## Sepsis Update: Early Identification and Management
### Q&A From the Live Webinar

**Presenter:** Tom Ahrens, RN, PhD, FAAN  
**Live webinar:** Thursday, May 09, 2013

The AACN Critical Care Webinar Series is not only an efficient way to learn from true thought leaders within our community, it serves as the seed of robust discussion among colleagues. To encourage continued discussion, our experts have responded to participant questions not addressed during the live webinar. Please enjoy reading the responses below.

| Q: Is the recommendation one hour for ED and inpatient areas? | A: Yes |
| Q: According to the new 2012 guidelines, the two care bundles (resuscitation and management) have been replaced with 3 and 6 hour bundles? | A: Yes, that is correct. |
| Q: How can we mandate the physician follow the protocol? | A: Incredibly important question. A physician leader, or administrative leader, must guide medical practice so this becomes an expected behavior. |
| Q: In a patient who has been adequately fluid resuscitated and now on multiple pressors but remains hypotensive, is there evidence to support the use of cortisone? | A: Hydrocortisone is recommended (200 mg daily, either continuous or in 50 mg doses) in this situation. |
| Q: Would you recommend fluid therapy and are there any EBP out there to support it? Do you think that a lab test CRP is a good indicator for early sign of sepsis? | A: CRP is not recommended as an early indicator of sepsis. Fluid therapy of normal saline is preferred but any fluids, including albumin, can be given. |
| Q: You mentioned involving the family to determine how aggressive care should be. At what point do you have this discussion? It seems we jump to putting in the central line, giving the fluid bolus, starting antibiotics, etc. but is there a point in there where we can talk to the families before that? | A: The discussion with the family should be done as soon as possible, especially if an end of life situation may be present (e.g. patient with dementia from a nursing home). The discussion can be done as soon as sepsis is recognized. |
| Q: What is your take on SVV as a superior method of determining fluid responsiveness as compared to CVP? | A: SVV is better than CVP. CVP is a static measure of pressure with no real indication of flow. SVV or stroke volume optimization are better indicators of fluid responsiveness. |
Q: How often should lactates be drawn? What is the value of Procalcitonin in diagnosing sepsis?
A: Probably every 3-6 hours. PCT is used to identify if a bacterial infection is present. It may be elevated in sepsis if sepsis is due to a bacterial infection. PCT levels are used to guide length of antibiotic therapy.

Q: Why is an insulin sliding scale q6hrs not adequate, rather than an insulin IV recommended?
A: Blood glucose levels can change too rapidly for a sliding scale to properly manage. Sliding scale insulin levels are not recommended for any acute blood glucose monitoring.

Q: Are there other conditions that raise lactate that we should consider?
A: Any condition that produces hypoxia can cause lactate to elevate, e.g. cardiogenic shock. Hepatic dysfunction can also produce elevated lactates.

Q: How early will Lactate levels start to rise in sepsis? Is there any value in screening all patients in ED and daily in ICU for Lactate levels?
A: Lactate will rise as soon as tissue hypoxia becomes evident. I would screen all patients with signs of sepsis with lactates. Then, if treatment has been started, continue to measure every 6 hours until the lactate has normalized.

Q: Is there a big difference between arterial and venous lactate levels? Is one recommended over the other?
A: Yes, arterial levels are preferred. However, from a trending perspective, venous levels can be used.

Q: SSC guidelines for fluid volume resuscitation are specific for when the blood pressure and/or lactate are affected. How do we aggressively treat patients with severe sepsis that are normotensive with a lactate <4?
A: One way of addressing this is to treat patients with lactates between 2-4 with fluid resuscitation. Unfortunately, there is a group of patients with sepsis who do not show signs of SIRS. In this case, clinical judgment needs to be followed. Until we get better diagnostic criteria, we will miss some patients with sepsis.

Q: As nurses, how can we better reach the public for early recognition/detection to have patients respond quicker to their PCP or clinic?
A: The Sepsis Alliance is trying to do this. Their web site illustrates their efforts. I would encourage AACN to work with them, or reach out the SA directly.

