# Hospital Inpatient Quality Reporting (IQR) Program

## Support Contractor

# Fluids, Lactate, and Champions: An Emergency and Preventive Medicine Physician's Perspective on Sepsis and the SEP-1 Core Measure

## Presentation Transcript


## Speaker

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## October 7, 2020
## 2 p.m. ET

**Candace Jackson**: Good afternoon and welcome to *Fluid, Lactate, and Champions: An Emergency and Preventive Medicine Physician's Perspective on Sepsis and the SEP-1 Core Measure* webinar. My name is Candace Jackson, and I am the Hospital Inpatient Quality Reporting Program Support Contractor Lead from the Inpatient Value, Incentives, and Quality Reporting Outreach and Education Support Contractor. I will be hosting today's event. Before we begin, I would like to make a few announcements. This program is being recorded. A transcript of the presentation will be posted to the inpatient website, www.qualityreportingcenter.com, in the upcoming weeks. If you registered for this event, a reminder email and a link to the slide was sent out to your email a few hours ago. If you did not receive that email, you can download the slide at www.qualityreportingcenter.com. This webinar has been approved for one continuing education credit. If you would like to complete the survey after today's event, please stay on until the conclusion of today's event. After the question-and-answer session, we will display a link to the survey that you will need to complete to receive the continuing education credit. The survey will no longer automatically be available if you leave the event early. If you do need to leave prior to the conclusion of the event, a link to the survey will be available in the summary email sent out one to two business days after the event. If you have questions as we move through the webinar please, type your question into the Ask a Question window with the slide number associated, and we will answer as many questions as time allows. After the event if you have additional questions, please submit your question to our speaker at the email address that will be provided at the end of the presentation.

Our speaker for today's event is Dr. Bobby Redwood who is an emergency and preventive medicine physician and chief of emergency medicine at Cooley Dickinson Hospital.

We would like to make note that the viewpoints shared in this presentation are those of the presenter and do not necessarily represent CMS's views.

This slide provides a list of the acronyms that we will use throughout the presentation today

This event will provide a physician's perspective on the SEP-1 measure as it relates to population health and sepsis care in the emergency department.

At the conclusion of today's event, participants will be able to discuss various sepsis definitions, identify key benefits of the 30 milliliters per kilogram fluid bolus in severe sepsis and septic shock, identify the utility of serum lactate and screening for septic shock and guiding resuscitation efforts, and identify strategies for engaging a physician champion in sepsis quality improvement efforts.

I would now like to turn the presentation over to Dr. Redwood. Dr. Redwood, the floor is yours.

**Dr. Bobby Redwood:** Good day and thank you CMS for inviting me to present today. I also want to thank CMS for keeping me honest. You'll see a bunch of cartoons throughout this presentation. So, these are hand drawn. I realized that my Google image sources were not copyright-friendly. So, these are all original images today. So, thank you for putting up with my cartoons. As stated, I'm a preventive medicine physician and an emergency physician. Preventive medicine is the medical specialty of public health, and my personal motto is that every emergency is a failure of prevention. There's a lot of room to save lives and prevent severe disability and illness with the SEP-1 core measure, and I'm a firm believer in this measure. I am a practicing clinician. So, I work 10 shifts per month in the emergency department, but I'm also an administrator. I chair the sepsis committee at my hospital, and I work with the Wisconsin Hospital Association on statewide septic quality improvement projects. I have visited a hundred plus hospitals to look at each sepsis committee and sepsis work, and I know a lot of what goes on on the ground in terms of boots on the ground medicine. Now, I understand we have a mixed audience today. We have emergency physicians. We have PAs and MPs. We have clinical nurses as well as quality improvement professionals and administrators. I hope there's something for everyone in this presentation today. Now, as we get started, we should of course be sure that we're all on the same page. I wanted to start with definitions. This is a challenging time in medicine in

terms of a sepsis definition because the definition really is evolving. The 1991 consensus conference definition is the definition you see before you, and it is my preferred definition and the definition that we use in the emergency department and with the SEP-1 core measure. Now, one of our faults, I feel, as physicians is that sometimes we're a little bit too concrete in our thinking. Some people say, "Well, the world has moved on to SEP-3. Why are we still using the 1991 consensus conference definition?" I would say that the definition you use really depends on your clinical environment. So, in the emergency department or if you're screening for sepsis on the floor, we're really trying to catch the widest catchment of patients and cast a broad net to find occult sepsis, which even master clinicians miss. So, let's just get into the definition. It starts with SIRS criteria (Systemic Inflammatory Response Syndrome). If you have an elevated temperature or a low temperature, that is one criteria. If you have an elevated heart rate greater than 90 not greater than 100, if you have an elevated respiratory rate, or a PaCO2, less than 32, or if you have an elevated white blood count, or white blood count less than 4000, those would all count as SIRS criteria. I do want to point out the bandemia as well. If you have greater than 10 percent bands for the emergency physicians out there, that lab value sometimes comes back in the delayed fashion. Now ,if you have two SIRS criteria and a source of infection, you now have sepsis, and the sources of infection are the usual suspects. So, we're talking about UTI, or urinary tract infection, lower respiratory tract infection, or pneumonia, intra-abdominal infection, like cholecystitis or appendicitis, or a skin infection like cellulitis. Now, obviously there are many other sources of infection out there, and you can certainly have viral sepsis or fungal sepsis, but primarily those are the key players when we're looking for garden variety sepsis in the emergency department or screening on the floor. Now, severe sepsis is triggered when you have evidence of end organ damage. That can be altered mental status. That can be renal impairment. That can be chest pain or EKG changes. There really is a laundry list of end organ damage, and you finally progress to septic shock when you have evidence of hypotension. Now, one of the main reasons I like this definition is because it really makes sense with the pathophysiology.

If you think about having an infection, let's say a cellulitis, you may notice some redness or warmth on your arm, an early cellulitis, but you don't have a fever. You don't have an elevated heart rate. As that infection feeds into the surrounding tissue, gets deep into your body, and spreads to your bloodstream, you now have bacteremia. You're going to start to see SIRS criteria, maybe a fever, maybe an elevated white blood cell count. You've now progressed to true sepsis. After a while, you start getting rigors, chill, maybe your mental static weight, maybe your renal function decreases. This is capillary leak syndrome. This is third spacing of fluid. These are the marks of severe sepsis where our immune system is kicked into high gear, and our body is responding. Then, sometimes our body over responds. We actually vasodilate to the point of septic shock where our blood pressure drops, and our heart rate can no longer sustain the blood pressure needed to keep our bodies going. This is, of course, concerning. This is, of course, a disease of very high mortality, and this is the reason we are all here today. In terms of screening for sepsis, this definition makes a lot of sense, and it's very useful in the setting of the emergency department where you're screening patients for occult sepsis.

The SEP-1 core measure is obviously complex, but these are the critical elements of the three-hour bundle. Within three hours we are measuring a lactate level. We're obtaining two blood cultures prior to the administration of antibiotics, and we're administering broad spectrum antibiotics. If the patient has septic shock, we are starting a 30 milliliter per kilogram crystalloid bolus, and I like to use the shorthand 30 by 3. It's very useful with your physician-nurse dyad to have shorthand because it's hard to say 30 milliliters per kilogram crystalloid bullet. It's very easy to say 30 by 3. So, I'll continue to use that throughout this discussion, and, if you're not using similar verbiage in your hospital, I'd encourage you to do so.

