

Science AMA Series: I'm Dr. Steven Q. Simpson, a pulmonologist, intensivist, member of the board of the American College of Chest Physicians and a sepsis researcher and expert. AMA!

Steven_{Simpson}¹ and r/ScienceAMAs¹

¹Affiliation not available

April 17, 2023

Abstract

My name is Dr. Steven Q. Simpson. I'm a Professor of Medicine and Interim Director of the Division of Pulmonary and Critical Care Medicine at the University of Kansas. As a sepsis and quality improvement researcher and educator, I've spent decades training hospital providers across the country to aggressively treat sepsis in all its forms. I'm also a member of the board of the American College of Chest Physicians, an organization representing 19,000+ clinicians practicing pulmonary, critical care and sleep medicine. Sepsis is the body's response to a life-threatening infection, most commonly caused by a bacterial infection, but it can also be caused by serious fungal or viral infections. In basic terms, your body goes into overdrive to fight an infection and ends up damaging itself. Sepsis does not just happen on its own, meaning a prior infection—like pneumonia or a urinary tract infection, is present in all cases. Sepsis can lead to tissue damage, organ failure and death in many cases. Sepsis strikes more than a million Americans annually and frequently impacts those who are over age 65 or less than 1 year, have a weakened immune system or chronic medical conditions like diabetes. However, it is not uncommon for normal, healthy adults and children to be affected when a seemingly simple infection progresses to severe sepsis. One of the main challenges of sepsis is diagnosis—often, by the time physicians become aware something is wrong, the disease may be advanced. Sepsis signs and symptoms are not very specific and may at first seem like a simple viral infection, which results in delays in patients seeking medical attention. There is no specific laboratory test that can diagnose sepsis or severe sepsis. Instead, physicians must be astute to recognize the signs and symptoms, recognize the infection and know when the combination is potentially deadly. Early recognition is key to patient survival; delays in delivering relatively simple treatments, such as antibiotics and IV fluids, are associated with increased mortality. Recently, a consensus statement was released that proposes to redefine the diagnostic criteria of sepsis, and that would eliminate the concept of the systemic inflammatory response syndrome (SIRS). The proposed syndrome would rely on known or suspected infection with a change in sequential organ failure assessment (SOFA) score. Shortly after the new guidelines were published, I released my rebuttal in the journal CHEST, New Sepsis Guidelines: A Change We Should Not Make (<http://bit.ly/1M8eKYZ>), expressing concern that many physicians and specialties have shared—widespread application of this new definition could cost patient lives, and it should not be adopted. Please feel free to ask about anything related to sepsis, critical care, or pulmonary medicine. I will return at 12 p.m. CST to answer your questions. Conflict of Interest Disclosure: My thoughts and opinions are my own. I don't have financial relationships with anyone—except my wife, who is a pediatric ENT surgeon and gets paid more than me. She takes everything I make, anyway, and makes sure that I don't spend it all. She gets her sepsis info from me, not vice versa, and she's pretty good at diagnosing it. For more information on my research: CHEST 2016: Did We Need New Definitions for Sepsis? (<http://bit.ly/2if7oW3>) YouTube: Surviving Sepsis: The Value of Real Time Surveillance (<http://bit.ly/2hV4mWl>) I'll be back at 1 PM EST to answer your questions! AMA!

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STEVEN_SIMPSON [R/SCIENCE](#)

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For more information on my research:

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What are your thoughts on the use of CVP and/or PAP as indicators of fluid responsiveness? Are your resuscitation targets in line with national protocols? What, specifically, is your choice of resuscitation fluid? Have you seen data on the use balanced salt solutions (e.g. LR) vs NS for resuscitation? What do you think? What types of monitoring do you use in your resuscitation protocols? Do you see a role for noninvasive monitoring (e.g. Edwards ClearSight and the like)? Do you follow all of the SCCM sepsis protocols? Do you disagree with anything else, aside from SIRS/SOFA?

[kmartparty](#)

OK, I don't know what happened to my first answer, but here we go again. Possibly not as longwinded this time. And I may take breaks and answer in parts.

CVP - I am 100% convinced that CVP does not give any consistent information about intravascular volume, stroke volume, cardiac output, or any other parameter that is of use to patients or to us. I much prefer measuring stroke volume and its responsiveness to either a passive leg raise or to a 500 mL bolus of crystalloid. I personally do this with either a non-invasive cardiac output monitor or with arterial pulse contour analysis.

Having said that, there is plenty of data that compliance with protocol that uses 30 mL/kg as the initial bolus and "optimizes" CVP between 8 and 12 mm Hg results in saved lives. This from the Surviving Sepsis Campaigns 30,000 patient database, and from Intermountain Healthcare's data. Additionally, there is more recent data from Vinnie Liu and Kaiser Permanente of Northern CA. They were interested in knowing what to do with fluids in the patient with a lactate >2 but <4. They developed a protocol for 30 mL/kg in these patients as part of an overall bundle and compared before and after results. In patients who were most compliant with the bundle there was a significant mortality improvement. Moreover, and this part I love, the lion's share of improvement was not seen in the "healthy" septic patient but in patients with CHF, a population that is vulnerable, because the average doc does not want to give them fluid.

With all this data supporting it, how does one provide better info about different fluid resuscitation protocols. My colleagues and I at KU are working hard on generating the data, as are others in multi-center trials. But we don't have it, yet. For the time being, in patients who are not on a protocol, we should stick to what we have the best evidence about. Currently, that is 30 mL/kg and CVP measurements. Like it or not, I challenge you to show me the data that another approach works better. As far as what we are attempting, for now, we use 500 mL crystalloid boluses or passive leg raise, tracking by either non-invasive cardiac output monitor (mostly) or by arterial pulse contour analysis. We bolus, at present, until there is no further increase in stroke volume. We are thinking that one may not have to go all the way to that point before initiating pressors. But one protocol at a time.

How can we better educate doctors and patients about the symptoms of sepsis and when to begin/seek aggressive treatments? I realize you are advocating against the redefinition. But it seems as though even before that timely diagnosis was an issue. Do you think a public health campaign would be effective?

And do you have recommendations for non-medical professionals about when to call their doctor?

Edit: Following the link from the AMA guest, here is the [acronym to remember for identifying sepsis](#):

- S - Shivering, fever, or very cold**
- E - Extreme pain or general discomfort ("worst ever")**
- P - Pale or discolored skin**
- S - Sleepy, difficult to rouse, confused**
- I - "I feel like I might die"**
- S - Short of breath**

For the pediatric version, [see here](#). The major difference is: a rash that doesn't disappear when you press it, very fast breathing, and has a convulsion. For under 5 look for a kid who is: not eating, vomiting, hasn't peed in 12 hours.

