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Ventilator-Associated Events: New Outcome Measure

Michael Klompas MD, MPH

The CDC switched from ventilator-associated pneumonia (VAP) to ventilator-associated event (VAE) surveillance in 2013. VAE definitions were designed to make surveillance more objective and to broaden the focus of surveillance to encompass all complications serious enough to compel a sustained increase in ventilator settings. Implicit in the VAE definition set is the understanding that many things can precipitate VAEs. Case series document a wide array of potential etiologies. Redesigning prevention programs around preventing VAEs instead of VAP has the potential to overcome many challenges. VAE definitions are more objective than VAP definitions, they broaden the focus of surveillance beyond pneumonia, and they focus surveillance on the subset of patients with potentially severe complications. An increasing body of evidence is beginning to clarify how best to prevent VAEs.

Airway Secretion Management of the Mechanically Ventilated Patient: Panel Discussion

Moderator: Ruben Restrepo, MD, RRT, FAARC
Panelists: Brady Scott, MS, RRT-ACCS, AE-C, FAARC
Keith Lamb, BS, RRT-ACCS, FCCM

When addressing management of airway secretions, there are typically a few questions clinicians are challenged to answer. These include: Are we following the guidelines put forth by the AARC? What can we do before secretions impact the airway? Are there changes on the ventilator parameters that can help us determine the impact of excessive airway secretions? Is additional research needed to determine the superiority of in-line versus open suctioning? What particular cautions need to be considered when managing secretions in patients with hypoxemic respiratory failure? In this issue of Clinical Foundations, two panelists with extensive experience in the management of ventilated patients share their impressions on the impact of airway secretions and address these important clinical questions.

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Faculty Disclosures

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Keith Lamb, BS, RRT, ACCS and **Brady Scott**, MS, RRT-ACCS, AE-C each disclosed no conflicts associated with this publication.

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Ventilator-Associated Events: New Outcome Measure

Michael Klompas MD, MPH

The Centers for Disease Control and Prevention (CDC) switched from ventilator-associated pneumonia (VAP) to ventilator-associated event (VAE) surveillance in 2013.[1] The CDC made the switch because of the complexity and unreliability of traditional VAP definitions, the concern that focusing on VAP alone underemphasized the importance of tracking and preventing the fuller array of complications that can occur during mechanical ventilation, and to facilitate the possibility of automated surveillance using structured data drawn from the electronic health record.[1,2]

Surveillance using traditional VAP definitions was complicated, time-consuming, and highly subjective. The CDC's old VAP definition included criteria like, "new or progressive infiltrates," "change in character of sputum," and "increased oxygen requirement." Despite the effort required to apply these definitions, different surveyors assessing the same population often differed in their determinations.[3-5] What one observer might call a new infiltrate, another observer might call atelectasis or pulmonary edema. Lack of specificity compounded uncertainty. Each of the core clinical signs for VAP (fever, leukocytosis, purulent secretions, and radiographic infiltrates) has a broad differential diagnosis. Over-diagnosis of VAP is common, and subsequent evaluation of patients diagnosed with VAP using various methods, including clinical, histological, radiological, and/or microbiological, demonstrated that a significant number of patients did not have VAP.[6,8]

VAE definitions, by contrast, were designed to make surveillance more objective and to broaden the focus of surveillance to encompass all complications serious enough to compel a sustained increase in ventilator settings.[1] VAEs are defined on the basis of trajectory changes in ventilator settings: the core VAE component, known as a ventilator-associated condition (VAC), is defined as a rise in a patient's daily minimum PEEP of ≥3 cm H_2O or daily minimum FiO_2 by ≥20 points that is sustained for at least two calendar days and that follows at least two calendar days of stable or decreasing daily minimum PEEPs or FiO_2s.

The VAE definition set includes additional criteria to help identify the subset of VACs that might be infection-related, including, but not limited to, pneumonia. Infection-related ventilator-associated complications (IVAC) require an abnormal temperature and/or white blood cell count within two days of VAE onset, and the start of a new antibiotic course that continues for at least four days. An IVAC can conceivably be triggered by serious infections outside the lungs (such as sepsis, skin and soft tissue infections, abdominal abscesses, etc.) that require an increase in ventilator support and a new course of antibiotics. The VAE definition set also includes a third tier to help identify the subset of IVACs that might be pneumonias. A possible ventilator-associated pneumonia (PVAP) requires a patient to fulfill all IVAC criteria as well as have a respiratory culture with a minimum quantity of growth of a potentially pathogenic organism, or the combination of >25 neutrophils per low-powered field on Gram stain and a positive culture growing any amount of a potentially pathogenic organism.[9]

Implicit in the VAE definition set is the understanding that many things can precipitate VAEs. Case series document a wide array of potential etiologies.[10-14] The four most common causes are pneumonia, atelectasis, pulmonary edema, and acute respiratory distress syndrome (ARDS). Less common causes include pulmonary embolism, pneumothorax, and sepsis.

