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From Bench to Bedside: Reducing Sepsis Mortality

Each year 750,000 people will develop sepsis and more than 210,000 of them will die. Can you identify who will benefit most from early intervention?

You are listening to ReachMD, The Channel for Medical Professionals. Welcome to the Clinician's Roundtable. I am Dr. Shira Johnson, your host, and with me today is Dr. Peter DeBlieux, Professor of Medicine for Pulmonary and Critical Care at Louisiana State University Health Science Center and Professor of Surgery at Tulane University. Today we are discussing surviving sepsis, improving mortality with early diagnosis and treatment.

DR. SHIRA JOHNSON:

Welcome Dr. DeBlieux.

DR. PETER DEBLIEUX:

Well, thanks. Thanks for inviting me on.

DR. SHIRA JOHNSON:

So first of all, why don't you define for us what you mean by sepsis, so that we are all talking in the same language here?

DR. PETER DEBLIEUX:

Sure, I think that it's important to understand that sepsis is considered really a systemic inflammatory response that the body has due to an offending infectious agent or cause.

DR. SHIRA JOHNSON:

Would you like to explain that a little more because there is just different types and different causes of sepsis, right?

DR. PETER DEBLIEUX:

Absolutely. So, the way that we look at it is first a compilation of vital sign abnormalities, so either temperature abnormalities, either very high temperatures, fever, or very low temperatures. Likewise, an elevated pulse rate and then also an elevated respiratory rate. If we combine this with an abnormality in your white blood cell count which is the count that helps us to determine whether or not you are fighting off infection or not. If you have abnormalities in white blood cell count (**01:30**), either very high counts or very low counts, that qualifies you as well. Now, there are a lots of different disease processes that can give you mimickers or things that look like sepsis, but when we think that it's due to an infection and it's those associated symptoms and abnormalities of either vital signs or white blood cell count that we then determine it as sepsis, and once you determine as sepsis, then it's broken down into sepsis which is simply those factors with the presumed infectious etiology, severe sepsis which is sepsis associated with an end-organ dysfunction, so either confusion or weak heart that results in a very low blood pressure, kidney dysfunction, bowel dysfunction, or cellular breakdown, things that would cause a blood lactate to be elevated, things that would cause your cell counts, either white blood cell counts or red blood cell counts to be depressed or for you to have a bleeding problem.

DR. SHIRA JOHNSON:

So what is the mortality of severe sepsis and septic shock?

DR. PETER DEBLIEUX:

Sure, so, the other marker is septic shock and that's sepsis associated with very low blood pressures and most people utilize Roger Bone dictum which is a systolic blood pressure less than 90 mmHg so when you then talk about what's the outcome, the outcome hasn't really changed dramatically over the last 50 years until just recently in the last two years. So that outcome is for severe sepsis, typically about 30% mortality. When we start reaching into septic shock, those people with severe sepsis (**03:00**) and then very low blood pressures, the mortality escalates to 40% plus and so it is a very mortal disease and it remains so, I mean less so in the recent past few years with the advent of things like early goal-directed therapy and far better science and far better clinical practice for the treatment of sepsis.

DR. SHIRA JOHNSON:

So before we get down to some of the specifics, but what do we know about sepsis today that we didn't know 20 years ago, what's some of the big changes in our science in our understanding up the disease.

DR. PETER DEBLIEUX:

Sure, some of the big changes in science for the disease is noting that early intervention actually yields better outcomes, and this makes sense to us intuitively right. We know that there is a golden hour for trauma in which if we impact the patient in that initial 60 minutes, we can change outcomes. We know that there is golden time associated with strokes and the interventions that are needed to reverse the pathophysiology that occurs in strokes. We also know that if we take acute coronary syndromes and bring them to a Cath lab early, we have far better results. This is along those lines so we know that if we use early intervention with antibiotics, we can change outcome in sepsis. We know that if we are early and aggressive with your resuscitation with both fluids and vasopressors that we can change outcome. We know that in those patients that have sepsis and anemia, that early intervention with blood transfusion again offers you a better chance in improvement in outcome and so those steps (**04:30**) are really critical and have been looked at most recently as changing outcome. Now these steps before were used once the patient had already made it out of the emergency department and into the ICU for you know 12 to 24 hours and then these steps were really addressed. When they are addressed late, they really aren't linked to improved outcomes. However, if you look at early intervention, the initial 6 hours of presentation of those patients in either the

emergency department setting or in the critical care setting, these outcomes are real and different than before.