In a streamlined health system, the patient won't be in the emergency department more than three hours. If you have relatively reasonable volumes and smooth processes, that patient will already be up to the floor or ICU, but, obviously, we acknowledge reality, and many emergency departments are boarding patients for a long time.

So, you may well have the patient in your ED for more than six hours. With the six-hour bundle, the critical elements are in the case of severe sepsis, re-measuring lactate if the initial lactate is elevated greater than two, and then, in the incidence of septic shock, remember that as hypotension refractory to fluid resuscitation, we are going to apply vasopressor medications and reassess fluid status. This is goal therapy. So, we are applying vasopressors with a goal of maintaining a mean arterial pressure greater than or equal to 65 millimeters of mercury.

Of course, the definition is evolving, and we as clinicians are trapped between two definitions. This is a busy table. I don't expect everyone to memorize this or even process it during this presentation, but I want you to have it as a reference. It essentially contrasts the definitions old and new. The Sepsis-3 definition is basically based on sequential organ failure assessment, or the SOFA score, and it eliminates the category of severe sepsis. It creates two categories of sepsis: regular sepsis and septic shock. The goal of the Sepsis-3 definition is to sort patients into those requiring ICU-level resources. So, in my mind, these two definitions can actually coexist quite well. If you are screening for sepsis, for example, if you're in an emergency department setting, or if you're screening for sepsis on the hospital floor, you would probably prefer to use the 1991 consensus definition which is based on SIRS criteria and designed to cast a wide net. If you're actually deciding which patients be admitted to the ICU or which patients require, for example, a central line or intubation, you might be using the Sepsis-3 definition which is based on the acuity of the patient and the level of resources they're going to need. If you're using the 1991 definition, look at the bottom of your table here where we talk about mortality ratios. You are in general going to find that your sepsis has lower acuity because you are casting a wider net and including more patients in this, and, indeed, when I treat patients with sepsis in the ED, based on the 1991 consensus definition, many of those patients never make it to the ICU. I fluid resuscitate them. I start early antibiotics, and I find that I'm able to reverse the SIRS criteria and that the patient actually improves on the floor significantly, are weaned off IV antibiotics, and are able to be discharged home on pills.

For those patients that meet high Sepis-3 definition criteria, meaning a high SOFA score, those patients are obviously going to have a higher acuity and a higher mortality rate because of selection bias, intentional selection bias, we've selected a thicker cohort. Now, every year, the Surviving Sepsis campaign continues to give us further guidance on which definition to use and how to use these definitions. I think this is an area where we have to accept the uncertainty, and we have to understand that the definition we use may be different depending on our clinical environment. As clinicians, I would ask you not to be too black and white about this. This is a gray area that's evolving in medical literature, and within five years or so, I think we'll have significant clarity on this issue and won't even be talking about the various definitions anymore.

So, what's in a name? Right? At the end of the day, you're a practicing clinician. You are in the seat in the ED looking at patients in front of you. In my hospital, we have 36 beds. We see 100 patients a day. We admit 20 percent of those patients, 20 patients a day, and when I am on clinically my goal is to identify the sickest patients in my emergency department and to provide them the care they need. What I love about the SEP-1 core measure is that it really helps me snap into critical care mode in a patient that previously we hadn't been snapping into critical care mode so aggressively. So, the old paradigm, the old stereotype, would be an elderly female comes in from a nursing home. They are found to have slightly altered mental status. It's presumed to be a urinary tract infection because that's so common, and the patient is parked in the corner. It's always bed 26. They're parked in bed 26 with a CBC pending, a BMP pending, and a urinalysis pending. Now, an hour goes by. The CBC and BMP look normal. Two hours go by. Three hours go by. You're about to sign out of your shift, and you realize that the patient's urinalysis hasn't come back yet. Am I admitting this patient? Am I discharging them? What's going on? You ask for a straight catch urinalysis sample, and it's dry. No urine comes out. You realize this patient is probably dehydrated. Lo and behold, they do have a low grade tachycardia of 110, and you start to maintenance fluid to see if you can improve the patient volume status and get a urine sample.

It's only another four hours later that that patient actually drops their blood pressures. The nurse comes in. They're now 50s over 30s. Lo and behold, this was early sepsis. Now, it's septic shock, and you missed the diagnosis early on because the patient wasn't showing you that usual Systemic Inflammatory Response Syndrome. Now, that's the old paradigm. In the new paradigm, that patient would come in, and we would acknowledge their altered mental status, their low-grade tachycardia. We would draw blood cultures times two. We would find that that patient's lactate is actually elevated, and we would have caught occult sepsis early on in the disease course. We can now administer a 30 x 3 fluid bolus. The patient starts making urine. We issue broad spectrum antibiotics, and that patient is now on the floor, and they never reach septic shock because we were able to change the course of the disease that early. Now, when I use the SEP-1 or 1991 consensus definition of sepsis in the emergency department, what I'm really looking for are, "Who are the sick patients?" The evidence shows us that if you meet sepsis criteria with a lactate greater than four, you have a 30 percent risk of in-hospital mortality, easily the sickest patient in my emergency department on a given day. If you meet sepsis criteria with a low blood pressure, we're now talking about a 36.7 percent mortality. Finally, if you meet tested criteria and you have a low blood pressure and a lactate greater than four, we are approaching 50 percent mortality. These are some of the sickest patients in our emergency department, arguably sicker than patients who have experienced trauma, stroke, or ST elevation MI. So, we need to staff it and snap into critical care mode. Doing so requires that we recognize early that sepsis could be on the differential diagnosis. Now, obviously not everyone who meets SIRS criteria is septic. A panic attack might come in with SIRS criteria. Diabetic ketoacidosis might come in with SIRS criteria. An asthma attack might come in with SIRS criteria. Just because someone meets those criteria doesn't mean we have to apply the SEP-1 core measure. That would be true cookbook medicine. Clinical is still very important here, but we can obviously apply our brains, apply our critical thinking, and sort patients appropriately, and if you're on the fence, if you're not sure if sepsis is present or not, I would recommend airing on the side of treatment for sepsis as outlined by the SEP-1 core measure.

Now, there is obviously controversy here. If any of you have worked on a sepsis committee, if any of you are a practicing emergency physician, you have probably heard the argument that we are harming patients with the 30 by 3 fluid bolus. Am I seeing some nods in the audience? You have probably heard that we are flooding CHF patients or congestive heart failure patients' lungs with fluid and causing iatrogenic harm. You have probably heard that End Stage Renal Disease patients are getting fluid overloaded and that we are causing harm. You probably have encountered a vocal physician who is against the 30 by 3 fluid bolus. Let's bust some myths.

So, I'd like to get right into the controversy. Here, the controversy came from the Promise, Process, & Arise Trials of 2015. Now, early goal-directed therapy (EGDT) has been around since Rivers in 1999 and 2001. In general, early goal-directed therapy refers to aggressive fluid resuscitation with goals of resuscitation like mean arterial pressure or clearance of lactate. That phrase has obviously evolved over the years, but the Surviving Sepsis campaign strongly recommends that we do aggressive fluid resuscitation in the instance of sepsis despite co-morbidity. Now, the Promise, Process, & Arise Trials of 2015 found something different. They said early goal-directed therapy is not superior to usual care for ED patients with septic shock, and if you're on a sepsis committee, you're going to find vocal physicians who hold up these trials and say, "Look at this. This is not evidence-based medicine. Why are we doing it?" Well, the answer is the literature has really evolved since then, and the most cutting-edge literature is that which is provided by the Surviving Sepsis campaign and with which the SEP-1 core measure is based on. More importantly, we are talking about population health intervention. The CMS Innovation Center really are doing something brand new, something we haven't done in medicine. They are doing large-based, population-based trials of early goal-directed therapy. The Promise, Process, & Arise Trials were all done in academic medical centers. It is well known that academic medical centers have lower sepsis mortality than community hospitals, and this is because the experts on sepsis work in these environments. This is because these are high resource environments.

