Eric Sullivan - Ventilator Associated Events: A Patient Safety Opportunity, presented today by Dr. Michael Klompas.

My name is Eric Sullivan and I’ll be hosting today’s webinar. As you may know, with the restructuring of the QIO Program, the atom Alliance was created as a five year, five state Initiative to ignite powerful and sustainable change in healthcare quality. Today we welcome participants from the states of Alabama, Indiana, Mississippi, Kentucky, and Tennessee. Organizational members of this Quality Innovation Network (QIN) include the Alabama Quality Assurance Foundation, Information and Quality Healthcare of Mississippi, and Qsource of Indiana, Kentucky, and Tennessee. Our expectation is that healthcare consumers and stakeholders will benefit from the collective strengths, depth, breadth, and efficiencies gained through our atom Alliance’s synergies and successes. If you would like additional information about HAI prevention in your state, please contact the atom Alliance representative listed here.

Before we begin with today’s webinar, I want to run through of a couple of housekeeping items. First, we want to encourage you to submit questions and comments in the Chat and Question functions as the phone lines have been muted. Also, this webinar has been recorded and will be posted on the atom Alliance website. As you listed to today’s webinar, please consider new ways of applying the concepts discussed and identify opportunities for action within your organization. We do truly appreciate all you do to improve quality and achieve better outcomes in healthcare at lower costs for the patients’ communities we all serve. With that in mind, it is my pleasure to introduce Dr. Michael Klompas, who is an associate professor at Harvard Medical School and associate hospital epidemiologist at Brigham and Women’s Hospital in Boston. He attends on the Infectious Disease and Internal Medicine Services at Brigham and Women’s Hospital, and he is published widely on the surveillance, diagnosis, prevention, and treatment of ventilator-associated pneumonia. His work to develop objective, automatable definitions for complications of mechanical ventilation ultimately led to CDC replacing their longstanding definition for ventilator-associated pneumonia with definitions for “ventilator-associated events.” Welcome, Dr. Klompas, you now have the floor.