In all cases, they recommend calling the doctor or 911 immediately and specifically saying you are concerned about sepsis.

[firedrops](#)

Nice going, firedrops! Keep that up on ALL of your social media outlets!

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[firedrops](#)

I absolutely believe that communication to the public is crucial. Just as the public has become aware of signs and symptoms of heart attack and stroke, they need to be aware of the signs, symptoms, and risk factors of sepsis and severe sepsis. Sepsis Alliance (www.sepsis.org) is a national organization that has as its focus the education of EVERYONE that sepsis is a medical emergency. SA also focuses on helping sepsis survivors cope with the aftermath of having had the illness. To do this work takes dollars. I know that we just did our year end giving, but take a minute to go to SA and think about donating \$5, \$10, \$20 or \$20 million. It could save someone's life.

Proviso: I am also a member of the board of Sepsis Alliance. I am DEFINITELY biased about this, but everyone needs to suspect it when they might have sepsis.

How accurate is the procalcitonin level in the body at predicting sepsis?

[bitcheslovebutter](#)

There are a few questions about procalcitonin up and down the page. I'll answer this one and see where it takes us. Procalcitonin is a pro-hormone or precursor to the hormone that controls calcium levels in the body. For reasons that are not perfectly clear to me (and I don't think to anyone else), it is produced in excess by numerous tissues in the body in the presence of (mostly) bacterial infection. It

has been proposed as a candidate to help identify sepsis and/or severe sepsis. My thoughts on this are that when a person has sepsis, the probability of antibiotics being able to control the condition go down with time. Some facilities have STAT procalcitonin (PCT) available. Most do not. When one sees a person with criteria for sepsis or severe sepsis, they should not delay giving antibiotics while waiting for the PCT level. If the source of infection is obviously pneumonia, or obviously cellulitis, or obviously pyelonephritis, I would not send a PCT, at all. If you're scratching your head, believe the person is at risk of infection, but can't put your finger on it, I'd give the antibiotics and send the PCT, so you can stop them if infection doesn't pan out.

The difficult thing about PCT is that it is, itself, not perfectly specific. When it's really high, or when it's zero, then it's great. What seems to happen a lot is that the resident sends it, and it comes back just a little above normal. Not normal, but not CLEARLY quite high. And the patient is clearly sick. Then we are literally right back where we started. In those cases, I keep the antibiotics going and keep looking to see if I'm wrong about infection.

What do you find to be more accurate, procalcitonin or lactic acid levels at predicting/diagnosing sepsis? I have worked in facilities that use each as a diagnostic tool.

[namkrav](#)

Lactic acid or lactate levels are not at all diagnostic of sepsis. Lactate can be increased for 2 reasons in a sick patient. The most ominous reason is poor tissue perfusion and/or oxygenation. Oxygen starved tissues produce lactate. The second one doesn't sound so bad, but it might be, nonetheless. High circulating catecholamine levels, whether endogenous or exogenous, stimulate beta receptors and cause non-hypoxic lactate generation. Either circumstance seems to portend a worse prognosis in sepsis, so it may not matter what the source is. But increased lactate is present in all forms of shock and can be associated with overdoses of metformin or continuous beta-agonist inhalation in asthma attacks. Lactate is not specific to severe sepsis, but is a prognostic factor.

More on procalcitonin farther down the page.

What is the most reliable criteria you can recommend for sepsis recognition in the prehospital setting so a sepsis alert can be issued as early as possible? Most EMS systems in our area have end-tidal capnography available as a diagnostic tool. How sensitive/specific and valuable do you feel capnometry is for sepsis screening?

[ShockdaMonkee](#)

There are a couple of questions about pre-hospital care down the page. This is important, because the data we have show that approximately half of patients who come to ERs with sepsis or severe sepsis are brought by ambulance. EMTs and Paramedics should be very familiar with the criteria for recognizing sepsis and with its treatments. In my own state, I am training pre-hospital personnel to recognize and to initiate whichever treatments their medical director will approve and protocolize for them. Our folks often are on runs that have them an hour or more from the nearest hospital, and delays in treatment can be deadly. I think that sometimes the diagnosis is subtle, even for experts, but there are plenty of times when it's obvious to everyone what is going on, and pre-hospital personnel can make a difference with their interventions.

I think the best criteria for recognition remain the standard criteria for sepsis, severe sepsis, and septic shock, as proposed by the first and second sepsis consensus conferences. However, it is VERY important to hone in on infection and be certain or very confident that infection is present. Having said all that, if you see qSOFA criteria, you had better move fast, because we know that they precede death

or a move to the ICU for a prolonged stay by only a few hours. About 5.

Capnography. First, the tool is neither sensitive, nor specific for sepsis. (So what else is new, right?) I will grant you that PaCO₂ < 32 mm Hg is used as an indicator for hyperventilation, either along with or in lieu of respiratory rate. I think it's great if it all adds up. DO NOT FORGET that this relies on FIRST having an idea of whether there is an infection. Hyperventilation does not mean sepsis, and the only information you get from your capnography is whether there is hyperventilation. It MUST be integrated with the overall picture.

HYPOverventilation is not particularly associated with sepsis, but it could certainly be present. Think about the IV drug abuser who has aspirated and has pneumonia, but is still injecting one CNS depressant or another, or taking them orally. You could have a septic patient with hypoventilation. Easily.

When do we make the next great leap and start to manipulate the inflammatory response itself? Fluid resuscitation helps, antimicrobials are obviously important, we don't have perfect evidence for pressor support, and seem to be making strides in managing the collateral damage of simply being admitted to a unit...but it's not always enough. Willing to speculate what that might look like...maybe a -mab/-mib for sepsis??

[NickRiviera_MD](#)

I don't know how old you are Dr. Riviera, but for the better part of two decades, from the late 80's to into the 2000's, this modulation of inflammatory response was the principal focus of our attention. We failed abjectly, and I think it's because we always took a single-pronged approach to a multi-pronged illness. I.e. the inflammatory response is far too complex for us to pick one component of it and think we are going to eradicate the entire inflammatory milieu. When I was young and naive, I thought maybe we could via anti-TNF. In fact, when I began researching TNF there were 6 whole articles in the literature, and we just did a search and found more than 200,000. That tells you how old I am. We had high hopes for TNF as the target of a "silver bullet". As you are probably aware, the next big thing was coagulation, and this has mostly died, though not completely. A soluble tranexamic acid molecule is in use in Japan. There are some other promising things, but I've been sworn to secrecy. If I told you, I'd have to send the Russian election hackers to knock out everyone's internet connections.