This is because the place where these trials are occurring are already providing state-of-the-art sepsis care. Remember that stereotype I talked before, the patient in bed 26 who's parked in the corner? That is the paradigm we're trying to change, and that is not happening in our nation's academic centers. When these three trials were done, they had already gained much of the gains they were going to make from early goal-directive therapy. They had selection bias in terms of the expertise in their hospital. We were trying to do early goal-directed therapy in the centers that were already doing maximal sepsis care. If your sepsis mortality is only 10 percent in your hospital, bringing it down to 9 percent is going to be hard to show the statistical significance. If you're a community hospital and your sepsis mortality is 30 percent, there's a lot of gain to be had there. So, we've had some rebuttals since the early Promise, Process, & Arise Trials. For example, Ding et al in 2018 analyzed the data of the three trials. They actually re-analyzed the trial, and they found, when the three trials were analyzed together, the data were more powerful and showed a lower risk of serious adverse events for early goal-directed therapy group compared to the usual care group. Now, if we look at Jaehne in 2018 they coined a new phrase. I love this phrase. It's "physician hydrophobia," the concept of an unrealistic fear of early goal- directed therapy or the 30 by 3 fluid bolus. They concluded that hydrophobia is unwarranted in early sepsis care and that recent early goal- directed therapy/septic shock trials used similar amounts of resuscitation fluids in the acute phase when compared to the original early goal-directed therapy trial and that this was associated with all-time lows in mortality. Then, Ding et al in 2018 actually came up with the meta-analysis. So, the meta-analysis is the gold standard. This is a summary of 16 different trials, over 6 000 patients. They found that early goal-directed therapy reduces mortality in adult patients with severe sepsis and septic shock and that lactate guided therapy may result in an even greater mortality benefit. So, if you're on the sepsis committee and you find the Promise, Process, & Arise Trials coming up, be sure to remind your clinician of the more recent evidence.

Now, even more sophisticated, we now have literature specifically about the 30 by 3 fluid bolus in CHF patients in the SEP-1 core measure. The literature in recent years is literally measuring the 30 by 3 impact in patients with CHS. So, let's see what it finds. Leisman et al in 2016 found that early initiation of IV fluids improved survival and severe sepsis or septic shock, and that patients with IV fluids within 30 minutes had a mortality of 13.3 percent compared to 18 percent in patients receiving IV fluids later. Interestingly, the patients who received fluids earlier in the study were actually the thicker cohort of patients, and they did better with the early IV fluids. Now, Rourke et al in 2019 analyzed over 1000 septic patients with 229 patients who had CHS. They found decreased mortality in the 30 by 3 cohort with no significant increase in iatrogenic pulmonary edema and actually found longer length of stays in the hospital if the 30 by 3 was not given. Now, I love this part of the study because they actually got into our heads a little bit. They said that physicians were reluctant to give 30 by 3 because of a CHF label on the chart not because of exam finding, not because they looked at the chest x-ray and saw pulmonary edema, not because they looked back and thought the last ejection fraction on the echo was 15 percent. This is a label of CHF on the chart. Now anyone who works in the emergency department knows that when a medically complex patient comes in they often have 20 plus diagnoses, and it's very common to see CHF, or congestive heart failure, on the chart. Well, we diagnosed CHF very early. We are screening for this in clinic. We are doing echoes on patients, and sometimes patients will have a diagnosis of CHF when they actually have an ejection fraction of 65 percent, a normal ejection fraction. Very often, we'll find patients who had a compromised echo two years ago where they had an ejection fraction of 55 percent, and now they are on ionotropic therapy, and their ejection fraction is back up to normal. These patients can absolutely tolerate a 30 by 3 fluid bolus, and a simple label of CHF on the chart should not dissuade us from aggressively hydrating patients with sepsis. Now, if we go on to Duttuluri et al in 2016, they also analyzed over 1000 septic patients, a third of which had CHS.

They found that in patients with pre-existing CHF presenting with severe sepsis or septic shock and hypotension inadequate fluid resuscitation, meaning less than 30 by 3, actually increase in hospital mortality and intubation rates. Now, isn't this fascinating. They had increased intubation rates in the patients who were not fluid resuscitated. This makes perfect sense to me. The argument against the 30 by 3 fluid bolus is that we in medicine should do no harm, and that this has the potential to cause harm. Well, in modern medicine, we actually do cause harm. If you think about chemotherapy, for example, we are intentionally giving poison to the body in order to kill the cancer and win the war against cancer. You know, kill the tumor cells while also killing healthy cells in order to win the war against cancer. If you think about sepsis, we want those antibiotics to get to every corner, every nook and cranny, of the human body. How are those antibiotics going to get to the brain, to the kidneys, to the heart, if those organs are not being perfused? We have to support the cardiovascular system before those antibiotics can take effect and that, sometimes in rare instances, may actually involve flooding the patient. It may actually involve iatrogenic pulmonary edema. Well, in the emergency department, we're very used to treating pulmonary edema. The cure for pulmonary edema is bypass or intubation and, spoiler alert, when these patients get admitted to the ICU, think about the patient on day three, four, or five of their hospital stay. They will routinely be seven liters up on their dry weights, 10 liters up on their dry weights, 15 liters up on their dry weights. The three, four, or five liters of crystalloid given in the emergency department is really a drop in the bucket compared to the overall fluid that the patients are going to receive during their hospital stay

At the end of the day, your patient may well need more than 30 by 3, and we would like to use defined endpoints to guide this fluid reception. My preferred endpoints are a mean arterial pressure of greater than 55, normalization of lactate or urine output greater than 0.5 cc per kilogram per hour. Now in CHF and End Stage Renal Disease, we should prioritize fluid resuscitation over respiratory status. I know that might sound difficult for some of us, but this is really hydrophobia when we are withholding fluids because of fear of iatrogenic pulmonary edema.

A estrogenic fluid overload is extremely uncommon in severe sepsis, and the argument that we could cause harm really forgets that we can reassess our patients. So, if you are concerned about a atherogenic pulmonary edema, reassess your patient; 500 ccs, listen to their lungs; 500 ccs, recheck a chest x-ray; 500 ccs, evaluate for hypoxia. We should not park the patient in the corner with three liters of fluid hanging and not check on them. We should check on them frequently and make sure that we are not causing harm. In my experience, iatrogenic pulmonary edema is quite rare. Finally, we should get past the idea that we're saving an intubation by not giving these patients adequate fluid resuscitation. We are really delaying intubations until when they get to the ICU and they are given adequate fluid resuscitation. So, it is our job to do good by our patients and to do good by our ICU colleagues as well and really administer the 30 by 3 fluid bolus despite comorbidities. As I said, we can always play the worst-case scenario game. Let's say you do initiate isogenic pulmonary edema. Let's say the patient does experience fluid overload. The cure for that is bypass emergent dialysis or intubation, all of which are routine in the emergency setting.