VAEs are relatively rare. The pooled mean VAE rate in small, non-teaching medical-surgical units reporting VAEs to the CDC in 2014 was 4.5 VAEs per 1000 ventilator-days.[15] The pooled mean rates in medical and surgical units of large teaching hospitals were 7.8 and 8.9 VAEs per 1000 ventilator-days respectively.

VAEs are morbid and potentially mortal events. Patients with VAEs are about twice as likely to die as similar patients without VAEs, and are also associated with prolongation of mechanical ventilation, ICU length-of-stay, hospital length-of-stay, and increased antimicrobial utilization.[14,16-18] The high morbidity and broad focus of VAEs invites hospitals to review and reimagine their approach to preventing complications in mechanically ventilated patients. Most hospitals have organized their best practices for ventilated patients around preventing VAP. There are four reasons, however, why this might not be optimal: 1) an increasing body of literature casts doubt about the efficacy and/or safety of many practices classically recommended to prevent VAP, 2) VAPs vary widely in their severity and certainty – the diagnosis encompasses patients with severe life threatening infections as well as those with mild disease that may be little more than colonization alone, 3) chart audits conducted by the Centers for Medicare and Medicaid services suggest that VAP rates have not meaningfully changed over the past decade despite widespread adoption of VAP bundles and multiple case reports of decreasing VAP rates, and 4) VAP is but one of many potential complications that can harm patients on ventilators.[19-21]

Many of our historic misimpressions about how best to prevent VAP and our lack of clarity about whether VAP rates have been decreasing over time can be attributed to the subjectivity and poor specificity of VAP diagnostic criteria. VAP prevention studies have been subject to bias because of circularity between VAP diagnostic criteria and prevention strategies.[22] Oral care with chlorhexidine, for example, will decrease the frequency of positive endotracheal or bronchoalveolar lavage cultures but this does not necessarily mean that VAP has been averted.[23] VAP surveillance studies have been subject to bias because so many VAP criteria are subjective ("new or progressive infiltrates," "increased secretions", "worsening oxygenation," etc.) and therefore allow for the possibility of lower VAP rates that better reflect stricter application of subjective surveillance definitions rather than true decrease in pneumonia.[24]

Redesigning prevention programs around preventing VAEs instead of VAP has the potential to overcome many of these challenges. VAE definitions are more objective than VAP definitions, they broaden the focus of surveillance beyond pneumonia, and they focus surveillance on the subset of patients with potentially severe complications. An increasing body of evidence is beginning to clarify how best to prevent VAEs.

There are essentially two main approaches to prevent VAEs. The first is to decrease duration of mechanical ventilation. The second is to target the complications that most frequently trigger VAEs (pneumonia, atelectasis, fluid overload, ARDS). In practice, these two approaches yield a similar set of prevention measures. These include: minimizing sedation, daily spontaneous awakening trials, daily spontaneous breathing trials, early mobilization, conservative fluid management, conservative transfusion thresholds, and low tidal volume ventilation.[25] These interventions are highly congruent with emerging best practices in critical care, including the ABCDEF bundle (Figure1), the Surviving Sepsis Campaign, the Choosing Wisely Campaign, and the Society of Healthcare Epidemiologists of America's recommended strategies to prevent ventilator-associated pneumonia and ventilator-associated events.[26-29]

Minimizing sedation

Deep sedation is associated with prolonged mechanical ventilation and increased risk of death.[30-31] Increasing the duration of mechanical ventilation increases the risk of adverse events including aspiration, volume overload, atelectasis, barotrauma, ventilation mismatch, delirium, and deconditioning. The risk is potentiated by sedation's inhibition of patients' capacity to self-regulate their breathing, gag reflexes, management of secretions, and mobility. Sedation may also increase infection risk.[32] Preferential use of shorter acting agents such as propofol and/or dexmedetomidine rather than benzodiazepines may facilitate keeping patients at lighter levels of sedation. Meta-analysis suggests that non-benzodiazepines are associated with shorter ventilator and ICU stays.[33] There are also data that dexmedetomidine in particular is associated with lower risk for VAEs compared to benzodiazepines and perhaps also propofol.[34]