How do you feel about levophed or other pressors for sepsis in prehospital environment? Should we be focusing on fluids alone or use pressors after a certain point? I work for an EMS service in rural Texas with transport times around an hour. Our current protocol is levophed if systolic is still below 70 after 2 liters NaCl. We give 4 - 12 mcg/min to maintain systolic of 100.

[Aggielopmedic](#)

Norepinephrine in the field: interesting idea. You must have a progressive medical director. Be careful to use a large bore IV in a large vein (I'm sure you do). Your strategy looks reasonable, and I know that people like Chris Seymour are working on developing data about pre-hospital treatment and transport, but they aren't out, yet.

I don't know if they're looking at vasopressor administration.

BTW, I often say that it doesn't matter a whit how I feel about a medical/scientific issue. It matters what I think and what the data can show. I don't know of any data on NE in the fields, but I'd certainly rather see that than a patient who dies from shock because we did not treat it.

What is the link between sepsis and diabetes? What can diabetic patients do to better prevent sepsis?

[ezzyrd](#)

Diabetes predisposes to infection. Most diabetic patients know about careful attention to foot care to prevent infection. Any infection can lead to severe sepsis. Diabetics should maintain as tight glucose control as they can, do their foot care, be careful around other individuals who may be infected, and report ANY symptoms that might be infectious to their doctor. Diabetics are pre-disposed to peri-rectal abscesses that may not cause too many symptoms until they cause sepsis or keto-acidosis or that may be VERY painful.

Is there a link between sepsis and chemotherapy? Should cancer patients be closely monitored for sepsis risk (preventative antibiotics provided prior to treatments)?

I'm asking this question after seeing a loved one recently pass away after developing sepsis a few days after their first chemo treatment for lung cancer.

[PetTheRock](#)

Absolutely. Chemotherapy often induces neutropenia, which means a lack of the first line white cells that fight infection. Even when there is not an absolute neutropenia, immunity may be impaired. This is a setup for infection and for rapidly progressing severe sepsis and septic shock. Sepsis is the number one killer of patients with cancer, accounting for just under 50% of all cancer deaths.

I almost died of sepsis (as well as a number of things) earlier this year by contracting H1N1 and Influenza B. It was the perfect storm leading to ARDS. I was intubated and put under for 8 days while they proned me. In your experience, have you seen many cases like mine?

[didenimvenom](#)

Yes, we see numerous cases similar to this every year. It does not have to be H1N1, either. H1N1 has a propensity for hitting younger, healthy people and hitting them hard, where other strains tend to focus on older patients or those with impaired immunity. But influenza, itself can cause ARDS, and influenza can lead to secondary bacterial pneumonia, which also causes severe sepsis and potentially ARDS.

I am currently a medical student, but my undergraduate background is in history, so I am curious about the non-scientific reasons that these sorts of changes get made.

Why did they decide to switch from the SIRS to the SOFA criteria? What kind of politics were driving that decision from behind the scenes?

[exmocaptainmoroni](#)

Well, I think it's some sort of nefarious plot that involves the CIA, the NSA (who, by the way, is reading this post), and maybe some rum runners from Cuba, now that relations are better.

Actually, I just don't know. What I believe that that there are different priorities for different people. You may notice that the new criteria would be more specific and better for interventional trials. The old criteria are clearly geared from the viewpoint of patients, so that we find sepsis early. It probably stems from different people having different reasons for how they approach sepsis. Now that I read that, it's pretty much a motherhood and apple pie statement, but I'm afraid that's what I've got. And I want to say that, although the Sepsis-3 folks and I disagree, they are colleagues and friends that I respect a

great deal.

What can paramedics do in to assist with the diagnosis of sepsis?

Are there any specific prehospital treatments that would be beneficial to the patients overall health?

Any tips or tricks that can help a paramedic who is thinking infection/sepsis that you know?

Thank you for doing this AMA, quick background. I work on ambulance in rural Manitoba, Canada and my transport times can be anywhere from 40 minutes to 2 hours. Our patients also have a lengthy travel time to be seen by a primary care physician so it does result in less visits and higher cases of uncontrolled diseases.

[OhShitSpiders](#)

Thanks, spiderman. If you don't mind taking a spin up and down, I have answered several questions about pre-hospital care. It is also crucial in my own state, where I am continually training EMS personnel to recognize and initiate treatment. Many of our patients' ambulance rides are of similar duration to yours. Manitoba is just Kansas, moved farther north. Or, I suppose you will say, vice versa!

As a layman who has trouble understanding 75% of the words in this AMA, it seems the symptoms like the ones [/u/firedrops](#) give are a lot like getting hit by the flu hard. My question is how do I know what I have is not something like a bad case of the flu and really sepsis? I would hate to have to wait in the clinic for 5+ hours to be told I have the flu, but also getting sepsis and thinking I can tough it out.

[kevpopo](#)

This is a really good question, and I appreciate it. So, you are so right, in terms of what it may feel like. Some things that could help you know whether you are REALLY sick vs. feeling sick. If you know how to take your pulse, do it. If it's above 90 it's concerning. If it's above 100 it's more so. If it's above 120 you may need ICU level care. If you have a blood pressure monitor at home use it. If your systolic pressure (the upper number) is less than 100 mm Hg, you may need ICU level care. If your significant other believes you are confused about where you are or what is going on, they should get you to the emergency room. If you are having fevers, feeling bad, and there is a focus of intense pain in some part of your body, so that you can't even touch it you should get to the ER. If you are really short of breath and if you are breathing more than 20 times per minute, then you should go to the ER. The flu knocks you on your butt and puts you in bed feeling horrible. If a real influenza does any of these things to you, then you likely have influenza-induced sepsis, which is a real thing. You need medical care, not lying in bed at home. And, like I said elsewhere, influenza not uncommonly leads to bacterial pneumonia, and if the pneumonia leads to sepsis you could experience these things. If you have any of these things, get to the emergency room and ask the doctor if this is sepsis.

Procalcitonin... Thoughts?

[karate134](#)

Hey, I am losing track of names, but I answered this for another redditor somewhere on this page. If you don't mind, looking up and down, you will find it.