Now, of course, there are exceptions. So, who should not get the 30 by 3? In general, it's very few people, but let's go there. The main one, really the one that I want to spend the most time on, is patients who are not truly septic. We are not automatons. We should not apply the SEP-1 core measure to any patient who meets SIRS criteria, and we should not give 30 by 3 to patients who are having a panic attack, an asthma attack, etc. So, you've got to be confident that your diagnosis of sepsis is real. For example, if a patient comes in with altered mental status and an elevated lactate, I might initially think they have sepsis. When the patient's been there for half an hour, their mental status clears entirely. I learned that are a patient with a chronic seizure disorder. They've been off their meds for three days, and they almost certainly had a seizure. This was actually a postictal state and an elevated lactate secondary seizure. At that point, I would stop the 30 by 3 fluid bolus. I would stop the sepsis measures, and I would acknowledge that I had initially misdiagnosed the patient and continue to treat for seizure.

We should, of course, apply Clinical Gestalt, and we could potentially do harm if we were to give thirty by 3 to any patient who comes in without applying critical thinking. I don't think anyone's doing that, but it's worth noting. An accurate diagnosis is worth its weight in gold. Now, another exception are patients who already have clinical pulmonary edema. So, let's say you have true sepsis, but there's a patient with an EF of 15 percent. You listen to their lungs, and they already have fluid overload. You get a chest x-ray, and they have bilateral pulmonary infiltrate. This is a tricky situation. This is the patient who actually may be intravascularly depleted, and you might need to do something like get your ultrasound. Look at their inferior vena cava to assess their fluid status. Now, sometimes patients who already have pulmonary edema need the 30 by 3 fluid bolus. We again we have to support the cardiovascular system, and these patients may well be intubated or beyond bypass, but this is the kind of territory where you want to start low, go slow, and reassess frequently. We are now in the era of COVID-19, and the Surviving Sepsis campaign has recommended a fluid restrictive strategy for patients suffering from COVID-19. There is nuance here of course; 20 percent of patients with COVID 19 will have a secondary bacterial infection. So, it is absolutely possible to have COVID-19 and superimposed bacterial sepsis on top of that. Then, the COVID crash, as they say, typically comes after seven days. So, patients may have been [inaudible] with large insensible losses for up to a week before they arrive in your emergency department. Although, in general, we are using a fluid restrictive strategy in the context of COVID, you may have patients who are profoundly dehydrated and actually need aggressive fluid resuscitation. That is an area where the literature is evolving, and clinical practice is evolving. We will want to reassess frequently and see how the patient is doing and gauge fluid resuscitation based on what we believe their volume status is based on all the evidence we have available to us. If a patient has comfort care, if they have opted to allow natural death, that patient does not necessarily need the 30 by 3, and then obviously, if a patient refuses care, we do not force care on our patients, but we should engage and share decision making and let the patient know that the 30 by 3 is the gold standard for resuscitation and sepsis.

Finally, just to reiterate, the patients who are not exceptions, the patients who should certainly receive the 30 by 3, are CHF patients with sepsis, End Stage Renal Disease patients with sepsis, and the morbidly obese who have sepsis. We are allowed to use ideal body weight if the patient is morbidly obese. I only use the ideal body weight formula if a patient is greater than 200 kilograms. If a patient, for example, is 150 kilograms, that is four and a half liters of fluid if you do the math on 30 by 3. That is not a large amount of fluid in the context of sepsis. I would feel very comfortable giving that amount of fluid in the context of sepsis.

Let's move on to lactate, another area that sometimes inspires controversy.

Before we talk about the two most common myths in lactate or the areas we have to address, I just wanted to go through some quick frequently asked questions. When I give med staff talks about sepsis, when I visit community hospitals about sepsis, these are some of the questions I commonly am asked about lactate. First is, "What is the cause of elevated lactate and sepsis?" For a long time, it was thought that it was tissue hypoxia and anaerobic metabolism causing an elevated lactate. The science has actually evolved. We all we now know that lactate corresponds with an adrenergic state and a cytokine storm. So, this is actually our body creating synthetic adrenaline and trying to keep our blood pressures up resulting in an elevated lactate. It's just the pathophysiology of it, but if you hear people talking about tissue hypoxia, that is an old school definition that has since been rethought. Now, people also ask, "Do I need an arterial lactate?" Back in the day, we were drawing our lactates off of arterial blood gases. That is more of an invasive procedure. It's more painful, and it's easy to miss your ABG and get a venous sample. Long story short, a venous blood gas sample of lactate or even a regular peripheral lactate are perfectly acceptable, and there is strong correlation between an ABG and a VBG and a peripheral lactate. "Are tourniquet samples okay?" Absolutely. Now, you want to apply some critical thinking. If the nurse has been trying to get a blood draw or the phlebotomist is trying to get a blood draw for 15 minutes and that tourniquet has been up and the arm has turned purple, you might want to drop it down.

Allow some reperfusion and start again, but in general, there are multiple studies on tourniquet samples. There's strong evidence that tourniquet samples are perfectly acceptable. "Is an un-iced sample okay?" It is for 15 minutes. So, if you have a tube system, if you have a phlebotomist who trots right down to the lab and you know that those samples are getting down in 15 minutes, it is perfectly acceptable to put it in a baggie and not use an ice sample if you have hospital processes where it's taking longer than 15 minutes. That sample should be on ice, but I would also challenge you to evaluate your hospital processes. A patient with sepsis should have their lab down to the lab within 15 minutes. Finally, "What is an acceptable turnaround time?" The test itself, a peripheral lactate, actually runs on the machine for less than one minute. It is an extremely fast test. The challenge is getting the sample down to the lab, having the test run, and having it be resulted in the computer in a reasonable amount of time. The gold standard is less than 30 minutes. If your lab turnaround time for lactate is greater than 30 minutes, that's a perfect opportunity for quality improvement projects involving the emergency department, phlebotomy, and lab services. Finally, "We're sending a lot more lactate in the era of SEP-1. Are we bankrupting our health care system? Are we spending too much money?" Not at all. The average cost based on the literature for a peripheral lactate is 40 dollars, and in many health systems, it's actually gone down from there. So, this is a very cheap test. It's a very high-yield test. It provides us with a lot of information that we need to not only risk-stratify the level of illness in our patients but to guide our resuscitative efforts.

So, let's get down to business, brass tacks. These are the two main critiques I hear about lactate: One, it's a non-specific test with a broad differential diagnosis, and we are relying on this too much in terms of making our judgment as to whether sepsis or septic shock exists. The second critique is the cutoff itself. "Why are we using 2?" The medical literature broadly uses a cutoff of 4, and it feels like someone has changed the goal post on us by giving this lactate cut off of 2. I understand this. I'm an emergency physician. I understand the frustration that we sometimes feel when you see a lactate of 2.1 and you feel obliged to repeat it. Let's dig right in.

You can go ahead and forward the animations on the slide. So, the first question critique is that lactate is a non-specific test with a broad differential diagnosis. Why are we relying so heavy on this? The reason we're relying heavily on this is because we're actually using this as a screening test. We want and expect false positives. We are sending lactate on anyone with two SIRS criteria because we want to ensure that we don't have occult sepsis. So, we apply screening tests all the time in medicine. The goal here is not to have the lactate be elevated with every single case. The goal is to cast a wide net and sort the wheat from the chaff, so to speak, to find the patients who are high risk for sepsis and septic shock.