Dr. Klompas – Ok, thank you very much. I really appreciate the opportunity to be here. And, thank you very much for everybody for tuning in. It’s a real honor to be able to speak to you today. I’m going to see if I can get control of the slides. (Pause, then dialogue in background re technical assistance with slides) I do have grant funding from CDC, let’s move ahead. The starting point for this talk is Ventilator-associated pneumonia because, classically, that’s been the way we tried to monitor quality, tried to monitor ventilator complications in our ICU populations. And, the reason for that I think, is really well-appreciated by all of us. VAP is a common problem. It affects 5-15% of patients, depending on how you define it, and it is bad for patients. The ventilator increases time in the ICU, hospital stay, has a very crude mortality rate, and adds substantially to costs. Our first motive for trying to prevent VAP is our clinical responsibility – we want to protect our patients from this nasty complication. The problem, though, is that VAP is not something that we can consider ourselves, internally, alone. It’s not something that is inside our hospitals alone, just for our own attention. In the past 10-15 years, VAP has really come under the national spotlight. There are many people outside of our hospitals that want to know about our VAP rates and are paying attention to them. And, what I’d like to argue is that it really changed our behavior around the way we do VAP surveillance and therefore how we have to interpret those results. We know that our states’ health departments are asking us to report VAP; we know that CMS has been interested in it; we know the Joint Commission has been interested in our VAP rates; sometimes some of our hospitals’ marketing departments have been interested in our VAP rates. Those sorts of internal influences have really changed the way that surveillance is conducted and its meaningfulness. So, to summarize that, here is the famous APIC map of the states with mandatory Healthcare Associated Infection (HAI) reporting legislation and, as you can see, that just about every state in the country has some sort of legislation requiring folks/hospitals to report certain kinds of HAIs. Now, most states actually didn’t require the reporting of VAP, a few did, Pennsylvania, South Carolina amongst them, but most did not because of the push-back from the clinical community. Nonetheless, a lot of states wanted to do so. I think, as hospitals and providers, we were all fearful that VAP would be added to that list of reportable hospital conditions. In addition, we know the Joint Commission, a couple of times now, have returned to the addition of VAP prevention as a national patient safety goal. We know that for a number of years, CMS has no longer been compensating us for preventable complications of care, such as wrong-site surgery or catheter associated UTIs, and, CMS has always wanted to make VAP one of those non-reimbursable complications. And, the only reason they did not do so is because of the push-back on the definition. So, at some point CMS said, ‘Well then, fix the definition’. This is an important strain in leading to the development of the <diagnosis> of VAE. The problem is that all of these initiatives presume that we can accurately identify who does and who does not have VAP. We can say, ‘This person has pneumonia, and this person does not.’ But, what I’d like to argue is that VAP is a particularly difficult diagnosis, and, difficult for anybody to say with confidence, who does or does not have the condition. The first question you might ask is simply, “How do you define ‘VAP’?” And, it turns out, there are at least six definitions for VAP inside their literature. Here’s a recent paper that summarizes those definitions, and the key thing point here is that all of the definitions are different on how to define this condition. And it won’t surprise you that, as there are six different definitions, they lead to six different kinds of rates as is shown on this kind of slide. And some of those rates vary widely, one to another. And the thunder of the problem is that we don’t know which one of these definitions is correct. We have no reason to say that this definition is better than that definition. That’s a real problem if we counted our VAP rate toward advertisements or accreditation materials. The fundamental challenge of VAP diagnosis, as I understand it, is that there are many complications in critical care that can occur, and, for a ventilator patient, a lot of the time it looks the same. If you think about the four clinical signs of a VAP: radiographic opacities, fever, abnormal white blood cell count, impaired oxygenation, and increased secretions – if you think to yourself about each of those criteria, I’m sure you’ll agree that each of those things are associated with lots of different kinds of problems. Lots of different capacities: lots of things can cause fever, so on and so forth. None of these things is either sensitive or specific. We did a study trying to demonstrate lack of specificity of different kinds of clinical science. So, this over here is an analysis of 14 different studies that tried to assess the accuracy of clinical signs for VAP using autopsy as the referenced standard. Check out the standards for patients that had the clinical signs, and these patients went under autopsy, and they worked out which of those patients had pneumonia and what is the correlation between those clinical signs of presence or absence of pneumonia. The way this slide reports the results is something called “clinical likelihood ratios”. And, to refresh your memory, a ‘likelihood ratio’ is a marker of how a test increases your confidence about the presence or absence of a condition. So, technically what you do is you calculate your patient’s pre-test probability about having the disease and what the probability of having pneumonia before you look for a clinical sign. Then, if you have a clinical sign, you multiply that pre-test probability by the likelihood ratio and that gives you the post-test probability. All of that is a complicated way of saying this: if the likelihood ratio is one, then it doesn’t really change your confidence at all whether the condition is present or not. Low and behold, here we have for each of the key clinical signs for VAP: fever, abnormal white blood cell count, purulent sputum, crepitations, hypoxemia, new infiltrate – the likelihood ratio are all essentially one. Meaning, they do nothing to change our certainty, our confidence as to whether pneumonia is present or not. Nothing brings this home better than looking at a radiograph. This is a radiograph, more or less chosen at random from one of the patients at my hospital, and the first thing that strikes us, of course, is that it is a very complicated film. The patient is rotated, the patient is leaning over, they are under-penetrated, and they have lots of overlying tubes and lines. Oftentimes that is the case with these patients. They are not such simple, straight-forward cases on which to take a chest x-ray. I think we can all agree that this is an abnormal chest x-ray. We can see there is extra white stuff at the bottom of both lungs. Yet, who can say with confidence as to what the underlying problem is? Is this pneumonia? Is this fluid? Is this contusion? Is it ARDS? Is this some combination of all of the above? Now, if you’re talking to a non-clinical audience, their instinct may be, “Well, hang on a second. Don’t you pay a cadre of professionals to interpret these radiographs for you? They’re called radiologists.” So let’s go ahead and see what the radiologists have to say about this film. So here’s a segment from the radiologist’s report: “Diffuse patchy airspace disease right greater than left with obliteration of both hemi-diaphragms. Opacities possibly slightly increased since yesterday accounting for changes in patient position and inspiration. This could represent atelectasis, pneumonia, or effusion.” In other words, the radiologists themselves can’t say with certainty what the underlying condition is. And, you’ll recall that, historically speaking, it was the infection preventionists who were doing surveillance cases, who would often look at radiographs alone. So what’s an infection preventionist to do when he or she reads this kind of report? Does this patient have pneumonia or not?