You should cross post this to [r/nursing](#) and [r/respiratorytherapy](#)

[mrscartoon](#)

GREAT idea. Nurses are with our patients 24 hours a day, and need to know how to recognize sepsis. Our RTs screen every patient who is receiving a respiratory treatment, and they do a great job of helping us find septic patients on the hospital wards. Our nurses also screen every patient, every shift, every day. GO NURSES! GO RTs! You guys rock!

Hello! 2nd year medical student here. Thanks for doing this AMA!

One of the questions that I found interesting in studying sepsis was learning about the debate over its etiology. As my professor explained it, there are two competing models of sepsis.

In one view, sepsis is caused when the infection overcomes the body's immune response to it. In the other view, sepsis occurs when the body's own inflammatory response becomes worse than the the infection itself.

How do you view this debate? Does it even make sense to have one set of criteria for a nebulous condition called "sepsis" that might actually be two opposing processes? Would there be a good way to separate these etiologies based on symptoms?

[exmocaptainmoroni](#)

Well, as I see it, these are not COMPETING theories. They are both part of a complete picture of sepsis. As I'm sure you are learning, most of life is not black and white, either/or, 1 or 0. What we understand is that a high pathogen load leads to highly active innate immunity, which should lead to acquired immunity (antibodies to the offending agent, T-cell memory, and all the other things you just learned about). Normally, you need to turn on the soldiers to clean out the enemy microbes, but then you need to call them back so there isn't too much collateral damage. With sepsis the soldiers are really angry and the signal to stop after antibiotics and so forth is equally revved up. Now what? There is collateral damage from the initial violent innate reaction, but if things don't go right there is also an immune paralysis. We think we know now that in most patients who die from sepsis the immune paralysis is predominant. Does this then mean that the microbes have won? Quite likely. Collateral damage due to innate reaction, then immune paralysis so that the damage in the name of staying alive turns out to have been in vain. OK, so I have anthropomorphized something that is a cellular reaction (I don't know - does anthropomorphized apply if it actually IS human? I think you get what I mean, though), but I really don't see these as competing theories. They both happen. One or another gains the edge. Patient lives or dies. Either way, our rapid aggressive response is important. The way I see it, not everyone with severe sepsis dies. Sometimes they are just lucky. In fact, at least half the time they are just lucky. It is our job as physicians to shift the equation more toward skill, more toward an organized, aggressive approach and less toward luck.

As a med student trying to decide on a specialty, why did you pick IM/pulmonology/intensive care?

Also, why academia vs community/private practice? (Or is doing a bit of both possible?)

[snowshoos](#)

Well, first because I like to use my brain. Take that surgeons out there! If you want rebuttle, get your own AMA!

Of course, I'm teasing my surgery colleagues. As you may have seen up above, I'm married to a surgeon who is one of the smartest people I know.

I love the thought processes and the piecing together of subtle signs and symptoms that IM folks tend

to like. Not that I pride myself on esoteric diagnoses; there are a lot of smarter IMs out there. But I like it. One of my adages for students is "If you let your patients talk, they will tell you what's wrong with them." I like talking and asking great questions and interacting. That's the IM part.

Here's what I considered in the mix along the way. Cardiology, specifically EP, until I figured out that you had to wade through a hell of a lot of chest pain to get to the EP fellowship. Yuck. Oncology, because I love palliative care; but I hated the conferences about what chemo for what cancer. So, it fit emotionally but not intellectually. Nephrology, because I loved and still do love fluid and electrolytes. Couldn't care less about dialysis. PCCM. I deal on some level with all of those things I just mentioned. In fact, as an intensivist I have the privilege of caring for the worst derangements of every possible organ. And every day is like a box of chocolates; you never know what you're going to get. Also, I love, love, love respiratory and cardiac physiology.

Academic vs. community/private practice. Some people can cross these boundaries, but not me. I have done both, and I find both of them to be consuming. In fact, even now, when I'm in the ICU or on the consult service, I find that I get very little of my academic work done (outside of bedside teaching, which is majorly important and which I love, but actually doesn't get you any academic brownie points). I love inquiry. I love thinking hard and testing questions. I love inspiring that love in the young folks I work with. That's why I'm in academic medicine.

As someone who has been outspoken against redefining Sepsis, how do you justify the continued use of SIRS when as physicians we all know it is incredibly sensitive and not remotely specific (i.e., if you briskly run up a flight of steps, you meet SIRS criteria). Also, knowing the prior 2 sepsis definitions were largely based on expert opinion isn't a definition that takes some data into account better than definitions which do not?

[anavrin0001](#)

There are a couple of questions regarding SIRS, sensitivity, and basing diagnosis on data rather than expert opinion.

Let me start with the non-specificity of SIRS, which is a common criticism. Straight from the keyboard of Drs. Charlie Sprung, who was a participant in the first sepsis consensus conference in 1991, "The original consensus conference was set up for several purposes. Among others, one was for uniform definitions and another was to try to diagnose septic patients who were being missed and if possible at the earliest possible stage." There is no question that SIRS helps to fill this bill.

Secondly, we all readily admit that SIRS is non-specific. What most of the people who criticize SIRS forget, however, is that we are not just looking for SIRS, we are looking for SIRS in the setting of infection. These are two different things. If one has a good idea of the infection, whether it is pneumonia, cellulitis, peritonitis, etc., then the SIRS has a different meaning than simply finding a fever and tachycardia. Two other ways of saying that – the infection gives the SIRS its specificity, and Bayes' theorem dictates that the likelihood of having the disease of interest is principally dependent upon the prior probability and the sensitivity of the test. In this case, SIRS is the test. SIRS in all comers, e.g. stair climbers, people receiving immunotherapy or even just a vaccine, people watching a horror movie, etc. is worthless. However, give me reason to believe that a patient has an infection – cough, chest pain, sputum (phlegm), maybe with blood streaks, bronchial breath sounds when I listen, as signs of pneumonia, for example – and now SIRS has more specificity. Now SIRS tells me that there is a risk of mortality associated with this infection; historically, 7 – 9%. In some studies higher. How high does the associated mortality need to be in order for us to pay attention? This is certainly high enough for me.