Q: Is there an educational tool that has been developed that is available for family education about sepsis?
A: Not yet, except the Sepsis Alliance does have some public information.

Q: Does anyone have any information on protocols for the identification of pediatric sepsis that they would be willing to share? We are expediently working to meet the criteria set forth in a new state mandate for developing protocols on the identification and management of pediatric sepsis, and would be very grateful for any information that people are willing to share.
A: In the SSC guidelines there is a section for pediatric sepsis. They might be helpful. On the tools page there is a sample protocol for adults that might be able to be modified for pediatrics.
Q: Do you have any suggestions as to who I could contact for further information regarding identification of pediatric sepsis?
A: The Surviving Sepsis Campaign website does have pediatric guidelines, plus the article in CCM January of 2013 contains pediatric guidelines.

Q: The new guidelines are 30mL/kg for volume. Since this is weight based, is this relevant to the pediatric sepsis population?
A: I would use the pediatric guidelines published by the Surviving Sepsis Campaign.

Q: I would like your thoughts on Procalcitonin as a tool to not only deescalate antibiotics but also help distinguish between a viral and bacterial infection.
A: PCT is used to identify if a bacterial infection is present. It may be elevated in sepsis if sepsis is due to a bacterial infection. PCT levels are used to guide length of antibiotic therapy, but not a marker for sepsis.

Q: What role does procalcitonin play in sepsis identification and treatment?
A: None. PCT is used to identify if a bacterial infection is present. It may be elevated in sepsis if sepsis is due to a bacterial infection. PCT levels are used to guide length of antibiotic therapy.

Q: Procalcitonin as a biomarker - opinion please for diagnosis and monitoring.
A: PCT is used to identify if a bacterial infection is present. It may be elevated in sepsis if sepsis is due to a bacterial infection. PCT levels are used to guide length of antibiotic therapy.

Q: Are you using procalcitonin levels for early identification of sepsis I didn't see the reference for the lactate and BP graph, will you please provide that?
A: No. PCT screens for infections, not sepsis. Lactate screens for hypoxia, not sepsis. Both can be drawn but lactate tells you more about the urgency of the situation. The reference is from my ED. The reference was provided by Chris Holthaus, MD as part of our quality review. I will also post the reference about taking lactates before vital signs.

Q: In the presentation, slide 17, you mentioned a ScvO2 of 82%, too high. I thought only SvO2 could be too high, but the ScvO2 should be > 70%.
A: No, both ScvO2 and SvO2 are interpreted about the same way. ScvO2 tends to be a little higher than SvO2 (5-13%).

Q: Our providers are reluctant to use ScvO2 despite our full ability to do so - any suggestions?
A: The community facility in which I work has recently implemented a sepsis protocol. The ICU/CCU has installed new GE monitors for the campaign and has had in-services on the Vigilos that correspond with the bedside cardiac monitors. Keeping in mind that our facility is small, the internal medicine physicians and hospitalists are a bit reluctant to use order sets. We do have a physician champion who is leading the campaign but there is still not a overwhelming amount of compliance. Do you have any suggestions for increasing the compliance to use the evidence based order sets for sepsis? Is there a physician leader who can help guide medical practice? This is where a strong central team is necessary. It is hard to change practice by yourself. Having physician allies who can help change practice is essential.

Q: Our hospital has just started a Sepsis initiative and will be doing bedside Lactate levels using the ISTAT. Has anyone used this before and if so the process of getting the result of the Lactate level quick?
A: We use the IStat Lactic acid for Sepsis all the time. Our docs order it every 2 hours, and resuscitate appropriately along with ScVo2.

Q: Case Scenario: In office as a provider and patient comes in with HR 105, T 100.5 with suspected URI or sinusitis, based on definition the patient has SIRS and therefore sepsis. Do I rush the patient to the ED to begin Sepsis protocol?
A: Yes.