I think we can agree that the infection preventionist would be very well within his or her rights to say that the patient does not have pneumonia because it is not a certain report. But, you know, maybe the patient does have pneumonia. So that is the fundamental dilemma here is that we’re not getting clarity as to what is or is not there. And in fact, when you do the intensive analysis of patients who look like they have VAP, you’ll find that most of the time, 60% or more will not have VAP. So this over here is an analysis of patients with fever and infiltrates looking to see what the actual underlying conditions are if you investigated it very, very intensively. You can see it’s a grab bag of conditions: ARDS, diffuse alveolar damage, thromboembolic disease, hemorrhage, infarction, fibrosis, cancer, bruising of the lungs – all sorts of things. What’s particularly interesting to me is that often these patients have more conditions that, individually, look nothing like pneumonia, but when present together, give the next clinical impression of pneumonia. For example, the patient might have increased secretions from a tracheobronchitis, or a fever from a CLABSI or UTI or a drug reaction. None of us would think that individually it looks like pneumonia, but, what if, at the same time, the patient was to have an abnormal chest x-ray because of some pulmonary edema, atelectasis, or some bruising, or fibrosis? Again, none of us would think that any of these things on its own would be pneumonia. But when we see these things at the same time we get the next clinical impression of pneumonia. So when you go to the analyses of what’s the accuracy of clinical evaluation for pneumonia and you compare that to autopsy, this is what you’re going to find**.** This is an analysis of 253 autopsies of ventilator patients and they’ve applied a loose definition first of all. Now there was an infiltrate in two or more of abnormal temperature/white blood cell/purulent secretions – kind of sounds like pneumonia. And, low and behold, the sensitivity over there is right around 60-65%; the positive predictive value is around 50%. So these investigators kind of kicked it up a notch and said, “Let’s try something a little more intense.” So they then applied a stricter definition, which is an infiltrate and all three of abnormal temperature, abnormal white blood cell count, and abnormal purulence secretion, that you and I know, that our commissions on the front lines, when they see this particular combination, that they are firing the Vancomycins, the Cefepimes, for they are treating it as it looks for all the world like a pneumonia. But what it actually is is a positive predictive value of that. That strict constellation substantially drops the sensitivity and barely budges the positive predictive value: we only get up to around 65%. So, you might be saying to yourself, ‘Hang on. You’ve left out the most important clinical sign, which is the microbiology. The pneumonia is an infectious disease, so the microbiology should be very helpful, very important. So, here is the analysis of the sensitivity and the positive predictive value of the quantitative BAL cultures relative to histology in autopsy. Now, they’ve put together data from five studies and, as you can see, the collective sensitivity is around 50-60%; and the collective positive predictive value between these studies is around 60-70%. They’re not perfect. How can that be? How can that be that the microbiology is not diagnostic? The answer is you can get less of false negative due to prior exposure to antibiotics that basically stun the bacteria - they no longer grow inside your specimen. And we know that the majority, the plurality of patients in ICU are getting antibiotics at some point. Or, you can also get a false negative, a failure to find the VAP, a failure to find the positive culture, because you’ve failed to sample the exact right segment of lung where the pneumonia is sitting. So, if the pneumonia is at the top of the right hand lung and you sample the middle of the right hand lung, you’ll miss the pneumonia. How about the false positive? How can it be that some of these patients with a positive culture do not actually have pneumonia? It turns out that, when you try to acquire the specimen, a lot of risk contamination occurs from bugs that are living inside the mouth bacteria will colonize the endotracheal tube. And so you can get false positives from those two sources of contamination. The bottom line that is stressing me is that none of our diagnostic tools for pneumonia are that good in this particular population. And, of course that has incredible implications for surveillance, for quality, for benchmarking.