A critique of the first two sepsis consensus conferences is that the criteria they proposed are based on expert opinion, rather than data. I have two things to say about that. First, is it really expert opinion that fever, tachycardia, tachypnea, and high white blood count are associated with severe infection? Does

anyone believe that is truly only expert opinion? Were there any research studies that demonstrated that before the first consensus conference? Yes. Search for the papers of Roger Bone and Bob Balk from the mid to late 80's. But even if there had not been such studies, where would anyone get the notion that these things are NOT associated with most severe infections? Since the times of Pasteur and Koch, when we proved that infections exist and that they are due to micro-organisms, we have known of the association of these signs and symptoms with serious infection. To suggest that there are not data supporting the association is, to be plain, silly. Now, look for the data of Richard Wenzel from the mid-90's (after the first consensus conference). They clearly demonstrate an association of SIRS with mortality, especially in the presence of infection. Now look at the scores of clinical trials for pharmacologic and other therapies of sepsis that have used these criteria. Were there no mortalities in these trials? What sort of data do you want? Finally, using these criteria, the Surviving Sepsis Campaign has demonstrated that it doesn't take clinical trialists to recognize sepsis or severe sepsis. They demonstrate that average docs can both recognize and treat aggressively and save lives. Save lives. The bottom line. Period.

By the way, I have zero problem with defining sepsis as the Sepsis-3 consensus authors have done, "life threatening organ dysfunction due to a dysregulated host response to infection", as long as we are talking about an overall entity. Our definitions from the first two sepsis conferences, however, are actually more complete. The new sepsis diagnostic criteria are functionally the same (albeit, based on slightly different markers of organ dysfunction) as what we call severe sepsis under our original definitions. The original definitions, in other words, already accounted for what the Sepsis-3 authors now want to call "sepsis". Our previous framework of infection, infection with SIRS (sepsis), infection with SIRS and organ dysfunction (severe sepsis), and sepsis with shock (septic shock) elegantly demonstrates to all that these findings are on a spectrum of disease, that patients with "milder" findings right now have potential to have more severe findings soon, if we do not intervene. The new framework seeks to say that infection + SIRS is really "just infection" (direct quote). That is dismissive of a condition that, as I said above, kills people, and that has high potential for progressing to organ dysfunction, which kills even more people, and to septic shock, which kills even more people.

Let me ask you all a question. As potential patients, which ALL of you are, when you come to my emergency department feeling crummy and having SIRS, do you want me to quickly give you antibiotics or do you want me to wait until you have organ dysfunction (by any of these criteria) before I do so? Remember – the mortality associated with "just" infection and SIRS is 7% to 9%. I, personally, do not indulge in ANY behaviors where I take that kind of chance of dying each time I do it. With the possible exception of failing to pick my socks up off the bedroom floor. My wife is going to kill me, someday.

With the legalization of marijuana more and more people are taking up smoking. As a pulmonary specialist how do you compare tobacco smoke vs cannabis ? Undoubtedly both are bad, but I seldom hear marijuana joints/bongs etc, being tied directly to diseases like cancer and emphysema ? I know it's bad for your lungs, but from your POV how bad is it ?

[darth6ixious](#)

I can say this. We thought for a long time that cannabis smoke might be worse than cigarette smoke for inducing emphysema. The thought was that cannabis smoke is purposefully held in the lungs for prolonged periods, so it had more time per joint to do its thing. But the more recent data show that it is not worse. It is about the same. And of course it depends upon how often you smoke, etc. So, if you wouldn't smoke cigarettes, because of the health effects, you probably shouldn't smoke pot. I don't know anything at all about edibles or mists. Sorry.

I am a total layman, here.

I ended up being referred to ER who diagnosed me as septic, presenting with tachycardia and fever and positive strep throat swab. Previously my (and I think in large part the general public) belief was sepsis had to be an infiltration of bacteria into the bloodstream. Is this not always the case? I have recently read that sepsis = SIRS criteria + infection, which does not translate exactly into sepsis = bloodstream infection, to me.

In other words, is sepsis the immune response generally (but not always) associated with bloodstream infections, not technically the infection itself? Can you be septic but have no bacteria in your blood?

[Bluekoolaide](#)

Dude, you nailed that one on the head! In cases not just of sepsis, but of even septic shock, only 30% to 50% will have positive blood cultures. We consider bacteremia an infection that can cause sepsis, but by no means the sine qua non of the condition. BUT, you CANNOT be septic without infection.

I know I'm probably way too late, but do you (or anyone else) have an opinion on how useful serial BNP's are as a measure of fluid overload?

[morningsunbeer](#)

I think it's an interesting notion that I would like to see data for. The BNP is produced in response to either right or left ventricular stretch. It seems like it might be useful, but I have not seen any studies that would support it. Anyone else out there seen anything along these lines?

Hope you might be able to answer two questions please, but if not, just the first would be amazing.

I recently have quit smoking and am going to do my best not to pick it up again. I've been thinking about ecigarettes as an interim but perhaps an ongoing replacement, how bad are using these for your lungs?

I've been smoking largely unfiltered cannabis joints nightly for about 5 years (quit that too), and maybe once or twice weekly for a few years before. How damaged are my lungs? I have no symptoms of any COPD as far as I can tell but imagine they aren't healthy. I did one of those breath expulsion tests a year or so ago and actually scored better than my nonsmoking friend, strange.

Thank you so much, and all the best.

[drboneswildride](#)

We don't know just yet what the long term effects of e-cigs will be. But we know that there are a lot of chemicals in that vape that could be injurious to you. I know they make it seem as if there's pure nicotine in there, but that isn't the case. These are often promoted as a way to stop smoking, but they actually seem more to be a "gateway drug". If you need a nicotine supplement for awhile, I would recommend patches or gum.

Not everyone gets emphysema or obstructive lung disease. You may not have any. Yet. It's always the best policy to stop smoking. There's not only the risk of emphysema, but of lung cancer.

Paramedic here, how do you feel about prehospital antibiotics in the treatment of sepsis? I have heard it purposed as something that might begin in the future. Has not been started were I am at on the east coast of Canada

[countryhick7](#)

Antibiotics in the field: I highly support this, if your medical director and your hospitals will let you do it. Some ER docs get persnickety about allowing these. But I think they can be life saving, especially when it's a pretty far distance to the ER from the patient's home. That happens a lot in Kansas. Also see some of my other answers to pre-hospital questions scattered through here.

Dr. Simpson,

Thanks for the AMA! I'm certain I've read a few of your publications! How cool! I have a quick question for you. I'm presently an ICU nurse and previously worked in ER.

It wasn't uncommon for some paramedic buddies of mine to bring me a capnography print out of a patient suspected of sepsis during transport.

I've tried to advocate for capnography to be added to our sepsis alert protocol.

What are your thoughts on capnography as tool for early detection of sepsis? If you support the use of capnography for sepsis screening, where along the continuum do you recommend utilizing capnography?

Thank you for your time Dr. Simpson

[Azrolicious](#)

Hi Azro, I've actually answered this question elsewhere on the page. I can't remember who asked the question, but it's on here. I hope you don't mind looking.