DR. SHIRA JOHNSON:

How do we learn this, how do we learn that early goal-directed therapy is important. What studies were done?

DR. PETER DEBLIEUX:

You know, the main study that was done was a New England Journal study done by Emmanuel Rivers and many people consider him a giant in the field of both critical care and emergency medicine. He practices both and his initial work called Early Goal-Directed Therapy is really the work that has changed our thought process in regards to goal-directed therapy and whether it makes a difference. As I quoted to you before, goal directed therapy has really been awash once it's done late, but the concept of doing this goal-directed therapy in an acute timeframe much like the golden hour of trauma, much like the golden hours of both coronary ischemia and cerebral ischemia when you roll it into a sepsis picture, that early intervention in the initial 6 hours is linked to improvement in outcomes.

DR. SHIRA JOHNSON:

If you have just joined us, you are listening to The Clinician's Roundtable (06:00) from ReachMD, The Channel for Medical Professionals. I am Dr. Shira Johnson, and I am speaking with Dr. Peter DeBlieux from Louisiana State University Health Science Center and we are discussing surviving sepsis, improving mortality with new therapies.

How do you achieve management and make it simple for the non intensivists?

DR. PETER DEBLIEUX:

You know, that's a challenge and a life-long goal for me at the medical center with LSU and the concepts are very difficult, but the way to break it down easily, the way I have kind of used it, is I use a pneumonic and I call it a " DAHAM " pneumonic, and " DAHAM " being daham, kind of a southern drawl to it, if you will, a DAHAM pneumonic.

DR. SHIRA JOHNSON:

Okay,

DR. PETER DEBLIEUX:

And the concept being that "D" is the diagnosis is suspected early, so the consideration of the diagnosis, so those patients who present with systemic inflammatory response syndrome which is SIRS syndrome that we consider that. Those people's aberrations in pulse and those people with elevated respiratory rates and those people with temperatures that are either very low or very high and in those

people with either low white blood cell counts or elevated white blood cell counts that we consider the diagnosis of sepsis in those patients. So if early diagnosis is considered and you have got sepsis in your differential diagnosis, then we would advocate early institution of antibiotics. Certainly within an hour of the consideration of the diagnosis of sepsis. We think that early intervention with appropriate antibiotics will actually change outcomes (**07:30**), and so that's the "D" diagnosis expected, the first "A," early intervention with antibiotics, and then the "H" is actually hemodynamic support, and the hemodynamic support is the concept that we would like a full tank and the way that we are measuring that we advocate measuring a full tank is to have a central venous line placed ideally in either the IJ position or subclavian position, again in the superior vena cava to go ahead and measure a couple of different things, one is the central venous pressure, and the goal being CVP of 8-12 mmHg would be our goal. Once we have attained that that would be our goal status for the patient in regards to assessing a full tank. Now, this is debatable and the science behind this is not actually robust, but the practice habit and the surviving sepsis campaign supports a CVP in 8-12 and in those patients who are on mechanical ventilation, we actually advocate may be 12 to 14, even higher central venous pressures.

DR. SHIRA JOHNSON:

Tell us some more about the Surviving Sepsis Campaign.

DR. PETER DEBLIEUX:

Sure. Well, the Surviving Sepsis Campaign has really embraced Emmanuel Rivers' Early Goal-Directed Therapy and then promoting early intervention of emergency medicine clinicians in impacting the outcome of septic patients. Initially, first, as I said, with a pneumonic, diagnosing early, giving early institution of the antibiotics, addressing hemodynamics early, not just CVP, but maintaining mean arterial blood pressures 65 mmHg and greater (**09:00)** in those cases, and in those cases measuring, if you would, serum lactate as well as a central venous oxygenation saturation. So sampling from the distal portion of the central venous catheter, saturation and maintaining saturations greater than 70%. In those patients that have saturations less than 70%, the mindset is to initiate vasoactive agents, in particular, a vasopressor dobutamine and the concept there is to improve cardiac output in those patients with ScvO2's less than 70%. Again, a portion of the goal-directed therapy. In those patients that have hemoglobins and hematocrit less than 10 and 30, the concept is to transfuse them with packed red blood cells to an ideal H&H of 10 and 30 in those patients that have ScvO2's less than 70%. So if I have an ScvO2 that's 70% or greater, I don’t reach for dobutamine and I don’t reach for blood transfusions. However, if my ScvO2 is less than 70, I would reach for dobutamine and have a consideration for transfusion of packed red blood cells in those patients that were anemic with an H&H less than 10 and 30. As we go further with that management in a septic patient, we also consider should we intubate these patients and should we control their airway, and for those people who are requiring either vasopressors to maintain a mean arterial blood pressure greater than or equal (**10:30**) to 65 or dobutamine to drive the cardiac output to maintain an SCVO2 greater than 70%, those people should be expectantly intubated so that we can control that airway and direct that cardiac output away from the muscles of respirations and toward other vital organs such as the brain, such as the heart, such as the kidney and the liver.