Spontaneous awakening and breathing trials

Spontaneous awakening and breathing trials are designed to challenge clinicians' perceptions about how much sedation a patient needs and when they might be ready for extubation. The trials require nurses to stop patients' continuous sedatives, even if the patient appears comfortable, to see if the patient can get by with less sedation or even no sedation. Spontaneous breathing trials challenge clinicians' assumptions about patients' readiness for extubation. They invite respiratory therapists to assess patients' capacity to breathe on their own even if the patient does not appear ready for extubation. These two interventions are synergistic.[35] Performing spontaneous breathing

Figure 1. Newly Modified Bundle

ABCDEF Bundle

- **A** Assess, prevent, and manage pain
- **B** Both SAT and SBT
- **C** Choice of analgesia and sedation
- **D** Delirium: Assess, prevent, and manage
- **E** Early mobility and exercise
- **F** Family engagement and empowerment

SAT Spontaneous awakening trial
SBT Spontaneous breathing trial

trials when patients are off sedation and thus more awake increases their chances of passing the trial. Both interventions have been associated with less time to extubation and shorter ICU stays.[35-37] Observational and interventional studies also suggest they can prevent VAEs.[18, 38]

Early mobility programs

Immobility puts patients at risk for deconditioning, atelectasis, delirium, and pneumonia. Mobilizing patients on mechanical ventilation can help maintain muscle mass, improve respiratory dynamics, enhance fluid mobilization, and prevent delirium. The trial data on early mobility programs are mixed. Multiple observational studies have reported shorter times to extubation following implementation of mobility programs.[39-40] Two randomized controlled trials reported significantly shorter ICU stays in the mobility groups. Three other randomized controlled trials did not find significant decreases in duration of mechanical ventilation.[41-42] Some of the differences in these results may be attributable to patients' baseline conditioning and the relative intensity of mobilization efforts. It stands to reason that standing patients up and enabling them to walk is more likely to prevent adverse effects of prolonged immobilization compared to passive movement alone. No studies to date have specifically assessed the impact of mobility programs on VAEs but to the extent that aggressive mobilization can shorten duration of mechanical ventilation and prevent pneumonia and atelectasis it is likely to help prevent VAEs as well.

Conservative fluid management

For many years the mantra in critical care was to encourage early and aggressive fluid resuscitation for all patients with hypotension with little concern for over-resuscitation and hypervolemia on the rationale that hypotension is deadly and excess fluids can be diuresed. This has given way to a more nuanced understanding in light of an increasing body of literature suggesting that too much fluids can be as harmful as too little fluids.[43] The VAE literature also suggests the potentially harmful effects of hypervolemia. Up to 40% of VAEs in case series are attributable to volume overload, including pulmonary edema and pleural effusions.[24] Not surprisingly, then, a randomized controlled trial of depletive fluid management in patients being weaned from mechanical ventilation found that depletive fluid management was associated with more ventilator-free days and a nearly 50% decrease in VAEs compared to usual care.[44]

Restrictive blood transfusions

Blood transfusions increase risk for three of the four most common conditions that cause VAEs including ARDS, fluid overload, and pneumonia. Like excess fluids, blood transfusions can precipitate congestive heart failure and pulmonary edema. Transfusions also have immunomodulatory effects that can sometimes potentiate hospital-acquired pneumonia or catalyze ARDS. Very few investigators have directly assessed the impact of blood transfusions on VAEs but at least one study in children reported that blood products were associated with higher VAE rates.[48]

Low tidal volume ventilation

High tidal volumes are associated with increased risk for ARDS, pneumonia, and atelectasis, three of the four primary conditions that can trigger VAEs. A nested case-control study confirmed an association between tidal volumes and VAEs: every extra mL/kg ideal body weight over and above 6 mL/kg was associated with a 21% increase in the likelihood of developing VAE.[49] Randomized controlled trial data suggest that low tidal volume ventilation can prevent the development of ARDS, and in some cases, lower mortality rates in patients with ARDS.[50-52]

These six interventions currently represent our best sense of how to prevent VAEs. Much work, however, remains to be done. Not all of these interventions have been directly evaluated for effect on VAE rates or how they may best be adapted to facilitate adoption and maximize impact. Additional studies are needed to better assess the extent to which each of these measures can prevent VAEs both individually and in combination. Integrated bundles do appear to be the most potent route to reduce VAEs.[53] No investigators to date, however, have prospectively tested a bundle that includes all the measures recommended in this article.