There is a concept called slow sepsis where bacteria slowly and constantly makes its way into the blood stream over a long period of time without any noticeable symptoms. Some speculate that this could be the reason for the correlation of poor dental health and mortality. Do you believe slow sepsis exists and if so, how prevalent do you think it is?

[SkylineCS](#)

I have always thought that there is plausibility to this concept, but I do not know of data. That doesn't mean there aren't any, but I spend a lot more time worrying about immediately life threatening disease. I'm sorry I can't help.

Do children generally have the same symptoms of sepsis that adults have?

Also, I have 2 kids diagnosed with asthma. Are there any steps that I can take as a parent that aren't generally well known to help them?

[Froggyloofa](#)

Let them have all the candy they want. Tell them they can stay up all night watching movies and not go to school. Teach them it's OK not to pay taxes or have any obligations to their fellow citizens. Wait, where am I going with this?!

I don't think there are any special asthma things that your doctor hasn't likely told you. If kids are allergic, avoid the offending things they are allergic to. Use the bronchodilators as indicated. Good sleep, good nutrition. If their asthma is unrelenting see a specialist. If they are small children some of

this may not be asthma, but tiny airways that wheeze because they are tiny. But it's not possible to tell that up front, so they should use their asthma meds as directed.

So if I have a child, what symptoms should make me worried and make the pediatrician suspect sepsis?

[barhanita](#)

I'm going to send you to Sepsis Alliance, www.sepsis.org. They have links for childrens' sepsis. I hesitate to say much, because I am not a pediatrician. I will try to get my friend Chris Carroll to comment. Or we can have another AMA on childrens' sepsis.

What treatments do you have available now compared to 10 or 20 years ago? Is this an active target for pharmaceutical companies?

[hypno_tode](#)

We have a few extra antibiotics. We have a more certain knowledge that severe sepsis is an emergency that should be viewed as seriously as a heart attack or a stroke. But our attempts to treat the illness with specific agents have been failures. Pharmaceutical companies have tested many new drugs and poured billions down the drain in the end. They look so promising in the animal studies, and there are great theories about why they should work. Then they don't.

Is there an organ or system that sepsis generally targets?

[silverhill](#)

Nice question. Yes, the lungs and kidneys are the two most commonly involved organs. However, any organ can be affected, and when we're examining someone to assess whether they have sepsis, we also focus on brain, heart and blood vessels, liver, and blood clotting mechanisms.

Does marijuana damage the lungs?

[Testicularwart](#)

Yes.

Why does meningitis often lead to sepsis and amputation ?

I had meningitis as a baby and was not diagnosed for several days. Why do I still have all my limbs ?

[blablabla1984](#)

Obviously, I know nothing about your case, but here is some general knowledge. There are two main bugs that cause meningitis - not the only ones, but the most common ones. One is called *Neisseria meningitidis* or meningococcus, for short. One often develops meningitis and bacteremia (bacteria in the blood stream) with this bug. It has a propensity for causing severe DIC, which stands for disseminated intravascular coagulation, a condition in which blood clots within your vessels and cuts off the blood supply. This is a mechanism of all sepsis, but seems more prominent in meningococcal sepsis and meningitis. It often results in fingers and toes requiring amputation. The other common bug

is *Streptococcus pneumoniae*, also known as pneumococcus. It causes severe sepsis, but it is less likely to result in amputations. This could explain it.

Dr. Simpson, what can you tell us about Capricor Therapeutics and the ALLSTAR stem cell trials. Are we in an era where plaque may actually be removed from the walls of arteries? If so, why isn't it a bigger deal? Thanks and thanks for doing this!!

[charleycoyote](#)

You know, I'm really sorry, but I don't have any more info about that particular thing.

My question is what is the link between sepsis and when to begin/seek aggressive treatments?

[batista2000](#)

I'm not 100% sure that I understand the question, but I would respond this way. If you have a infection that is causing you high fevers, shortness of breath, intense pain, mottling or purple coloration or overly pale skin, you should get to a doctor, preferably an emergency room. You should ask the doctor - "Is this sepsis?". That forces her/him to think of it and make sure it isn't. When it IS sepsis, aggressive treatment should be started immediately.

Do you currently (or have you in the past) worked with Health Catalyst on Sepsis?

[thistlegypsy](#)

I do not, and I have not. But from what I know about them, I like what they're up to.

I actually just went to the ER a few days ago with chest pains on the lower right side of my chest that had been happening for the better part of a week. More than half of a deep breath would cause a sharp (scale of 1-10: 6) pain. Doctors have diagnosed it as 'costochondritis', but in the scans taken, have told me that I have a small (less than an inch) nodule on my lung. They couldn't tell me much more than that, and I've got a follow up scheduled, but I was wondering if it were possible that the nodule is the cause of the pain? How concerned about a nodule should I be? Answered below.

Also, as a second question if I may, as someone with severe psoriasis on my lower legs (and taking remicaid via infusion), do you have any thoughts on the possibility of psoriasis being prevalent in people with some type of long term or undiagnosed infection (bad oral health or something along those lines)? My thought being it causes the immune system to go into over drive or become overly stressed / taxed and behaves erratically?

Hope those made sense, and thank you for your time!

[motherfacker](#)

This is an interesting question, because Remicaid is an anti-TNF drug and TNF alpha is an important component of the pathophysiology of sepsis. Anti-TNF therapies did not work in established sepsis for reasons that I've outlined in another answer. However, blocking TNF up front theoretically might help to prevent sepsis. I don't know any data about that, but I'm curious now. The drawback is that infliximab does not prevent infection; in fact there is an association with developing tuberculosis, precisely because of its anti-TNF actions. I'm sure your doctor has discussed that risk with you.

What are possible long term effects in infants who have had sepsis? Are there any steps caregivers or doctors can do for patients after onset of sepsis or is treatment more acute?

[SkiHole](#)

I'll tell you, I'm a grownups' doctor. I am mostly ignorant on this issue. Now I put my friend Chris Carroll to the test - Chris are you looking at this at all? Chris is my good friend and Assoc. Prof at UConn. When I need to know critical care info about kids, I go to him. if he doesn't answer naturally, I'll first give him some noogies, and then Tweet him to see if he has an answer.

Hello Dr. Simpson. My colleagues and I work on building early warning systems for sepsis using predictive models similar to SOFA and NEWS, and making it a part of our EPIC system. What do you envision as the next steps in the evolution of such models?