Let’s try to consider some of those implications together. So this over here is CDC’s old surveillance definition for VAP – the one that got replaced two years ago. What you can see is that it required the patient to fulfill radiographic criteria, systemic criteria, and pulmonary criteria. The patient had to have a new, progressive infiltrate. We saw together how that is certainly not diagnostic of the patient’s underlying condition. An abnormal temperature, an abnormal white blood cell count are very non-specific signs. Any two of the following: new onset of purulent sputum or change in character of sputum, or increased respiratory secretions, or increased suctioning requirements; worsening gas exchange, increased oxygen requirements, or increased ventilation demand. Now the problem with these is that they are complicated and non-specific. But, in addition to that, they are very subjective. So there’s nothing in this definition to say, ‘What constitutes new onset of purulent sputum? What is the increase in respiratory secretions? What is the worsening of gas exchange? Who makes that call? Is it the nurse, the doctor, the infection preventionist, the respiratory therapist? How many mls of sputum are required to constitute an increase in secretions? How long does that change have to be sustained for?’ All of this is not defined and therefore left to the discretion of the observer. And you can imagine how two very conscientious, two very thoughtful infection preventionists might interpret this quite differently. One might think there are more secretions and one might say, “Well, it’s only lasted for a day or so – that’s not enough.” And so, it’s highly problematic as far as different, reasonable, decent people trying to do a good job coming to very different conclusions. In essence, that definition is complicated. A lot of clinical criteria require a highly trained nurse or equivalent to be able to do surveillance. It’s a lot of work, yet, because it is subjective, and with a criteria that is very non-specific you have no assurance that a.) you’ve actually found a true pneumonia, or b.) when you do that surveillance again, that you’ll be able to come to that same conclusion. So it’s a lot of work and not much credibility to the result itself. Regardless of the issue of subjectivity, this is a study that demonstrates how different observers that look at different patients come to very different kinds of rates. This is a study of 50 ventilator patients, all with respiratory deterioration, and they’re independently assessed by three different infection preventionists. And, when asked how many patients of these 50 actually have a VAP, one IP found 11, one IP found 20, and one IP found 15. Collectively, they only agreed on seven. Now, what’s distressing about this is in this day and age, when C-Suite is looking over your shoulder at your VAP rates and you’re worried that the state health department is going to be asking you about it and on your next Joint Commission visit they’re going to be asking you about your VAP rates, in that kind of environment, IP Number 1, who only found 11 VAPs of course is very popular. He or she is going to have a bunch of job offers, whereas IP Number 2 has been relegated down to the basement. And the fundamental problem there is that is unfair. We actually have nothing to tell us that IP Number 1 is more correct or less correct than IP Number 2. We don’t know which one of these is correct and yet we have a two-fold variation of a sensible rate. So again, <this is> a fundamentally unreliable definition. Here’s another demonstration of the same problem. This was a national survey of people who are responsible for doing VAP surveillance at a number of hospitals. What these investigators did is that they created six vignettes of patients who may or may not have had VAPs and what they asked of each respondent to work out was how many of these six vignettes actually did qualify using the old CDC definition. And you can see over here is that equal numbers of respondents voted them as one case of VAP, two cases, three cases, four cases and five. In other words, the answers were more or less random and a major problem, of course, if you’re trying to have any notion of comparability from one hospital to another. The other idea that comes out of this is that, if you’re a little bit cynical for a moment, you can take the information I’ve shared with you and actually work out a way to drop your VAP rate, using the old definition, to work out your VAP rate to nothing without doing anything to change the way you do patient care. So you could narrowly interpret your subjective clinical signs. “Are there more secretions today compared to yesterday?” No. Narrowly interpret radiographs – the radiologist is not absolutely certain it’s pneumonia – “I’m going to say ‘it’s not’”. Seek consensus between multiple surveyors – we saw how, when we looked at those 50 patients evaluated by three different IPs, that one found 11 and one found 20, but collectively they only agreed on seven. Anytime you’re trying to find consensus, you’ll find that they’ll be a smaller number of patients on which you can all agree. Allow clinicians to veto surveillance determinations – now there are two problems. The first is you are adding extra opinions so of course you are going to lower the number of people who have consensus. The other problem is that clinicians are not applying surveillance definitions. Clinicians are applying a clinical gestalt. And the problem is that my gestalt might be different than your gestalt. And if we allow our gestalt to be our determinate, we toss out of the window the whole idea of having a surveillance definition which intends to bring a comparability, reproducibility, objectivity to surveillance that allow me to compare the rates at my hospital to the rates at your hospital. If we allow gestalt to take place, then we have no idea really of what we’re getting out of the system. Then finally, increase use of quantitative BAL – and we saw how that’s an imperfectly sensitive test that’s going to lower your VAP rate. Now, I think that what’s disturbing about this particular list that we have here, is that, at first blush, these might all appear to be good things to do to increase the rigor of your infection surveillance program. Why not be stricter in your interpretation? Isn’t it good to seek consensus? Don’t you want to get feedback? Shouldn’t we be relying on a quantitative microbiological test? These all sound like a good way to go for your program. And yet, because of the weaknesses of the old CDC definitions, we know that each of these strategies will lower the observed VAP rate and you’ve done nothing actually to change patient care. When we look at U.S. national VAP rates in the past decade and we see this beautiful decline, as shown in the slide over here, to the point that the Surgical ICU rate and the Medical ICU rate is almost zero, we don’t know if this decrease is because you’re providing better care for patients, or because we’re changing the way we apply the loose definitions. And when you can’t tell whether you’re dropping rates to better care or different surveillance, then arguably that surveillance definition has failed you. And we have some