There are some additional commonly mentioned interventions that do not appear on this list of best practices to prevent VAEs. Four in particular bear discussion: head-of-bed elevation, oral care with chlorhexidine, subglottic secretion drainage, and stress ulcer prophylaxis.

Head-of-bed elevation

The data on head-of-bed elevation are mixed. Radiolabeling and observational series suggest that recumbent patients are at increased risk for VAP, particularly when receiving enteral nutrition.[54-55] A recent Cochrane review identified 10 randomized controlled trials assessing head-of-bed elevation, three published in English and seven in Chinese.[56] The collective enrollment across all 10 trials was only 878 patients. On meta-analysis, there was a significant decrease in clinically suspected VAP but no difference in microbiologically confirmed VAP, duration of mechanical ventilation, mortality, or antibiotic utilization. There are very little data on head-of-bed elevation and VAEs: two observational studies found no association, mirroring the lack of impact on microbiologically-confirmed VAP or duration of mechanical ventilation in the meta-analysis of randomized controlled trials. The evidence in favor of head-of-bed elevation is sparse but at the same time the intervention is relatively simple, cost-free, and may yet prove beneficial if larger, more rigorous studies can be completed. Continuing to elevate the head-of-bed seems reasonable while awaiting more data.

Oral care with chlorhexidine

Oral care with chlorhexidine has long been touted to prevent VAP. This recommendation is primarily based upon early meta-analyses that reported lower VAP rates.[57-58] These analyses were potentially biased, however, by large numbers of cardiac surgery patients (most of whom are extubated within 24 hours and therefore at low risk of VAP) and failure to distinguish between blinded versus non-blinded studies (an important distinction given VAP definitions' subjectivity). An updated meta-analysis that excluded cardiac surgery patients and stratified by blinding status reported no significant impact on VAP rates and the possibility that oral care with chlorhexidine may *increase* mortality rates.[59] The investigators speculated that higher mortality rates might be due to some patients occasionally aspirating chlorhexidine leading to ARDS. The possibility of higher mortality rates has also been noted in two other studies.[18,59]

Subglottic secretion drainage

Early meta-analyses suggested that subglottic secretion drainage significantly reduced VAP rates, mean duration of mechanical ventilation, and possibly ICU length-of-stay.

> Immobility puts patients at risk for deconditioning, atelectasis, delirium, and pneumonia.

Close review of the studies included in these analyses, however, revealed substantial heterogeneity and possible misabstraction of data from one study.[60] When the studies with heterogeneous and questionable results were excluded from meta-analysis, the signal suggesting lower VAP rates persisted but differences in duration of mechanical ventilation and ICU length-of-stay between subglottic secretion drainage and control groups were no longer seen.[60] One study evaluated the impact of subglottic secretion drainage on VAE rates and found no difference, mirroring the lack of impact on duration of mechanical ventilation seen on the revised meta-analysis.[61]

Stress ulcer prophylaxis

Stress ulcer prophylaxis was originally included in ventilator bundles because of the perception that mechanically ventilated patients are at particularly high risk of developing ulcers. Recent series, however, have reported very low rates of spontaneous upper gastrointestinal bleeding compared to historical rates.[62] Moreover, stress ulcer prophylaxis may *increase* risk for pneumonia and perhaps *Clostridium difficile*.[63] This was reflected in a recent observational study that found stress ulcer prophylaxis was associated with higher rates of PVAP.[18]

Conclusions

VAE surveillance invites hospitals to review and redesign their prevention programs to better reflect emerging best practices in critical care that are more likely to lead to better outcomes for patients. The traditional VAP bundle has been found lacking in many ways: some of its components are potentially harmful (oral care with chlorhexidine and stress ulcer prophylaxis) and it does not include several interventions that are possibly beneficial (minimizing sedation, early mobilization, conservative fluid management, restricting blood transfusions, low tidal volume ventilation). VAE surveillance can help identify the most common and serious complications in mechanically ventilated patients (pneumonia, fluid overload, ARDS, and atelectasis) and thus inform the selection of more targeted and effective prevention strategies. VAE definitions are objective and potentially programmable thereby sidestepping the arguments that surveyors and clinicians used to have over VAP. Additional studies may be required, but the literature suggests that the best practices to prevent VAE may include minimizing sedation, daily coordinated spontaneous awakening and breathing trials, early mobility, conservative fluid management, restrictive transfusion thresholds, and low tidal volume ventilation. Ongoing VAE surveillance can then be used to monitor the impact of adopting these revised best practices on improving patient outcomes.