[bharathbunny](#)

If you Google me, you'll see that I have done some work along those lines, myself. The key Achilles heel of all early warning systems for sepsis right now is that it is difficult to tell whether a patient has a known or suspected infection, so they tend to trigger on non-specific things like SIRS, even NEWS and MEWS are non-specific (which isn't necessarily bad - when I read that NEWS in the setting of infection has better predictive ability than either qSOFA or SIRS I just realized that if it's a good predictor of overall badness, it should be a predictor of infection-induced badness, since that's a subset of overall; however, if you're looking for sepsis or severe sepsis you need a way to inform the test to make it more specific; see some of my other answers here, as well). As you may see in some of my other answers, it is the knowledge that there is an infection that informs the utility of any of these scores. If docs would immediately fill out the problem list in their EHR, this would be an easy problem to fix, because those are searchable, discrete data fields. But they don't and they won't. Longwinded approach to this - I think we need programs with natural language processing, so that when the doc writes a note that says "pneumonia" or the radiologist reads the CXR as pneumonia, the background search for SIRS and organ dysfunction triggers a flag. Not before, not after. If you're actually talking about predicting when sepsis is coming, I have some thoughts on that, but the best thought on that right now come from Matt Churpek at the U of Chicago. At least that's who I think is leading the pack right now.

Have you or anyone else developed and gained regulatory approval for a specific therapeutic agent for the adjuvant treatment of sepsis?

Has your research revealed any promising specific therapeutic agents for the adjuvant treatment of sepsis?

[jLionhart](#)

I can't really answer that question. Adjuvant therapy is a concept in oncology, where a patient receives chemotherapy for a cancer that is no longer visible after the main tumor has been removed by surgery. I'm not certain how to translate that concept to sepsis care.

Is there anything on the horizon for pulmonary arterial hypertension? I'm currently taking Opsumit and sildenafil. They work fairly well, but I'd like to lower my numbers a bit more.

[aintsuperstitious](#)

The best that I know of is some exploration of combo-agents, but I have partners that specialize in that the way I specialize in sepsis and acute respiratory failure. That's all I've got right now. I'm sitting in Chicago at CHEST HQ, or I would phone a friend.

Daily CXR for intubated patients or PRN? I have spent time at institutions which do both. Tube mispositioning is common with routine turns and can be devastating. Thoughts?

[PictureDoc](#)

Tube malpositioning can be found in any of a number of ways. Ventilator mechanics. Bedside US, which most of us should now be comfortable with. The "malpositioning" that can be common with turns is also known as the tube "fell out". It cannot be prevented by an x-ray. Only by caution.

Infectious disease physicians and clinical microbiologists (an entity which I know exist in Ireland) have very different perspectives when it comes to sepsis, the former preferring a top down approach ie use very aggressive antibiotics and stepping down as their condition improves, whereas the latter prefers escalating following worsening severity. Is this something you've had to deal with much in your career, and guidelines development ?

[magibaconite](#)

Not the specific jobs, but concepts. There are plenty of data (see Marin Kollef, see Anand Kumar) to show that patients with severe sepsis and septic shock, MUST receive broad spectrum antibiotics initially, because they will die if you choose an agent that their organism is not sensitive to. Period.

Hello Dr. Simpson

Thank you for doing this AMA today. My grandfather is currently bedridden due to a hip fracture, but surgery was successful and he should be out and about in a few months. I've heard that pneumonia is a common risk for patients like him. What can our family do to minimize this issue for him?

Thank you very much.

[MonteDoa](#)

For any patient with a hip fracture the single most important component is ambulation ASAP. Lying in bed breeds pneumonia, and if you are on this topic you know what that leads to. Walking is the single best thing.

What is your favorite ventilator? Our department is looking to get some new vents.

[Trigger3x](#)

OK, well first you know I can't give specific brand recommendations. Unless, of course, you ventilator guys are out there and want to send an electronic deposit to www.simpleton.fund.org.argentina. If it's large enough, we can surely work something out.

Again, I will not endorse anyone, and I have not used every ventilator on the market. In fact the one I want the most, my RT department just won't get me. But there are a couple of ventilators out there that provide esophageal pressure monitoring that can be used for either or both of triggering and (what I think most important) measuring transpulmonary pressure for better PEEP titration during ventilation of

patients with ARDS. Then, other capacities that I appreciate are proportional assist ventilation.

I will be honest, though. It is my strong opinion that you should be able to give me a Puritan Bennett MA-1, and I should be able to figure out how to use it for whatever my patient needs. The truly important component should be the brain of the user.

Do you have any thoughts on out-of-hospital fluid administration for patients who fulfill severe sepsis criteria (however you define it) but are not hypotensive?

[6wolfy9](#)

I do think that nearly all out-of-hospital patients are intravascularly "dry" to one extent or another. They have had insensible losses and poor intake, usually for at least a couple of days before they either call EMS or get themselves to the ER. How much do I think they need? Which day is it today? I feel relatively confident that patients who are having an organ dysfunction/organ underperfusion of one kind or another are likely to respond to a fluid bolus. I do not believe it needs to be 30 mL/kg at that point, and there is no data that it should be. However, there is some recent data from Kaiser Permanente of Northern CA about patients with lactate >2 but <4 . They instituted a protocol for 30 mL/kg in those patients and found significant benefit. The cool part is that the benefit they found was nearly all in patients with CHF. In other words CHF is NOT a reason to avoid fluids in patients with sepsis. That wasn't exactly your question, but it seems close enough that you might find it interesting.

Hi there. I have a tricky one for you. Also apologies for the long post.

My Dad died 4 and a half years ago and although he spent his last 11 weeks in a hospital and when he did pass away, a postmortem was performed, they couldn't give us a definite cause of death. They listed it as ARDS. Acute respiratory distress syndrome I think it stands for. Some background..... My Dad was a 61 year old ex smoker (gave up 12 years previous). He was building an extension on our house and was very fit, slim, didn't drink at all and exercised very regularly. (2-5 miles with the dog daily) His first symptoms were flu-like. Aches and pains and he said his teeth felt loose like he had been poisoned.

He went to the doctor that day and collapsed at the doctor's so my Ma brought him to the hospital. There, his blood oxygen levels were between 68 and 75. The staff couldn't understand how he was responsive with levels that low. Obviously blood tests came back negative for any toxins and he was treated with broad spec antibiotics, oxygen through the C-Pap and bedrest. He gradually got worse over the next 7 weeks after multiple tests for cancer, autoimmune diseases and even HIV. All came back clear but he kept getting worse. So at around week 8, his doctors were starting to panic and he was transferred to a ward of a public hospital in Dublin where they put him on ECMO. This did not have the required effects and approximately 3 weeks later, his other organs were starting to fail so we turned off his machines as the lungs were not responding. I know it is very hard to speculate about what the cause of his death was but if you were a betting man, what would you think it was?