The critique of course is based on lactic acidosis having a broad differential diagnosis. That's true. If you look at the broad differential diagnosis associated with an elevated lactate, you have the adrenergic state, or Type A, lactic acidosis and the non-adrenergic state, or Type B, lactic acidosis. You could get an elevated lactate if your patient's been doing cocaine, if your patient's taking metformin, if they have a thymine deficiency, if they have liver failure. It's a laundry list. I admit it, but if you advance the slides for the animation, the number one cause of elevated lactose acidosis or elevated lactate is actually a shock state. It's the most common cause. So, when we screen for septic shock, the most likely reason for an elevated lactate is indeed the shock state. As we mentioned, at the end of the day, an elevated lactate is a predictor of high in-hospital mortality. Is that information not useful regardless of the cause?

The second critique is, "Why are we using a 2.0 cut-off when the meaningful cutoff based on the literature is 4.0?" Well, this really has to do with epidemiology. This has to do with true positives and false negatives. So, I'm not going to bore you to death, but you all remember the chart below from medical school. That dashed line there is what cutoff are we going to use. If we move the dashed line to the left, we're going to catch more true positives, but we're also going to catch more false positives. If we use the lactate cutoff of 0.0 or 0.1, that would be a useless test. We would be catching a positive result on all patients. If we used a lactate cutoff of 10, that would be a largely useless test.

We would only be catching the patients who are already dying of septic shock or who have extremely high elevated lactate from some other cause. It wouldn't be particularly useful. So, what is the correct cutoff? The Surviving Sepsis campaign is currently advocating for a pretty conservative cutoff, 2.0.

I anticipate that at some time in the future they're actually going to adjust this closer to the 4.0 cutoff that the early literature is based on, but at this time, it's appropriate. We are screening widely with a goal of catching as many patients as we can. As an emergency position you're sometimes going to get that that level of 2.1, and the answer is, "Where do I go from here?" If you've ruled out sepsis, for example, that patient that we talked about who was having a seizure, I would not necessarily send a repeat lactate. You already know that this patient is not septic. You have a competing diagnosis that explains the situation, and you're planning on discharging the patient. It is perfectly acceptable to cancel that repeat lactate. If, however, the lactate is elevated at 2.1, and you're planning on admitting the patient or you have clinical uncertainty as to whether sepsis exists, I would absolutely wait for the repeat lactate. Many a time the repeat lactate has saved me and actually identified a patient with occult sepsis, where I was sort of 51 to 49 sure that they did not have sepsis. So, this is very useful in particular with the extreme elderly or the immunocompromised patient who will sometimes hide sepsis or present with only modestly abnormal vital signs.

Moving on, I want to address a topic that's important not just for the physicians in the room or the APCs in the room, advanced practice clinicians, but also for the quality improvement professionals. Let's talk about position champions or sepsis champions.

The key to having a successful receptive program in your hospital or your health system is to have an engaged physician who knows about sepsis who believes in the sepsis core measure and who is going to help you advance your agenda. When we talk about the role of the SEP-1 position champion, we really want someone to be available for specific tasks.

Now, sometimes these are one-time responsibilities that will help move your project forward. For example, you might want someone to test out a new order set, you might want someone to present at a med staff meeting, you might want someone to help you revise a policy or protocol. Physician champions in general are ambassadors for the QI team. They can provide real-time feedback on QI projects. They can be your guinea pigs, go first when you want to initiate change or innovation, and they can help guide the expectations and accountability when building ongoing measures into the medical staff structure. In short, they are your worker bees. They are your helpers, but they are not quality improvement professionals. So, your physician champion, there's a lot of volunteerism in medicine, and physician champion is generally a volunteer role. It is uncommon that this role is paid for, and you don't want to abuse someone's time. You don't want to use them, for example, for data abstraction, or building Excel sheets, or building graphs and tables, or for making their own PowerPoint presentation for the med staff presentation. You want to support this person and really engage their time for key moments when they need to broadcast the importance of the Sep-1 core measure to a key audience that really requires the authority and clinical expertise of a physician or advanced practice clinician voice.

Now, what are the key traits of a step one physician champion? The number one trait, the absolute bare minimum must have trait, is you need to have a believer in the SEP-1 core measure. Now, that sounds funny. How would we ever choose a position champion who's not a believer in the SEP-1 core measure? I have seen this happen time and time again. The default clinician champion is the medical director, and the medical director may not be a believer. They may be looking at the 2015 literature at the Promise, Process, & Arise Trials, and they may be under the assumption that the SEP-1 core measure is causing harm. If that is the case, you need to find a new clinician champion. They are going to actually poison your efforts, and they are going to derail the efforts to apply the SEP-1 core measure across your patient population. You also want a consistent presence in your hospital.

Many emergency departments are actually staffed with physician staffing groups or have low compendiums present, meaning visiting physician presence where someone is not in the hospital all the time. If somebody is not in your hospital more than 50 percent of their clinical time, they're not going to have the clinical know-how and the relationships with the frontline staff to advance your agenda appropriately. Even though they might be a believer in the SEP-1 core measure, even though they might have the right attitude and show up to the meetings, if they don't have that regular clinical presence, that could be a non-starter for your quality improvement work. Obviously, it's a bonus if the person has a personal commitment. You know I am a firm believer in this. I have a strong engagement in population health topics. This is something that's personally very meaningful to me, and I like to think that I'm very engaged in the work because of that reason. If the person has professional credibility, meaning they treat patients with sepsis on a regular basis or they are interacting with ICU clinicians, emergency clinicians, floor clinicians on a regular basis and able to have those clinical conversations, it's bonus if they have active participation in the quality improvement team or have skills directly related to quality improvement. With this new generation of positions, we're seeing a lot of people who actually have undergone quality improvement curriculum in med school and even residency. So, you'll find a lot of people who speak the same language as our quality improvement professionals in terms of implementation science and really moving the needle on population health. Finally, if you have a person who has effective relationships with other groups in the hospital, especially with administration and nursing staff, those are key relationships that will really advance your clinical care in terms of SEP-1 core measure.

Now, let's say you already have a position champion. How do you retain that person or, more importantly, how do you lose that person? Retention is really about asking the right question. We should ask our physician champions for clinical expertise, but we should not expect a volunteer QI professional. Now, I understand that we live in the real world here and that quality improvement is not always well -funded in our community hospital.

I understand that sometimes the nurse supervisor is also the chief quality officer, is also the sepsis committee leader, is also you know etc. etc. We are all stretched thin, but the production pressures on clinicians are particularly high. Physicians have very little opportunity or very little time to engage in quality improvement work on a regular basis. So, you want to be strategic. Does that clinician or physician need to be present at every single meeting? If you're having a meeting where you're working on a dashboard or working on Excel sheets, that physician may only need to be there for the last 15 minutes of the meeting. "Hey, here is our dashboard. What do you think about it? Is there anything you'd like to change?" You want to be creative about respecting your physician's time. For example, I'm in the emergency department. I work shift work. If you have a Monday morning meeting every Monday at seven, that might not mesh with my clinical schedule. Can I call into the meeting? Could you pop down next time I'm on shift and give me a summary of the meeting or go over the meeting minutes with me? I'm a big fan of non-meetings. They are quick top-end, where my quality improvement professional comes down to the ED. They actually are very nice at my shop, and they come down with some coffee and cookies. They say, "Dr. Redwood, here's the latest sepsis data. What do you think? Dr. Redwood, we just initiated a best practice alert in the HER. Can you try it out and let us know if it works well for you? Dr. Redwood how is the nurse screening process for sepsis going? It's been live for one month now. What have you heard from the frontline staff?" We don't need to have a one hour meeting. You can just come and meet me at my desk next time on that shift and we can talk about it in between patients. Now, what are the wrong asks? You never want to push for interventions that are overly burdensome to clinical workflow. If you ask your physician champion, for example, to have all of the docs write a one-page note every time a patient has sepsis, that is going to be a major impediment to clinical flow. That is going to be more time with the ER than with the patient. That is going to be an unpopular intervention and will ruin their professional credibility. You never want to put your clinician champion or your physician champion in an adversarial position with their peers or medical director. Let's say that you have a very crowded emergency department and the priority for the emergency department is increasing patient throughput.