clues that surveillance changes might be playing a large part in that decrease in VAP nationally. See, here’s one clue. This is a look at international VAP rates and what you can see from the slide here is that the VAP rates in our country are literally a full order of magnitude less than our colleagues in Europe. And I don’t think that any of us would have the gumption to argue that we provide ten times better care than our friends in France and even Belgium. The other thing is that there are increasing numbers of studies where clinicians and infection preventionists did simultaneous surveillance on ventilator populations. So basically, they’re using their clinical judgement as to whether or not their patient had VAP and IPs using NHSN definition. You’ll see a substantial mish-match over here between the cases found by clinicians verses the number of cases found by the IPs. You can see that the clinical surveillance again is failing you. Finally, we know that if you go up to an ICU at any given time you’ll probably find that about 15 percent of patients are getting antibiotics for nosocomial respiratory infections. Yet how do we mix that with zero VAP rates in this country? Within this math and mismatch underlies a credibility failure of the old system. This lead to a fundamental tension: on the one hand, we need to publicly report VAP rates to catalyze improved quality of care and save lives; at the same time, though, we had those who said, ‘Well, hang on. The definition of VAP is ambiguous, it’s open, and it’s hard to be gauged’. And stuck in the middle is you. So folks at the CDC said, ‘We’ve got to do something about this’. So about three years ago, people at the CDC gathered together representatives from each of the key stakeholder societies and said to them, ‘You’ve got to do something about this; you’ve got to help us change this definition to something that’s more credible, particularly in the current benchmarking, quality surveillance, public reporting, etcetera.’ And this was the group that came up with the Ventilator-Associated Event framework. The thinking behind this group was really interesting. What the group said is that, if the core problem is that we can’t say with confidence who does and does not have pneumonia, then why are we pretending that we can? Furthermore, why is it that quality surveillance is only focused on pneumonia? Aren’t we worried about all the other things that can go wrong with a ventilator patient, i.e., the pulmonary edema, the ARDS, the atelectasis, the pulmonary embolism, and so on and so forth? Don’t we want to track these as well? Don’t we want to know about these so we can prevent them? And so what they said, ‘What if we shift the focus away from the pneumonia itself and to the syndrome, and focus on the complications in general.’ And the big advantage is that, Number 1, it allows us to substantially simplify the definition because we are no longer pretending that we can actually define the pneumonia, all we have to look for is respiratory deterioration, and we can tell that by using ventilator settings and one or two other things, so we can allow for a much simpler definition. And at the same time, by looking for the syndrome of pulmonary complications, it forces us to expand the gamut of our prevention activities to prevent much more than just pneumonia alone. The core condition, as you should know by this time, is called a VAC, or a ventilator-associated condition. Conceptually, you are looking for a patient who is stable or improving on a ventilator for at least two days and then has sustained worsening for at least two days. In other words, you have a patient who has had a trajectory change from stability or improvement to worsening, and, effectively, the way the surveillance is done, we use two ventilator settings, one is call PEEP, the other is called the FiO2. The PEEP is the Positive End Expiratory Pressure and the FiO2 is the Fraction of Inspired Oxygen. You want to take the patient’s best value of the day, which is their lowest value, and you want to lay out those two values per patient per day. And then, when you have it in a table like this, it’s very easy to scan your eye down this list and see that they started on very high ventilator values on January 1st, quickly jumped down on January 2nd, and then stayed low for the next couple of days and on January 5th you can see the patient had a jump in their peak and that meets the VAE threshold. That over there is the patient’s VAC. I hope that you can appreciate with me how simple it is to do the surveillance once you can acquire and organize the data. Also, it would be quite straightforward to teach a computer to do this if you could feed the computer the raw data. And that, of course, is beautiful, because it takes away the opportunity for argument with the frontline clinicians. There is no argument with the patient whether VAC criteria or not because the computer can tell you the result. And secondly, if you can automate the surveillance, then you can allow the infection preventionist to free up his or her time to spend at the bedside to be teaching, to be interacting with clinicians, which is a much better use of human resources than surveillance alone. It’s easy to recognize that this first tier of respiratory complications alone is non-specific. It’s a grab-bag that includes lots of potential kinds of complications. Now that was intentional. We wanted to expand the surveillance to include more than just pneumonia, but it doesn’t give you any clues as to what the other conditions might be. There are sub-criteria in the VAE definition set to try to identify which fraction of those VAEs might actually be infectious. The infection-related ventilator-associated complication, and that’s based upon the patient’s temperature, white blood cell count, and decision to start antibiotics by condition. And, if possible, if you get an infection that started in the chest, it might be bad enough to actually bump the ventilator settings. So, just because you do an IVAC doesn’t necessarily mean you have pneumonia. There’s a third tier of the VAE framework that says can we actually localize this infection to the line and to do so we look at the pulmonary secretions, the inter-tracheal aspirate, the BAL, and looking for any traces or pearls of positive culture. So here’s an example of an IVAC, or an Infection-related ventilator-associated complications <table>. Same patient as before who had a jump in their peak around January 4. We simply scan out two days in either direction and look to see if we find an abnormal temp or white blood cell count, and, if so we look to see did the patient had an antibiotic start for at least four days – yes, we did, so this patient has an IVAC. And here’s the example of a PVAP over here - same patient as before. The patient qualified as an IVAC; we look to see if the patient qualifies as a PVAP as well. We are looking for evidence of purulence on a gram stain now, so we’re not just simply eye-balling whether or not the secretions are purulent or not, we’re actually looking to see on the gram stain white blood cells are polys that are cellular markers of purulence. If so, and the patient has a positive marker, then the patient qualifies as a PVAP.