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Clinical Foundations
Panel Discussion

Airway Secretion Management of the Mechanically Ventilated Patient

Moderator: **Ruben Restrepo,** MD, RRT, FAARC

Panelists: **Brady Scott,** MS, RRT-ACCS, AE-C, FAARC

Keith Lamb, BS, RRT-ACCS, FCCM

The endotracheal tube (ETT) has been cited as a possible reservoir for infecting microorganisms in the respiratory tract. As an externally communicating foreign body, the ETT is acknowledged to constitute a risk factor for ventilator-associated infections by providing a site for microorganisms to organize, encase in a matrix (biofilm), and adhere to the surface of the ETT. This biofilm is known to be relatively resistant to antimicrobials and host defense mechanisms. In addition to the biofilm, the great majority of intubated patients experience an increased load of secretions in the airways, regardless of prior history of pulmonary disease. Therefore, the intraluminal patency of the ETT is constantly being challenged by biofilm and other airway secretions. Every available airway clearance therapeutic strategy has been designed to minimize the well-known side effects of excessive mucus in the airway including obstruction, atelectasis, and infection. In 2009, the American Association for Respiratory Care (AARC) Clinical Practice Guidelines (CPG) Steering Committee updated the CPG on Endotracheal Suctioning of Mechanically Ventilated Patients With Artificial Airways. As Chair of the committee, I tried to address on this CPG significant issues that required revision from when it was last published in 1993. Aspects of humidification, suction depth, and normal saline instillation became the highlights of the CPG. In June of 2010, our journal *Respiratory Care* published the revised CPG with evidence-based recommendations. When addressing management of airway secretions, there are typically a few questions clinicians are challenged to answer. These include: Are we following the guidelines put forward by the AARC? What can we do before secretions impact the airway? Are there changes on the ventilator parameters that can help us determine the impact of excessive airway secretions? Is additional research needed to determine the superiority of in-line versus open suctioning? What particular cautions need to be considered when managing secretions in patients with hypoxemic respiratory failure?" In this issue of Clinical Foundations, two panelists with extensive experience in the management of ventilated patients share their impressions on the impact of airway secretions.

The AARC clinical practice guideline for suctioning of artificial airways was published seven years ago. What percent of clinicians adhere to the recommended practice and how much do you believe endotracheal suction may contribute to the incidence of ventilator-associated infections?

Keith Lamb: I think that at our institution close to 100% of the RCPs follow the new AARC CPG on endotracheal suctioning. I think that a couple of the recommendations from this CPG have impacted the incidence of ventilator-associated infections. Specific to secretion management is recommendation #1, which suggests that suctioning only be performed when there are visible secretions, and #5 that discourages the routine instilling of normal saline.[1]

Brady Scott: In terms of following guidelines, I feel that respiratory therapists follow the guidelines closely. However, despite proper suctioning practices, excessive airway secretions may still occur. Fortunately, the practice of using normal saline regularly during suctioning has been significantly reduced over the years.

Ruben Restrepo: As primary author of CPG, I am very happy that the practice of deep-suctioning and routine normal saline instillation has dramatically decreased.[1] Long ago, Inglis had suggested that fragments of the ETT biofilm may be dislodged upon insertion of the suction catheter and carried further into the lung by ventilator gas flow.[2] That being said, both practices for suctioning, shallow and deep, would equally affect the dislodgement of any accumulated secretions into the lower part of the airway and potentially contribute to increasing the rate of ventilator-associated infections. That is why I believe Keith's point on the drastic reduction of unnecessary suction events along with normal saline instillation could be more important in reducing these infections than the depth of suction itself.

What measures are employed to remove secretions before either impacting airway resistance or promoting ventilator-associated infections (VAI)?

Keith Lamb: Once a ventilator-associated infection is identified, most efforts are focused on resolving the infection. Anecdotally, I have had a handful of patients with difficulty being liberated from mechanical ventilation that have been morbidly obese and have been found to have a large airway secretion burden in the endotracheal tube. This has become only obvious during tracheostomy. My impression is that when a patient has excessive airway obstruction due to secretions, that the increased airway resistance can have a big impact on their ability to overcome that impedance and delays successful extubation.

Brady Scott: I agree with Keith. Once the infection has started, our attention is on the treatment. With that said, I admit that we do mention "biofilm" more now than in previous years due to the evolving literature on biofilm buildup.