DR. SHIRA JOHNSON:

So if you are an intensivist, this should be your bread and butter, but if you are a non-intensivist, may be you are not recognizing sepsis. Why is it commonly missed in the ER or in the hospital?

DR. PETER DEBLIEUX:

Well, that's a great question. When we talk about common misses for sepsis, there are many mimickers for it. So things like if we think this is a pulmonary embolus, then we would expect what; your heart to be high, we would expect your respiratory rate to be high as well, and you might even be hypotensive. No if that was your working diagnosis and then suddenly you come back with a CAT scan or other studies that showed there was no venothromboembolism, then there was a delay in as much as two to three hours while we are chasing

an alternative diagnosis and not considering sepsis. So we wind up missing not considering the diagnosis, not instituting antibiotics early, not instituting the appropriate measures of early goal-directed therapy or you know, as I coined to you the DAHAM pneumonic, we are not chasing those hemodynamic measures, we are not transfusing blood, we are not aggressively intubating these people, controlling their airways and then the last component of the DAHAM pneumonic is the "M" stating from metabolic measures, different things that we look at, whether we are talking about a tight (**12:00**) glycemic control, whether there is a consideration for steroids or whether there is a consideration for other measures such as activated protein C, what is commonly known as Xigris and you know those other metabolic considerations in those patients who are septic.

DR. SHIRA JOHNSON:

Thank you for being our guest.

DR. PETER DEBLIEUX:

Thank you, Dr. Johnson.

DR. SHIRA JOHNSON:

Our thanks today goes to Dr. Peter DeBlieux who has been our guest. We have been discussing surviving sepsis, improving mortality with early diagnosis and treatment.

I am Dr. Shira Johnson. You have been listening to the Clinician's Roundtable from ReachMD, The Channel for Medical Professionals. Please visit our website at www.reachmd.com which features our entire library through on-demand pod casts or call us toll free with your comments and suggestions at 888-639-6157. Thank you for listening.

You are listening to ReachMD, The Channel for Medical Professionals. Welcome to Patient Safety News provided by the Food and Drug Administration, the FDA, Protecting and Promoting the Public Health.

Today's highlight is hosted by Mark Barnett and Anita Reiner.

ANITA REINER:

In October 2007, FDA warned healthcare professionals about reports of acute pancreatitis in patients taking the antidiabetic drug Byetta or exenatide. Since then FDA has received reports of 6 cases of hemorrhagic or necrotizing pancreatitis in patients taking Byetta, 2 were fatal.

MARK BARNETT:

Byetta and other suspect drugs should be promptly discontinued if pancreatitis is suspected (13:30). There were no know patient characteristics that can determine whether a case of pancreatitis that's associated with Byetta will develop into the hemorrhagic or necrotizing forms. If pancreatitis is confirmed, it should be treated without delay and the patient should be carefully monitored until recovery.

FDA is reminding healthcare professionals that patients who take amiodarone along with drugs that contain simvastatin have an increased risk of rhabdomyolysis. It's a type of muscle injury that can lead to kidney failure and death. Amiodarone is an antiarrhythmic drug and simvastatin is used to lower cholesterol.

ANITA REINER:

This increased risk has been described in labeling both amiodarone and simvastatin since 2002, but FDA continues to receive reports of rhabdomyolysis in patients treated concurrently with these two drugs, especially when the simvastatin dose is greater than 20 mg per day. Prescribers should avoid doses of simvastatin greater than 20 mg per day in patients who are also taking amiodarone.

MARK BARNETT:

When patients start taking simvastatin or their dose is increased, they should be told about the risks of rhabdomyolysis and advice to promptly report any unexplained muscle pain, tenderness, or weakness.

Thank you for listening to Patient Safety News provided by the FDA. To hear pod casts of this show and other, visit us at www.reachmd.com, register with promo code "radio."

Hello, this is Dr. Clifford Callahan at Middlesex Hospital in Connecticut. You are listening to ReachMD XM160, The Channel for Medical Professionals.