If you were to introduce, for example, a quality improvement project where every single step of the SEP-1 core measure requires 10 click boxes or a best practice alert in the EHR, you are going to create alert fatigue and undermine the overall mission of patient throughput. You are basically going to have your physician champion burn bridges with their medical director. That is a good way to lose a physician champion. Never ask a physician champion to abandon their clinical voice. So, when you go to substance advocacy websites, or when you look at hospital posters about sepsis, you will hear a lot of advertising talks, or you will see anecdotes about patients who are healthy patients in their 20s who suddenly developed sepsis and decompensated. When you go to the ED, the usual experience is patients who have a high degree of comorbidity, patients who are extreme elderly, patients who are already very sick and succumb to sepsis after months and years of severe comorbidities and illness. That experience on the poster might grab a lot of people's attention to think about sepsis in the community, but that isn't actually the same as our clinical experience. So, when you ask your physician champion to present, for example, to the community or to the med staff let them use their own organic voice. Let them share their own experiences. Don't expect them to match the language that we sometimes see on the internet or in the marketing sphere regarding sepsis. They have a very different voice but a very important voice, and they will have that much more effectiveness if they use their own organic voice

In summary, the 30 by 3 is safe. It should be administered in severe sepsis and septic shock despite comorbidities. A CHF diagnosis on the chart does not mean )percent ejection fraction. Most septic patients with CHF can actually tolerate a 30 by 3 fluid bolus. Iatrogenic fluid overload and ED sepsis resuscitation are quite rare. It's highly avoidable with frequent fluid resuscitation, and in a worst-case scenario, if we do fluid overload a patient, we can always start bypass or intubate the patient. For the peripheral lactate, this is a screening test. We are screening for shock, and the test also represents an independent predictor of in-hospital mortality. You will certainly have false positives. Those are expected, but they're easily dealt with, and they have little effect on clinical care.

Finally, with physician engagement, find your physician champion. Find a believer. Find a stable presence in your hospital and make sure that you understand that their main role is promoting education and behavioral change. Try to make their job easy for them. Prepare the data for them. Prepare their PowerPoints.

When you need them to engage, make sure that it's a role where they are advancing your mission not doing the in-the-trenches work of data abstraction or chart review unless you need a physician voice on that chart with you.

As I said, I have traveled all over the state of Wisconsin, and we have really had tremendous success in terms of advancing sepsis care in Wisconsin and improving statewide mortality. If you look at the figure to the right, this shows the statewide sepsis mortality from 2010 to 2017. Our largest jump in mortality, our largest improvement in mortality, came just months after CMS announced the sepsis bundle. Now, we pulled all of the high performers, all of the hospitals that have low mortality and high compliance with the SEP-1 core measure, and we said, "How do you do it? What is your secret? What is the road map to success?" Here are the consistent themes that we found over and over again. Some of these you're going to recognize because you're doing it in your home hospital, but others might be new to you. If there's anything on this list that you're not doing, consider that as a quality improvement project for the 2021 year. You can create and activate an interdisciplinary team, including the ED and ICU and especially the hospice, if possible. It takes work to create an interdisciplinary team, but sepsis is a team sport. The more that we work together, the more we can move the needle. Establish a process for routine screening in all patient areas with a standardized screening tool. Most of us already have screening in the emergency department. What about on the floor? What about in the ICU? These are additional opportunities to catch sepsis for patients with level of care upgrades. Design automated alerts for severe sepsis and septic shock. This could be a Code Sepsis. This could be the patient's name being highlighted on the electronic medical record.

This could be a protocol where the charge nurse comes and alerts the physician when patient screen positive processes. So, make sure it's automated. Make sure that it is a process that doesn't have to be triggered by an individual but flagged by some sort of screening tool or the electronic health record. Standardized care protocols for patients who screen positive: My preference is nurse initiated protocols for blood cultures lactate and IV fluid. Then, engage the clinician or physician to choose which antibiotics based on source control. A lot of systems have initiated Code Sepsis much like we have Code Stroke, Code [inaudible], Code Trauma. This is perfectly appropriate. It triggers our front line staff to snap into critical care mode as we discussed. Finally, keep the conversation going. Showcase your results, good or bad. This should be a publicly placed scoreboard in the clinical area. This could be monthly that is sent out to all medical directors. This could be your hospital newsletter emphasizing how we're doing on sepsis this month and how we can improve next month. Finally, keep your compass true north. Never give up. I think we all experience the dip in SEP-1 performance during COVID. Obviously, the world is going to through new challenges. We are implementation scientists. We are front line emergency clinicians, and we are going to beat this disease as we have continued to do since early goal- directed therapy came into existence.

Finally, we started this conversation talking about individual health and population health. When you apply the SEP-1 core measure broadly against a large population, you are really able to improve mortality and save lives. In Wisconsin, between 2010 and 2018, we saw sepsis mortality decrease statewide by 32 percent. Thank you for the work you do. What you do is important. What you do matters. The collaboration between quality improvement professionals, physicians, nurses, and APCs is really sacrosanct in medicine. This single graph shows it all, what you're doing to save lives

With that, I'm going to allow time for questions. My door is always open. My email is always open, and I look forward to your questions about sepsis, SEP-1 measure, fluids, lactase, and physician engagement in SEP-1. With that, I'm going to pass it back. Thank you.

**Candace Jackson:** Thank you, Dr. Redwood. That was excellent information, and I'm sure all of our attendees today will find it beneficial and help them with their goals of reducing their sepsis rates. We do have time for a short Q&A session. So, we will try to address some of the questions that were submitted during the webinar today.

We'll be good go ahead and begin with our first question. "The lactate has to be repeated within six hours, correct, but it can be less than six hours when it's repeated."

**Dr. Bobby Redwood:** Yeah. That's correct. So, operationally, I like to teach the repeat lactate a lot as drawn along with the initial lactate since, that's the most effective way to actually do this in real life. In centers that I find that perform well with the lactate and repeat lactate, there is either a reflex order, where the lactate is automatically redrawn in two or three hours if the initial value is greater than 2, or there's an automatic two lactate order, much like we do with troponin, where you order a lactate, and then there's a repeat lactate in either two or three hours. Technically, you do have to six hours to finish the repeat lactate. Good question.

**Candace Jackson:** Thank you. Our next question: "Our challenge is the ED society still does not support the fluids. Suggestions on how to convince ED docs to ignore their society recommendation and follow the Sepsis Surviving campaign recommendations."

**Dr. Bobby Redwood** That is a great question. First, I would have to say I think it's a myth that the ED society does not support fluids. Obviously, there's mixed opinions on this. I am a member of the American College of Emergency Physicians. I'm on the quality improvement and patient safety section of that college as one of their counselors actually, and I am the previous president of our Wisconsin chapter, board of directors of our Massachusetts chapter. There's a strong cohort that supports the SEP-1 core measure. I believe it's the majority of emergency physicians, and there's a very vocal online cohort that is against it.