Q: Do these guidelines apply to pediatrics?
A: The article in Jan 2013 CCM from the Surviving Sepsis Campaign provides pediatric guidelines.

Q: Evidence surrounding whether to draw a venous vs. arterial lactate?
A: Arterial levels are preferred. However, from a trending perspective, venous levels can be used.

Q: Our hospital has just started a new Sepsis initiative and will be doing bedside Lactate levels using the ISTAT. Has anyone used the ISTAT and is the process of getting your lab result quick?
A: I have used the iStat and it is great! It can be used for to perform ABGs, chem panels, etc. It provides rapid results but the cartridges cost quite a bit. We carried it with us when we responded code situations outside of the ICU as well as for morning labs. The problem that seems to come up in facilities is that it requires a CLIA waiver and someone to oversee the program. I don't see it being any different than a point of care blood glucose test.

Q: Do you see Ultrasound measurement of IVC used more often than CVP readings for volume status?
A: Not yet. I think this will happen over the next 3-10 years, especially as nurses start to become proficient in ultrasound usage.

Q: What line items do you think will be selected as a core measure for sepsis?
A: The 3 and maybe 6 hour criteria.

Q: What do you think is the better way for nurses and physicians to follow the guidelines a policy or a protocol?
A: Both can work. It is really a matter of how strong the sepsis leadership team can implement either. I prefer a protocol, but that is just my opinion.

Q: Have you seen Hydrocortisone gtts used in place of bolus doses?
A: Yes, there is some suggestion that a drip may be superior to bolus dosages.

Q: You mentioned STO2 as a non-invasive parameter in one of the case examples in the webinar. What is your expert opinion on utilizing STO2 as warning parameter in early detection or combining STO2 and SCVO2 for guiding sepsis interventions?
A: STO2 has the potential to be a great early warning sign in sepsis or any condition of hypoperfusion. Research is building to support this application, but definitive studies are still lacking. Right now, it is more of a theoretical benefit. I would certainly encourage it’s use as a warning sign and if the ScvO2 decreases, investigate if hypoperfusion is developing. This can be done by measuring stroke volume (non-invasively) and obtaining a lactate (which may not have elevated yet).

Q: What is the value of SvO2 monitoring in regards to sepsis? Are there any other ways to monitor tissue supply/demand that have an acceptable risk vs. benefit ratio as opposed to Swan-Ganz catheters?
A: ScvO2 is the target for resuscitation, e.g. achieving a value > 70%. No PA catheter is needed, just a central line.

StO2 is a completely non-invasive measure of tissue oxygenation that I believe is very helpful.

Q: Should we aggressively treat hyperthermia in sepsis?
A: No, no evidence exists to support aggressive treatment of hyperthermia.

Q: Are there guidelines for transfusions in sepsis?
A: Yes. Blood transfusion guidelines are:

Once tissue hypoperfusion has resolved and in the absence of extenuating circumstances, such as myocardial ischemia, severe hypoxemia, acute hemorrhage, or ischemic heart disease, we recommend that red blood cell transfusion occur only when hemoglobin concentration decreases to <7.0 g/dL to target a hemoglobin concentration of 7.0 – 9.0 g/dL in adults (grade 1B).

Not using erythropoietin as a specific treatment of anemia associated with severe sepsis (grade 1B).

Fresh frozen plasma not be used to correct laboratory clotting abnormalities in the absence of bleeding or planned invasive procedures (grade 2D).

Not using antithrombin for the treatment of severe sepsis and septic shock (grade 1B).

In patients with severe sepsis, administer platelets prophylactically when counts are <10,000/mm3 (10 x 109/L) in the absence of apparent bleeding. We suggest prophylactic platelet transfusion when counts are < 20,000/mm3 (20 x 109/L) if the patient has a significant risk of bleeding. Higher platelet counts (≥50,000/mm3 [50 x 109/L])

Q: What is the latest thinking on using Xigris?
A: It has been withdrawn from the market. Not sure if it will reappear, but if it does, it will be highly limited in application.