Ruben Restrepo: I agree with both. I don't believe that as important as airway obstruction by biofilm or airway secretions is, the term "biofilm" comes up in any discussion when respiratory therapists give report or when physicians round. It has been confirmed that ETT biofilm is present in almost 100% of the intubated patients and that it develops within hours of intubation with pathogens present in high concentrations.[3-7] I want to add that except for optimizing humidification and tracheal suctioning, no other measures are taken to remove secretions before they cause increased airway resistance or VAI. One of the best explanations we can give to what factors promote secretion build up is that the presence of an ETT inhibits host protective responses such as the cough reflex and ability to clear secretions. The presence of chronic respiratory conditions, patient acuity, hydration status, ETT size, number of suction events, use of antibiotics prior to intubation, type of humidification device used, trauma during intubation, or even ventilatory modes come to mind but I don't believe anyone has found a correlation between these, biofilm formation and VAI. To Keith's previous point, if VAP bundles are used, do patients have better staging of biofilm or better secretion control? Don't know the answer to this either. Several authors have described that the presence of pathogens on the ET may occur as a result of gastropulmonary reflux, or from a colonized oropharynx.[8,9] Should we routinely address reflux to prevent airway colonization? I don't believe the evidence is that strong either.[8,9]

> One of the best explanations we can give to what factors promote secretion buildup is that the presence of an ETT inhibits host protective responses such as the cough reflex and ability to clear secretions.
>
> *- Ruben Restrepo -*

Some of us believe that not enough attention has been paid to optimal humidification for intubated patients. Does type of humidification used in ventilated patients play a role on secretion formation and incidence of ventilator-associated infections?

Keith Lamb: One would certainly think so. Inadequate humidification to the airways impacts the native mucocilliary mechanism and viscosity of secretions. Heated and humidified inspired gas should be provided by active systems. Although it seems intuitive, I am not sure that the literature supporting this is robust.

Brady Scott: It is not yet known if the type of humidification has an impact on secretion buildup inside of an endotracheal tube. There have been studies that attempt to determine the impact of humidification devices on ventilator-associated infections. The last published Cochrane meta-analysis that compared heat moisture exchangers with heated humidifiers showed no differences in VAP occurrence.[10]

Ruben Restrepo: Brady is right. As far as I could research, type or adequacy of humidification has not been specifically linked to airway secretion buildup. I agree with Keith that it would make sense, since pooling of mucus may provide an ideal medium for bacteria to aggregate.[11] However, I also think that it could have a greater influence on the secretions moving back and forth inside the lumen of the ETT. We just need more information on how lack of mucus clearance directly affects the formation and/or staging of the microbial matrix.

When there is an increase in airway pressures, how often is increased airway secretions suspected and what steps are taken?

Ruben Restrepo: Most clinicians focus on outcomes related to the presence of biofilm or excessive secretions, such as airway resistance. Airway occlusion is always suspected when there is an abrupt change in airway pressure. A combination of physical assessment and analysis of waveforms often confirms airway obstruction before bronchoscopy is indicated. And although airway resistance could significantly impact ventilator settings, ability to wean, patient comfort, and synchrony with the ventilator, this topic has not been as well studied as it should. Wilson *et al* reported that biofilm itself could significantly increase airway resistance measured by the pressure drop of ETTs and that it was unpredictable relative to the duration of intubation.[7] Although only published in abstract form, we have reported that in a small sample of patients (n=17) extubated in a medical ICU, the average percent of endotracheal tube occlusion was around 10.4%, which is consistent with reports by Van Surel and Shah.[12,13] However, ETT degree of obstruction has been reported as high as 25%[14] to 50%.[15,16] Similarly to Wilson's report, we found no correlation between length of intubation and degree of ETT obstruction.[17] The most conventional method used to resolve airway obstruction caused by secretions is tracheal suction through a catheter connected to negative pressure. The use of other devices can be considered but the evidence to support their routine use is limited.

Brady Scott: Ruben, you make a good point about the outcomes related to the presence of biofilm and airway secretions. In a study we conducted at our institution, we sought to better understand how much airway resistance would change, if any, when using a device to remove intraluminal secretions. We found a statistically significant decrease in airway resistance before and after using the device.[18]

What role do in-line suction catheters play in the overall reduction of VAE?