I hope you’re aware that the CDC does have an online calculator to help people with VAE determinations. Here’s a screen shot of the calculator and you can see how you enter your patients’ raw data and the machine will pop-out as to whether your patients’ have a VAP, and IVAC, or a PVAP. There are also a couple of reports from hospitals who have entirely automated their surveillance for their entire hospital. They’ve been getting the computer to do all of their surveillance. And we spoke about the wonderful advantages for their infection prevention program because it allows you to free up your few resources to do observation and education rather than surveillance. Now, what do we know about VAE epidemiology? So there are an increasing number of studies and we actually know quite a bit at this point and let me just share with you a few highlights. These are representative studies, but there are others that echo the same messages as the ones that I will show you. So this one over here is from a Canadian Critical Care trials group that did simultaneous surveillance on VAE and VAP over a two year period in 11 ICUs and they tracked over 1300 patients. And the first message is that they found that VAE and VAP rates were pretty similar. Many hospitals think that their VAE rates are always higher than their VAP rates, but this is true to say that their VAE and VAP rates were pretty much the same. However, when they actually looked at the individual patients who had a VAE vs. a VAP, it turned out that it was a quite different population. You can see the overlap of the VAE and the VAP was pretty small. And we’re going to talk a bit more about this to try and understand the 100 patients who had VAE who did not have VAP and the 109 patients who had VAP who did not have VAE. That is an important part to have a topical discussion, so we’ll get to that. But the key message is that VAE and VAP are not the same and you can see that on the slide. That begs the question: What is a VAE?