[Bessieshuman](#)

You know, it's going to be tough to say. This sometimes is the way that severe sepsis and septic shock go, especially if you either don't ever figure out which organ is infected and don't actually get the infection eradicated. We call this "chronic critical illness". There are a number of ways it could go down. Perhaps he was never bacteremic, so blood cultures were negative. Perhaps he got his antibiotics before cultures were taken, so they wouldn't grow. Speculating, pneumonia could do this and if it abscessed or flooded the pleural space, which is the area outside the lungs, but still in the chest, a chronic infection could have set in. Being in the hospital for so long, he could have developed additional infections. It is likely that by the time he died his immune system was in more of a state of

paralysis. This does certainly seem like a rapidly progressive sepsis. You don't really say how soon after admission the ARDS was diagnosed, so I'm not certain whether he had it on admission or developed it over the first few days in hospital. Legionella pneumophila or Legionnaire's disease could cause this and it is normally not cultured but discovered by different tests. I'm not sure how common that bug is in Ireland. You don't say which town or city you're in or what sort of facilities you have there. I'm really sorry not to be helpful. However, the rapidity of the initial illness does sound as if it were sepsis of some origin. I just don't get a fix on the source of infection.

ER nurse here... We just started calling sepsis alerts in my ED, and it's been crazy. Disorganized. Etc. I know it's growing pains and just getting the change down, but, is there any research on CALLING the alert overhead vs not calling it that gives insight on its effectiveness? I mean, I could see that things are already in motion without the alert, does it help in efficiency and ultimately M&M??

[eziern](#)

I don't actually know of any research on whether the overhead callout is important. I do know that many places have used a "code sepsis" approach. However, it isn't how you call it, it's what you do once it's been called that counts. The key is to have, at least on paper, an organized approach that represents your ideal way of doing things. Think ACLS or ATLS. Then practice it. In an unused room, at random times, until everyone knows their job. Notice in your practice sessions what parts don't work the way you thought they would. Fix them in the practice sessions. Then do them with real patients. Again to ACLS - would you let some neophyte walk in and run the code without any experience or without studying and getting certified? Sepsis is just as important and just as deadly. It's worth the effort.

My daughter has sickle cell disease and suffers from severe acute chest syndrome. She's been placed in the PICU 6 times in the past 4 years and intubated due to respiratory failure three times. The doctors are stumped by her as typical treatment does not work. They are often very confused by her. She is well 90% of the year, but that 10% she gets really really ill. She has had sepsis several times. She also has a strange reaction to Epstein-Barr virus. Once it skyrocketed her WBC to 80,000. It's almost as if her immune system is weak until exposed to something and then it overreacts. She is treated for asthma even though she's never really had a true asthma attack. Oral steroids sometimes help but her doctors seem to think it's just a coincidence since oral steroids are not usually effective forms of treatment. She recently suffered a reaction to a blood transfusion as well (she developed cold antibodies).

Do you have any advice for us/her? She is eight years old and biracial. She has Sickle Cell Disease type SD. Possibly undergoing a BMT in the next year or so.

[NurseJoy1622](#)

I would love to be able to help, but I really can't comment on individual cases. I haven't seen or evaluated your daughter, and it would be totally irresponsible of me. I know you must feel desperate from what you describe. I am sorry that I can't be of use. I wish you and her the very best.

What is the overall ICU survival rate? Has it changed much over the past several decades? If so what has been the biggest contributors?

[todaylact](#)

Well, that depends upon where the patient became septic and who was treating them. For example, patients who present to our ER with severe sepsis have a mortality rate of 7%, and those with septic

shock have a mortality of 17%. The mortality if they develop sepsis in the hospital is twice that on both accounts, as is the mortality of patients who transfer to us from other hospitals. These numbers are consonant with estimates from other studies, as well. Aggregate national numbers suggest septic shock mortality around 30% - 40%.

what do you think of the NEWS score and do you think it has an utility in the emergency room for early sepsis identification?

[Doctorpayne](#)

In general, I rather like it. It, of course, needs to be something that is calculated frequently and done automatically by your EMR. No one is ever going to memorize the point scale, just as they are never going to memorize the SOFA scoring system. Of course, all you over-industrious medical students who want to show off have done it, but you're going to forget after the exam, anyway.

Anyway, the AJRCCM has pre-published a study by Matt Churpek from the U of Chicago that compares NEWS, MEWS, qSOFA, and SIRS for predictive ability for mortality or ICU transfer. NEWS performed the best. NEWS and SIRS identified the outcome substantially earlier than qSOFA. The tradeoff between SIRS and NEWS would be specificity. I have an editorial/review coming out in Annals of Translational Medicine sometime soon that talks about all of this. Keep your eyes open for it. I thought it might be out in January, but no such luck.

A point I make in that article is important, I think. SOFA, qSOFA, SIRS, MEWS, NEWS have all been compared using the ROC curve. What the ROC curve tells us is that NEWS out of all of those is the best OVERALL test. However, we don't use any of those things as an overall test. We use specific cutoffs with specific sensitivities and specificities. It is those sensitivities and specificities that we will use in real life that need to be compared with one another. You will see in the ATM piece that severe sepsis has more sensitivity and more specificity rolled into one test than any of these other scoring systems. The enemy of "good enough" is "better".

Why is a type and screen part of my hospital's sepsis protocol?

[user_41](#)

Damned if I know.

My husband had his right lung (spontaneous) collapse 2x in 6 months about 11 years ago. After the second time he had the procedure where his lung is folded down I guess? And stapled "like a paper bag". What are the chances of another incident happening? He fits the profile, slender white male with long arms. He is always concerned it'll happen again. Thanks!!

[chocolateandpretzles](#)

The chances are less that it will recur, now that he has had surgical repair. In general, the indication for a surgical repair is the second episode, which presages that there will be multiple episodes, unless something permanent is done. This says nothing about the remaining lung, which is possible still at risk. All of this is probabilities, BTW. I obviously know nothing about your husband's specific case. If he smokes, though, tell him to stop.

How long before CMS catches up and utilizes qSOFA in identifying sepsis? They seem to still be stuck

on using SIRS

[Jawolelampy](#)

CMS is appropriately not interested in changing the diagnostic formula. See my answer to anavrin0001, above.