Ruben Restrepo: Endotracheal suctioning is a procedure that may constitute a risk factor for VAE. It can be performed with an open suction system (OSS) or with a closed in-line suction system (CSS). In view of suggested advantages being reported for the CSS, a couple of systematic reviews comparing both techniques were conducted in the last ten years. Results from 16 trials showed that suctioning with either CSS or OSS did not have an effect on the risk of ventilator-associated pneumonia or mortality.[18,19] In one of those trials, Lorente et al analyzed the prevalence of ventilator-associated pneumonia (VAP) using a closed-tracheal suction system (CSS) vs. an open system (OSS) in 443 patients requiring mechanical ventilation for >24 hrs. No significant differences were found in either the percentage of patients who developed VAP (CSS 20.47% vs. OSS 18.02%) or in the number of VAP cases per 1000 mechanical ventilation-days (CSS 17.59 vs. OSS 15.84). There were also no differences in the VAP incidence by mechanical ventilation duration.[20] Changing a CSS weekly or as-needed has been associated with significant cost reduction without a higher rate of ventilator-associated events.[21] One of the most recent open-labeled randomized controlled trials compared costs and clinical outcomes of OSS with CSS in 200 mechanically ventilated medical intensive care patients. CSS was associated with a trend to a reduced incidence of VAP (P = 0.067). A significant benefit was, however, observed with CSS for late-onset VAP (P = 0.03). Mortality and duration of ICU and hospital stay were similar in the two groups. The cost of suction catheters and gloves was significantly higher with CSS.[22]

> Inadequate humidification to the airways impacts the native mucocilliary mechanism and viscosity of secretions.
>
> *- Keith Lamb -*

Are there any special precautions or maneuvers that need to/or should be utilized when suctioning patients with ARDS?

Ruben Restrepo: Patients with ARDS or severe hypoxemic respiratory failure are more susceptible to side effects associated with endotracheal suctioning. This group of patients is routinely placed on higher positive end expiratory pressure (PEEP) levels, lower tidal volumes, and higher inspired fraction of oxygen (FiO_2). Applying negative pressure increases the risk for tidal volume reduction, PEEP reduction, and atelectasis. Although the recommendations we made in the CPG should apply to any patient who is intubated, special precautions should be considered in this patient group. These recommendations include: routine pre-oxygenation prior to the event, using in-line suction systems, limiting the suction event to no more than 10 to 15 seconds, applying not more than 150 mm Hg of negative pressure, and contemplating recruitment after suctioning. However, there is no evidence that these measures impact clinical outcomes.

Is there a benefit to "sweeping" or "scraping" the interior lining of the ETT as compared to suctioning the ETT?

Keith Lamb: Yes. There are a couple of devices that I am familiar with and have used. These devices have been proven safe and effective at removing secretions. Moreover there have been small studies reporting a reduction in ventilator-associated infections with the routine use of these devices.[23,24]

Brady Scott: There has been some discussion about using the tube cleaning devices available on the market now, (see example of device Figure 1) in addition to suctioning, to keep tubes patent and free of secretions. While this sounds promising, it remains to be seen if doing routine tube cleaning will impact measures such as length of mechanical ventilation, infection rates, etc. Recently, our facility was involved in a study that looked at impact of biofilm/secretion buildup on airway resistance in the endotracheal tube. We found a statistically significant decrease in airway resistance before and after using the sweeping device. The decreasein airway resistance from pre- to post-tube scraping was from 15.17 ± 3.83 cm H_2O/L/s and 12.05 ± 3.19 cm H_2O/L/s, respectively ($p < 0.001$).[18,25] This, along with the newer research like the randomized control trial by Pinciroli et al, where they looked at a way to reduce luminal narrowing that can happen inside of an ETT due to secretion buildup, have resulted in a bit more focus on the issue.[14] Another device was also evaluated by Berra et al in 2012. They too found the device to be safe, effective, and efficient for endotracheal tube cleaning.[11] It is hard to say that these devices can reduce ventilator-associated infections at this time. There is certainly evidence that pathogen-containing secretions can be removed, but it is unclear if this impacts pneumonia incidence. The study by Bardes et al did not find a statistically significant reduction in pneumonia when Endoclear® (Endoclear® LLC, Petoskey, Michigan) was used.[6] It is worth mentioning that this study was small and more studies are needed to understand if the removal of biofilm can result in the overall reduction in pneumonia in ventilated patients.

Ruben Retrepo: I agree with Brady. Novel devices have been designed to clear mucus and debris from an ETT in order to restore luminal patency but the number of studies to support their routine use is very limited. In addition, some of the devices tested are not commercially available. Pinciroli and his group have reported in two different articles the effects of removing ETT secretions with an Endoclear.® First, on three life-threatening cases[25] and later last year in the clinical trial already mentioned by Brady. This clinical trial showed a trend towards a reduced biofilm in 37 treated ETTs.[15] It is also clear that the use of any ETT cleaning device has limited, if any role at all, on the formation of biofilm. Although the use of an additional antibacterial coating may be required to impact the incidence of ventilator-associated infections, it has been known that bacteria encased in this biofilm are relatively resistant to antimicrobials and host defenses.[26,27]

Figure 1. Cleansweep™. Teleflex, Morrisville, NC

When using a device to remove intraluminal secretions, we found a statistically significant decrease in airway resistance before and after using the device.