If it’s not pneumonia, what is actually causing the VAE? Now there are at least four studies that I know of where they’ve done intensive clinical reviews of their VAEs to try to work out what is the clinical event that lead to the jump in ventilator settings that qualified as a VAE. And the results from all these studies are all about the same. They find that there are about four conditions that, by and large, account for all VAEs. Now those four conditions are: pneumonia, fluid overload, atelectasis, and ARDS. The proportions vary somewhat from study to study, but all of the studies have found the same thing. These are the four conditions that account for the vast majority of VAEs. VAE and VAP are not the same thing. VAE is a bundle of pneumonia, fluid overload, ARDS, and atelectasis. Now, the nice thing about that is that your front line clinicians may not know much about VAEs; but they’re pretty darn familiar with pneumonia, ARDS, pulmonary fluid overload, and atelectasis. They can draw upon their mixed experiences of the past 20 or 30 years on what are the problems, what are the things that caused those conditions, what are the things that we can try to do to prevent those conditions. The next message though that is emerging from the VAE research literature is that VAE is a highly morbid event. So, what I’ve played out over here is six different research studies trying to look at the attributable mortality of VAE, and it turns out they all found about the same thing: VAE approximately doubles the patient’s risk of dying. It’s a really nasty event, as compared to similar patients without a VAE. A few of these studies actually did do simultaneous VAP surveillance using traditional definitions. In most cases VAE was more morbid than that, although there was one study that found the opposite. Now, I’m sure you’ve heard there is some criticism of the VAE framework out there, and I’d like to summarize what those criticisms are and try to think through them with you. Number 1 – most VAEs are not pneumonias. Number 2 – VAE surveillance misses many pneumonias. Number 3 – VAE surveillance can be gamed. And Number 4 – where is the evidence that VAEs are preventable. So here is the summary of the positive predictive value of the VAE relative to VAP and you can see that the sensitivity is probably around 40% and the positive predictive value is around 25%. Now the positive predictive value at 25% shouldn’t surprise you at all because we said that lots of patients with VAE will not have pneumonia and that was the very mission of the new definition set. It was designed to expand the gamut beyond the definition of pneumonia alone. We wanted to be able to capture the pulmonary edema, the ARDS, the atelectasis, so on and so forth that were also causing problems for our patients. So, the fact that lots of other things are causing VAEs – that’s expected, that’s good, there should be no surprises there. But what you might be bothered with are the red dots over here showing the surveillance misses of 50% or more clinically diagnosed VAPs. Let’s think about that. There are two points to consider. Number 1 – we spent the first half of our talk describing how VAP diagnosis itself is unreliable, VAPs are “being missed”. So how do we know that those are actually true pneumonias? So when do we ever want VAP to be our referenced standard given all the subjectivity and lack of accuracy around VAP definition? Point Number 2, which I think is a little bit more deep, is that, if you think about it, for VAE to miss a VAP, it has to be the best VAP to not require an increase in ventilator settings after the VAE threshold. These are pneumonias that did not require more ventilator support. In other words, these are mild pneumonias or arguably some of them are not pneumonias at all – they’re insensitive to colonization alone, and as we’ll see, we have to remember that VAE is a surveillance concept and not a clinical concept. And the mission of surveillance is different from clinical care. In clinical care, we want to put our focus on sensitivity. Heaven forbid we should ever miss a case of true pneumonia because if we miss it and we fail to treat it, that patient is going to be in trouble. But the mission with regard to surveillance is different. With surveillance, we want to emphasize much more objectivity, reproducibility, efficiency and severity. And so therefore, we often make the decision in surveillance to focus on a subset of patients who have a much more unambiguous disease, it’s much more serious. But we take the lessons we learn from analyzing those patients, and apply it to the entire population, and presume we are therefore able to prevent both the mild and the serious conditions. So, for example, with regard to the surgical site infection surveillance, we put most of our focus on tracking deep surgical site infections in deep organ space, right, rather than the superficial SSIs. The superficial SSIs are ambiguous and not particularly serious; whereas, the deep SSIs are a big deal for patients, and there’s not much argument as to who does and who does not have a deep SSI. We do the same thing in sepsis surveillance: we focus on severe sepsis and sepsis shock rather than people who have surface criteria alone. And, what I’d like to argue is that VAE makes that same kind of strategic decisions, which is to focus on the subset of the complications which are the most unambiguous and the most severe. But, if you use those cases for root cause analyses or to look deeply at what lessons you can learn, and you apply those lessons to the entire ventilator population, you should be able to prevent both the mild and the severe cases in the future. The other issue that you’ve probably heard about is the opportunities for gaming with VAE. Never, ever bet against the human being when it comes to the possibility to game the system, thinking we are always smarter than any kind of rule set that might be out there. 42:12 The same is true for VAE. So, here’s our patient from before. This time the patient has a VAE on January the 8th. What if you were to take that patient’s PEEP and you were to raise it by one point every other day? So instead of 5-5-5-5-5, you have 5-6-5-6-5-6. Now clinically, that’s absolutely insignificant; it’s going to do nothing to the patient; it’s not going to help them, it’s not going to hurt them. It’s a clinically meaningless action. But what this will do is that it will eliminate the stable base time for that patient, and therefore, that patient will no longer qualify for a VAP because that patient doesn’t have a stable baseline against which to measure the VAE - so no VAC anymore. Now the problem with this is, of course, because this particular manipulation of ventilator settings is done purely for the purpose of gaming away your VAEs, no way you can make the argument that this is actually part of patient care, that you’re doing this to improve the care of the patient. I would argue that this would actually constitute fraud. Now if anybody actually started looking over our shoulders at our VAE surveillance and they do a review of our patients and they see that this is our pattern, I think they would be subject to penalties. And, as we know, Joint Commission, CMS, and state health departments do audits of our care. The other question that has arisen is, are these VAEs actually preventable. It’s all very well to say these are very morbid events over there, but, if it’s something that is to be used for quality or for benchmarking, can it really drive better care? That’s really all we want to know; that’s why we’re all here today to try to achieve. Here’s a study from Washington-St. Louis, where they did this prospective surveillance for VAE for a year and they found 67 VAEs and they analyzed them and they found they were caused by the same four conditions I mentioned to you before – pneumonia @ 31%, ARDS, pulmonary edema, atelectasis. What they also did is that they looked at each case and tried to work out was that case preventable or not. What they adjudicated was that 37% of those VAEs were potentially preventable. Over here we found, if you’re a glass half empty glass half full kind of a person, do you say, ‘37% were preventable - that’s great because we have the opportunity now to improve the care for a third of these cases over here who have lessons for us’? Or, if you are a glass half empty kind of a person you say, ‘Oh gee, only 37% are preventable; what about the other 63%’. So there’s a philosophical issue at play over here, but, I would argue that working out what’s really, really tough because sometimes the VAE might not look preventable at all. What if the patient vomited, for example? How could you have prevented that? But, what if you had been managing that patient in the best way possible and, a. you never intubated them in the first place because you used non-invasive, positive pressure ventilation or high-flow oxygen? Or, what if you had been doing minimal sedation on that patient who had limited mobility and you were able to extubate that patient three days prior to the vomiting event? Maybe that event would have never happened in the first place. Retrospectively trying to work out what could have been prevented is really, really tough. A better guide is to prevention, I think, is to use prospective studies, interventional studies where we try to actually see in practice how much can we prevent. And the good thing is now we have a small but growing body of studies that show us you can prevent VAEs and you can prevent more than 37% of them. So let me share that with you. 46:15