So, I would say just like the hospitalists, like the intensivists, there is no 100 percent consensus on this, but I wouldn't villainize the specialty. I think emergency physicians in general are supportive of the SEP-1 core measure. So, that being said, I would think our task is to actually show people who are not convinced, show them the literature. We want to we want to explain the population health argument that we laid out in this webinar. We want to normalize the practice. You know a lot of this is fear-based. It's hydrophobia? Am i going to flood all my CHF patients? After you see enough examples, you know. Those of us who sit on sepsis committees can attest to this. We are not flooding our CHF patients. We're actually saving their lives. Then, you want to leverage their peers to bring them into the fold of this safe and life-saving practice. So, on a population health level, this is excellent practice, but we obviously we need to listen to and be able to counter the individual patient concerns of physicians who are hydrophobic, who do not buy into this yet. Honestly, science evolves. So, I have watched this conversation evolve over the past eight years, and we want to hear those people, have an honest discussion, have a club in your hospital, and talk about what the state of the science is. To me, the Surviving Sepsis campaign is an authoritative body that gives very good recommendations, and, in general, I see emergency physicians actually agreeing with those. You know, a lot of times we have a silent majority, where, you know, those quiet physicians who are there taking care of patients. They're in the trenches. They're not going to the med staff meeting and waving articles in the air. They're just doing their thing, and they believe in this measure. So, in my experience in hospitals, it's usually one or two people who are quite vocally against it. That's okay. That's a good conversation to have, and that's part of being
a scientist.

**Candace Jackson:** Thanks, Dr. Redwood. This is one question out of a four-part question, but I think it fits appropriately here. They asked, "Why don't you discuss recent papers that argue against 30 by 3?"

**Dr. Bobby Redwood:** Yeah. Absolutely. I think it's unrealistic in 15 minutes to do a thorough journal club on this topic. There are obviously papers that speak against the 30 by 3, as well. I addressed the three papers against it that really started the conversation in 2015, as well as the meta-analysis of the 16 recent studies. I think it would be a great exercise to have a journal club with your physician group, and let the group draw their own conclusions. My opinion is not the end-all-be-all. I am one person with a lot of expertise in this area who has interacted with a lot of different hospitals on both the physician side and the quality improvement side. My goal is to share my perspective with you. I know I'm reading in the question the [inaudible] et al article that was a retrospective data. I was not particularly impressed with that data. It was 1000 patients, and they found no difference in the usual care versus 30 by 3 arm. So, you know that data are included in the meta-analysis. Kahm et al in 2019 was also a 208-patient study that found no difference, but there are quite a few studies that show a marked improvement. That, you know, that is the state of the medical literature. You'll see some that show different, some that don't, and then you'll get the meta-analysis that hopefully serves as the tiebreaker and gives us the preponderance of literature. Yeah, I'm not trying to portray a one-sided view where there's no negative studies for this. There absolutely are.

**Candace Jackson:** Thank you. Our next question: 'Does the x3 in the 30 by 30 mean that you give it within three hours. I missed the part. Why is it 30 by 3. I know you give thirty milliliters per kilogram bolus."

**Dr. Bobby Redwood:** Yeah. Absolutely. In general, I don't like to use shorthand, and we discussed this at the beginning of the conversation. It just takes a really long time to say you know, "Dr. Redwood, did you give the 30 milliliters per kilogram normal saline fluid bolus?" When you're taking care of a critical patient in the emergency department, I find that the 30 by 3 language is very helpful for clinical communication. That is shorthand, and it's important to understand what we're talking about.

We are talking about giving it within three hours. So, 30 cc per kilogram of crystalloid within three hours.

**Bob Dickerson:** This is Bob. Just as a clarification, as far as what the measure requires, the SEP-1 measure requires that the 30 be started within three hours, but it does not require be completely infused within the three hours. The difference is in that, as Dr. Redwood has referenced, getting those fluids on board as quickly as possible is very important for patient care, but when you're measuring that kind of thing, what we will do with measures sometimes is give a little bit more time knowing that there are going to be those cases where they get it started within the three hours but maybe because of how the patient is presenting and their symptoms are manifesting, that it just isn't possible to get it all infused within the three hours, but you've still provided good patient care. So, I just want to throw that clarification as far as how that would potentially relate to the measure

**Dr. Bobby Redwood:** Bob, Thank you for that clarification. You know, I always tell my teams as well that, sometimes when you set a metric, there's this perception that everyone should be getting 100 percent, and if we all scored 100 on this SEP-1 core measure, we would be vastly over treating patients. So, no health system in the nation scores 100 on this and nor should they. So, we have to apply clinical judgment to this as well. So, you know, I see the highest performers around 70 or 75 percent, and I think that's kind of a nice point to be at. Because good clinical care, like you said, you know, things come up. It can be hard to get that IV. The patient might not tolerate a central line. They might need X, Y, or Z intervention before the SEP-1 core measure. This isn't this isn't cookbook medicine in the sense that we're just throwing the same thing at everybody. We're using our Clinical Gestalt. We're using our clinical judgment, but, in general, you know, when you look at the average patient, you should be able to get these done in the timely fashion as outlined by the measure.

**Candace Jackson:** Wonderful. I have a question here that, again, I think we might need both Dr. Redwood and Bob to address as it affects both the measure writer and Dr. Redwood as a clinician.

The question is, “Patient’s with clinical pulmonary edema. Her CMS abstraction guidelines will not pass the measure. I am wondering why CMS did not use this condition as an exception to receiving all the required 30 milliliters per kilogram fluids, and, as a result, we’ll pass the sepsis core measure.”

**Dr. Bobby Redwood:** Hey. I can take a stab at this. So, if I can paraphrase this, “Why did CMS not give an exception for patients with pulmonary edema?” In my experience the SEP-1 core measure, I mean this is revised year after year and continues to improve. There is no perfect measure, right? We’re always striving for perfection and never getting there, like everything in life I would say. This suggestion comes back up year after year from key stakeholders, from hospital associations, as a question. Why aren’t we giving an exception for this? The Surviving Sepsis campaign is really holding steady that the 30 by 3 should be administered despite co-morbidities. So, I happen to agree with them, but it is a lively and perennial debate, and I think one we should continue to have.

**Bob Dickerson**: Thank you, Dr. Redwood. This is Bob again. I would agree with that. One of the things that is really a challenge when you’re developing a measure is to try to account for all the different what-if type situations or the different comorbidities or way that patients may present. You try to develop something that will work for the majority of patients knowing, as Dr. Redwood mentioned earlier, that the goal is not 100 percent with every patient all the time. You will have outlier cases and patients but, that said, we do continually review the feedback, and keep an eye on the literature, and look at what the performance of the measure is, and what is happening in the field of sepsis care. Then, we take that information on a regular basis, at least annually sometimes more frequently, to identify if there are some changes or visions we should make to the measure to account for these different types of things. So, a wonderful question.

**Candace Jackson**: Thank you both, Bob and Dr. Redwood. Next question: “How do you define time 0 in a gradually worsening patient?”

