used more frequently. I think what is really challenging is what to do empirically. Carbapenems have been used widely in many institutions, particularly for multidrug-resistant gram negatives, so they are not as useful as they have been in the past. Carbapenems have been a mainstay of therapy; when we identify carbapenem resistance, then we locally use meropenem here. Then we are going to move to a colistin strategy. We do not use tigecycline currently, in part related to our ID colleagues here who help us and assist us in management. They do not favor using multiple agents. I would say on occasion we move to tigecycline, but it is a rare occasion. We get the antimicrobial susceptibilities. We sit down with our ID colleagues and review what is best.

MRSA: We have learned to control methicillin-resistant staph aureus. We are having much less MRSA than in the past. I think a lot of our preventive strategies — particularly in the ICU — have worked, so we are seeing much less hospital-acquired MRSA. On the other hand, we still see MRSA that is community acquired. We just recently had a severe ARDS patient that had H1N1 viral pneumonia with MRSA super infection that was a necrotizing pneumonia that we had on ECMO. Again, though, that was community-acquired necrotizing MRSA pneumonia. We still see MRSA, but it is much less of an issue.

Standardizing Approach Lowers Central Line Infections

Our CLABSI rates are markedly lower than they used to be, particularly in the ICU. Every day, twice a day, we are talking about getting the lines out as a part of our daily goals. We keep a close track on our line days. We know that for central lines in IJ, subclavian, femoral position, there is an exponential increase in risk for CALBSI at day 7 and beyond. We try to get those lines out, and usually move to a PICC line, if the patient can get a PICC. It is just an alternate line. It still has a risk for CLABSI, so we try to get that line out as well. I think we are doing better. I think 1 area where we are clearly doing better is insertion. We are now separating our CLABSI into insertion-site related (within 72 hours of insertion) versus maintenance related, and the vast majority of our CLABSI are maintenance related and not insertion-site related, so we really have standardized our approach to placing central lines in a very sterile, aseptic way.

Urinary Catheters: Currently, we are not as successful at getting urinary catheters out as we are central lines. This is harder, particularly in patients who have epidurals. It is hard to get them out because, even when we get them out, they go into retention and end up putting them back in. In the very critically ill patient population, if we are diuresing, it is hard to get them out, so this is an area we are still working on.

New Guidelines Established for *Clostridium difficile*

*Clostridium difficile* infection is a big problem for surgical patients, even though we have learned much more how to treat it and our rates of surgery for it are much lower than they have been over the last couple of years.
**Treatment:** We do utilize the new guidelines. Most of our patients fall into the severe *C diff* category, so they are not going to get metronidazole, they are going to get enteral vancomycin. If they are going to get metronidazole, it is intravenously.

**Recurrence:** With the first recurrence, we usually do a vancomycin taper, a more prolonged treatment with enteral vancomycin knowing full well that once 1 recurrence occurs, the risk of having subsequent recurrences is very high. If there is a next recurrence, patients get what we call a fidaxomicin chaser — they have both a prolonged vancomycin taper plus fidaxomicin. There are some patients in whom we will use the fidaxomicin chaser up front in the first recurrence. It is usually the immunocompromised patient population, like a transplant patient. They will actually get a prolonged vancomycin taper and then a fidaxomicin chaser. This strategy has worked quite well for us and is better than moving to fecal transplant after the first, second, or third recurrence.

**Fecal Transplant:** The fecal transplant comes in later than that, and usually I am not involved at that level. Our ID colleagues run that service. The goal is not to have to go that route.

**Preventing *C diff***

I am intrigued by the article by Dr Gerding about repopulating the colon with friendly *C diff*. At the University of Michigan, there is a research group funded to do *C diff* research, and they are intrigued by the study as well. On the other hand, I worry a bit about long-term risks and how truly to define the differential organisms. For instance, when a patient comes in with *C diff*, we do not identify clinically whether or not a patient has the hyper producer of toxins, the NAP1 strain. We are not doing this clinically in our care of these patients. We are treating them all the same. I think we are having some significant breakthroughs in the prevention of *C diff* and are seeing it less. We really are trying to reduce our duration of antimicrobial therapy. I think many clinicians treat way too long. I just came back from giving oral boards. We would ask candidates how long they would treat for diverticulitis. The standard answer was always 2 weeks. The evidence for short-course therapy in intra-abdominal infection is quite clear, so we have been trying to do short-course therapy for infections where we have good source control, and I think that has really helped with reducing *C diff*. A lot of our early *C diff* in the hospital setting was poor infection control, and we are doing better there.

**New Candidiasis Guidelines Released**

Ted Pappas and colleagues did the 2016 update for candidiasis. In the past, the recommendation for candidiasis was fluconazole as a first-line therapy as long as the patient had not been exposed to prior fluconazole therapy. The new guideline is very clear, and it is a different recommendation — echinocandin is the first-line therapy. There is high-quality evidence to support using echinocandin initially — micafungin, anidulafungin, caspofungin. We have had to change our practice. We do not have a high incidence of fungal infections in our ICUs, but the underlying rationale is that you start strong to cover all organisms and then deescalate therapy once you have these susceptibilities. This is not a practice we have used in antifungal therapies. They are also recommending antifungal susceptibilities be performed which is a major change for most institutions. The recommendation is based on the evidence that we are seeing higher fluconazole-resistant candida organisms. In many ways, it is the strategy we are using for antibiotics, it is just something we have not done previously.