Fewer VAEs – How are we going to get there?

I think that there are two major strategies for preventing ventilator associated events. The first idea is try to minimize the duration of mechanical ventilation. Avoid mechanical ventilation altogether if you can, but if you have to put a patient on a ventilator, you ought to minimize the duration of the mechanical ventilation. Less time on the ventilator means less opportunity to suffer a ventilator complication. Parallel, we know about the four conditions that primarily cause most VAEs: pneumonia, ARDS, atelectasis, and fluid overload - unless put into place, the measures that prevent these four complications. And, what I think that you’ll find in practice is that often these are one in the same. The same intervention to decrease duration of mechanical ventilation also prevents those four conditions. So, some strategies that I would consider that meet those two standards are: minimize sedation, paired daily spontaneous awakening trials (SATs), spontaneous breathing trials (SBTs), early mobilization, low tidal volume ventilation, conservative fluid management, and minimization of blood transfusions. This, I think, is the ‘Sweet Set’, the bundle for 2015 if you will, to optimize and prevent mechanical ventilator complications associated with VAE. And, if you’re an intensivist looking at this list over here, none of this will surprise you. These are all the same sorts of things that we are hearing from all sorts of parties that we should be doing for our patients. This is on the ‘Choose Wisely’ campaign, the ACDEF bundle, the Pain Agitation Delirium Guideline, the Sepsis campaign – all these emerging from the same best practice clinical care are saying the same thing. And, as it turns out, low and behold, VAE is a good way to track these complications, a good way to track, therefore, your success in implementing best clinical care. Let me actually show you the studies, to date, that have tried to do VAE prevention. So, the first study again is from our friends the Canadian Critical Care Trials Group, and what they did is they worked for a two year period to try and enhance adoption of best practices for ventilator patients as they defined it. Outlined on the slide over here are some of the interventions they tried to get their ICUs to adopt and the change in the practice rates of interventions over time. As you see, they did manage to move the needle. They were able to improve adherence with some of these interventions, although not perfectly; it was a huge amount to move, so, originally, modest adherence to best practice, but, nonetheless, some improvement. And that was associated with a significant decrease in their VAE rate by about a quarter. The next prevention study I’d like to share with you is one that tackled fluids. So, we saw before that excess fluid, fluid overload, pulmonary edema accounts for a third or more of VAEs, so better potent management decreases your VAE rate. So this was a study that took a look at this question, it was a randomized control trial of patients who were ready to be weaned to the ventilator, and what they did was that they randomized patients to daily BNP levels verses usual care. Now what BNP stands for is blood denatured peptide, and basically it’s a hormone that’s high in patients who have excess fluid in the body. What they found, is that patients who had a high BNP level, then they would manage those patients with a depletive fluid management strategy, meaning less fluid in and more fluid out. And they found that those were associated with less time for associated extubations, more ventilator-free days, and a 50% decrease in VAEs. Now again, we saw that study from Washington-St. Louis where they said that only 37% of their VAEs were preventable, over here with just one intervention, they were able to knock out 50% of their VAEs. So again, it’s showing us that prospective studies are a much better guide to prevention than retrospective analyses. The third intervention study I’d like to share with you was the CDC’s Prevention Epicenters’ ‘Wake Up and Breathe Collaborative’. This was a one and a half year effort by 12 ICUs in seven hospitals to try to prevent VAEs through less sedation and earlier liberation from mechanical ventilation. And, what the mechanism that they chose was to increase the use of spontaneous awakening trials (SATs) and spontaneous breathing trials (SBTs) paired together. The logic behind this particular study was saying that VAE is a grab bag of different kinds of complications; therefore, the best way to prevent VAE is better sooner. And if you look at the literature, really one of the strongest ways to decrease mean duration of mechanical deterioration is minimizing sedations through spontaneous awakening trials and early extubations through spontaneous breathing trials. Those are the rationale behind the study. And so here you can see the change between SAT rates and SBT rates. Overtime, a one and a half year period study; you see substantial increase of both spontaneous awakening trials and spontaneous breathing trials. There’s also substantial increase in the pairing of SATs with SBTs, meaning, do it when the patient is off oxidation and therefore while the patient is awake as possible and the patient has the best possible chance of passing the spontaneous breathing trial. Now what did that do for them? There was an associated 37% decrease in their VAE rate and a 65% decrease in their IVAC rate; again, showing us that better care can lead to a decrease in the VAE rate by a substantial amount. This was also accompanied by a 3 day decrease in the mean duration of ICU stay and a 2.4 day decrease in the mean duration of the mechanical ventilation. So, there are clear positive benefits to the patient as well. So, even if you’re not interested in doing VAE prevention but you just want to provide better patient care, these kinds of interventions are unambiguously good, as well. To put it all together, I’d like to argue that VAEs are a patient safety opportunity. I say that because this new metric broadens our awareness, provides us with a fuller picture of the population suffering from the serious complications in our ICUs. If we know about those broader set of patients who are suffering really morbid complications, then that’s going to be the spark that is going to move us to try to do more for our infection prevention. Now we have a much more objective metric to reflect and inform on our progress. We don’t have to worry if we see a decrease in our VAE rates, that’s simply because we’re doing stricter application of our subjective surveillance signs. We can feel much more confident that we’ve actually, truly been able to do something to help our patients.

So, thank you very much. I appreciate the opportunity to be able to speak to you and I hope this has been helpful. I think we’re open for questions now.

Eric Sullivan – Thank you Dr. Klompas. This has been a great, informational webinar that you’ve presented to us. I appreciate your being willing and able to do that for us today. I want to encourage those of you who have questions to go ahead and type those into the Chat Room. Kim, do you see any questions currently in the Chat Room?

Kim – Eric, we had one question and it was for you. It was about the project, so will you please share with us about the project?

Eric – Sure. So not to take up too much time, if you are interested in working on this particular collaborative within your state and would like more information from your representative within the QIN, feel free to e-mail me at [esullivan@qsource.org](mailto:esullivan@qsource.org) , and I can help put you in touch with the correct lead for your state.

Dr. Klompas, do you have advice for those who may be early in their journey and working together on projects to start VAE prevention?

Dr. Klompas – Yeah, make a lot of friends. You want to make friends with your respiratory therapist, your nursing, your medical ICU, your medical people inside each of your ICUs, your pharmacists because, one of the key things about VAE that is different than other infection preventionist normally tracks: it’s much more than infection. In addition, a lot of the interventions I think that we’re appreciating are good for VAEs, minimizing sedation, minimizing conservative third management, etc.; they are not things that we in infection control are all that familiar with. But those in critical care, our friends in pharmacy are very familiar with these concepts, and so they’re the ones that can really help us bring those two to light. And I think that, for people who are in the critical care world, these concepts are very familiar and comfortable to them – the idea of minimizing sedation, so on and so forth. So make friends and form the alliances in order to be able to get started on this work.

Eric Sullivan – Thank you again. And I’d like to thank everyone who joined us today. We do appreciate all you do to improve patient safety. And, once again, a big thank you to Dr. Klompas for sharing your expertise with us today.

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