**Dr. Bobby Redwood**

Time zero is when you suspect sepsis based on SIRS criteria and a source of infection. That can be when the chest x-ray comes back and shows an infiltrate. That could be if you have a best practice alert, and it says, "Do you suspect sepsis?" and you click the best practice alert. It kind of depends on what is charted when and what information comes back at what time. So, there's, you know, this is one of these things where there's no perfect way to capture reality. Some of us have built an electronic health record that tries to capture that, and some of it depends on time stamp documentation. There's so many different EHRs out there, and I find that the health systems that have, you know, scanned medication, scan fluids, everything is time stamped, based on barcodes, will generally start that clock a little bit earlier. If your electronic health record is based on physically entering information, then in general the time 0 perspective tends to be a little delayed. You know, even more nuanced than in the modern era, is EMS-activated sepsis. A lot of times, time 0 will be pre-hospital, which really in the ED gives us a pretty significant challenge of getting this all done in three hours. It's all good work, and I always encourage people to stay positive and to embrace innovation because a lot of smart people are working on snapping into critical care mode earlier and finding ways to do this in a timely fashion.

Candace, you were fine. You skipped over a question that I think maybe you thought was like a bullying question or something, but I would actually like to address it. Somebody said that being a believer statement is an anti-scientific statement. You know, how can you stand as a scientist with this? So, I love docs because we speak our minds, and we should always speak our minds in medicine and a scientist. So, I totally agree with it, and I totally hear this person. This webinar is given to a broad audience from many different role groups, and that section that the statement was made was about finding a physician champion. So, the terminology used by quality improvement professionals, by administrators, and others sometimes strikes physicians as unusual or non-scientific. One of the things that really harms our patients is when we're not a team. We have got to be a team, and I think of quality improvement professionals as implementation scientists.

They study the science of improvement. They study the process of getting best practices to the bedside in a rapid fashion because medicine moves at a glacial pace sometimes, and we all know that there's 20 year old dogma that has still not gotten to the bedside. Part of the reason for this SEP-1 core measure is to jump start clinicians and really get these evidence-based practices to the bedside in a rapid fashion. Now, I think that is the process that has become a little bit politicized. There was a recent study by Merrick at al. Merrick is a physician with a very strong online presence against the SEP-1 core measure that actually called the whole thing a hoax, you know. That's language that you're not used to seeing in the medical literature. So, some of this has become charged because we are talking about population health medicine. You know, to a group of professionals, this is relatively recent. We've done a lot of individual medicine for decades, and we're now talking about population measures. I would not call that anti-scientific necessarily. I would talk about the merging of two worlds, and I agree with you that "believer" is not necessarily germane to the physician vocabulary, but that many people use that in the quality improvement professional sphere. I think the crux of the argument is really talking about finding a physician champion. You want a physician champion who has weighed the literature and who believes they can advance the agenda of what the health system is looking for. In general, I encounter health systems looking for improving sepsis mortality through the SEP-1 core measure.

**Candace Jackson:** Thank you, Dr. Redwood, for addressing that. I think that will be very beneficial for everyone to hear. We do have a time for a few more questions here. "What is your opinion on early presser intervention along with IV fluids?"

**Dr. Bobby Redwood:** Oh, this is such a hot topic. I would say that there's a strong body of evolving literature that we could be giving pressers early. The conventional wisdom is fluid resuscitate the patient first, and then if your fluid resuscitation fails, move on to pressors. That sort of escalates care pressors. We are now giving peripheral pressures for up to 24 hours and an 18-gauge IV in the antecubital fossa or higher.

People are talking about other non-central line options like midlines, ultrasound-guided midlines, that are less of a risky intervention than the central line. We're essentially discovering new ways to deliver pressor therapy early on in the patient course. So, I'm going to watch his body of literature intently. I expect the Surviving Sepsis campaign to weigh in on this shortly, but, in general, I see it as a positive trend, and I'm very open to that concept.

**Candace Jackson:** Thank you. We'll kind of switch gears here and talk a little bit about antibiotics and blood cultures. "Can the antibiotics start time take place after the first blood culture collection time, or are the antibiotics to be started after the second subculture collection time?"

**Dr. Bobby Redwood:** Let's see. I'm not sure I totally unpacked that correct question completely but let me just outline the common situation. In general, we should be able to give blood culture or draw two blood cultures prior to giving antibiotics. That being said, I work in real life. I understand that sometimes it's really hard to get those blood cultures, that we have poked a patient 10 or 12 times, and we feel that we are doing harm by delaying antibiotics. If that's the case, we should never delay antibiotics. We should give the antibiotics and document in the chart what the delay is. We may fall out of the core measure, and that's okay. These are cases where you want to practice the best medicine possible, but a best practice is clearly to get blood cultures prior to antibiotics. In the ED setting, we don't always realize the value of that, but there are patients with multi-drug resistant organisms. There are patients who fail antibiotics, and three days later in the ICU those blood cultures can be worth their weight in gold. Most patients are going to improve, and you won't actually need that blood culture data necessarily, but it is also useful for antimicrobial stewardship in terms of narrowing your therapy. So, we start with broad spectrum agents, and you want to narrow that down to a narrower agent. Once you know what your exact pathogen is. In the arc of surviving sepsis, blood cultures are very important, and that's part of the thing I love about the SEP-1 core measure. It's a teamwork approach.

We are now doing things in the emergency department that help our colleagues on the floor, that help our colleagues in the ICU, that help get these patients discharged on pills in a more rapid fashion. Yeah, I wouldn't beat yourself too much if your antibiotics are given after blood cultures. In general, we want to do that, but, hopefully, there's a good reason that happened. One thing that I do see time and time again is that the nurses will hang the antibiotics and then they will scan them later. If you do that, it creates like an automatic time lag. So, maybe they scan the antibiotic ten minutes after they gave them, and then, in the EHR, it appears that the antibiotics were given afterwards when, in reality, they were given beforehand. So, I would say good nursing practice is to scan the antibiotics first and then immediately hang them afterwards. That's, you know, more of systems issue, but sometimes that can create the false impression that antibiotics are being given too late.

**Candace Jackson:** Thank you. This has been wonderful. Go ahead, Bob.

**Bob Dickerson**: Just a little bit of clarification in terms of how the measure treats a data element called *Blood Culture Collection Acceptable Delay*. That data element is designed to account for situations where it is clinically appropriate for whatever reason for the antibiotic to be started before the blood culture. So, I would urge the person that asked this question to look at that data element. If you have questions about that data element in the data dictionary, submit a question via *QualityNet*, and we can help clarify that for them, but we do try to account for these different types of situations in the measure.

**Candace Jackson:** Thank you, Bob. Unfortunately, we're running out of time. So, I think we're going to end our Q&A session with one last opinion from Dr. Redwood: "Do you think the sepsis one-hour bundle will ever be implemented?"

**Dr. Bobby Redwood:** There's a controversy and a half for you. I work with the Wisconsin Hospital Association, and our current position is that it's not ready for prime time.

I could imagine a future where we could implement the sepsis one hour bundle, but I have a lot of questions still and would like to see the literature evolve more on that. The Surviving Sepsis campaign is known for pushing the envelope and really advancing things in a rapid fashion. So, I feel like this conversation kind of got put on hold during COVID, and it is going to pick back up again in 2021. So, I look forward to you inviting me back and we can have a webinar about the one-hour bundle. How about that?

**Candace Jackson:** Okay. Thank you, Dr. Redwood. Again, we'd like to thank Dr. Redwood for providing all of this useful information today. The discussion and presentation were very excellent. Next slide, please.

As we indicated, this webinar has been approved for one CE credit. The information that you need to get that is on the link on the slide. Next slide, please.

We thank you for completing the survey which is on the next slide. Next slide, please.

We'd like to conclude with thanking everyone for joining us today, and we hope that you have a great rest of your day. Thank you, again.