- Brady Scott -

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Ruben Restrepo, MD, RRT, FAARC is Professor and Director of Bachelor's Degree Completion program in the Department of Respiratory Care at the University of Texas Health Science Center at San Antonio, Texas. Trained in medicine in Medellin, Columbia, Dr. Restrepo went on to academic positions at Georgia State University and then at the University of Texas. He has been published widely in the field of respiratory care, including book chapters and articles, and has given many presentations. He is a reviewer for several major journals in respiratory and pulmonary medicine and is the recipient of many honors and awards for his teaching, publications, and volunteer work.

Keith Lamb, BS, RRT-ACCS, FAARC is currently the supervisor of Adult Critical Care, Department of Respiratory Care, at UnityPoint Health System, Des Moines, Iowa. Keith's responsibilities include oversight of all adult respiratory critical care, research development, quality improvement, critical care education, and bedside clinical critical care. He also serves as the Extracorporeal Life Support Coordinator and as the Vice-Chair of Society of Critical Care Medicine, Respiratory Section. Keith has written and published numerous peer-reviewed articles pertaining to critical care, and lectured locally, nationally, and internationally.

J. Brady Scott, MSc, RRT-ACCS, AE-C, FAARC, FCCP is the director of clinical education and assistant professor for the respiratory care program. He has been a respiratory therapist for more than 15 years, with clinical practice experience in adult emergency/critical respiratory care. In 2007, he was named the Adult Acute Care Specialty Practitioner of the Year by the American Association for Respiratory Care (AARC). He has lectured at regional, state, national, and international conferences on topics pertaining to respiratory care. His research interests include simulation-based education and emergency/critical respiratory care. Brady is a Fellow of the American Association for Respiratory Care and the American College of Chest Physicians.

Ventilator-Associated Events: New Outcome Measure- Continued

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Michael Klompas, MD, MPH, FRCPC is the Associate Hospital Epidemiologist at Brigham and Women's Hospital in Boston. He attends on the infectious disease and internal medicine services of Brigham and Women's Hospital. He has published widely on surveillance, diagnosis, prevention, and treatment of ventilator-associated pneumonia, ventilator-associated events, and sepsis. He was a member of the American Thoracic Society – Infectious Disease Society of America panel that recently released revised guidelines for the management of hospital-acquired pneumonia. He was also co-chair of the SHEA panel that revised the Compendium of Strategies to Prevent Ventilator-Associated Pneumonia. Additionally, Dr. Klompas works on enhancing public health surveillance using electronic health information in two major spheres: hospital-level surveillance for nosocomial complications (supported by the CDC) and population-level surveillance for infectious diseases and chronic conditions.

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Questions

1. **What are the most common conditions that trigger ventilator-associated events?**
   A. Pneumonia, pulmonary embolism, pneumothorax, sepsis
   B. Pneumonia, pulmonary edema, ARDS, atelectasis
   C. Pulmonary edema, ARDS, atelectasis, pulmonary hemorrhage
   D. Pneumonia, ARDS, pneumothorax, pulmonary fibrosis

2. **Which of the following strategies are most likely to prevent VAEs?**
   A. Low tidal volume ventilation
   B. Elevating the head of the bed
   C. Daily spontaneous breathing trials
   D. Oral care with chlorhexidine

3. **Which of the following interventions was associated with fewer VAEs and more ventilator-free days in a randomized controlled trial?**
   A. Oral care with chlorhexidine
   B. Conservative fluid management
   C. Early mobilization
   D. Restrictive blood transfusions

4. **Which of the following interventions may increase pneumonia risk?**
   A. Oral care with chlorhexidine
   B. Conservative fluid management
   C. Subglottic secretion drainage
   D. Stress ulcer prophylaxis

5. **Why is it hypothesized that low tidal volume ventilation may lower VAE risk?**
   A. Low tidal volume ventilation is associated with lower rates of pneumonia
   B. Low tidal volume ventilation is associated with lower rates of ARDS
   C. Low tidal volume ventilation is associated with lower rates of atelectasis
   D. All the above

6. **According to the last published AARC clinical practice guidelines on endotracheal suction, normal saline should be routinely instilled prior to suctioning the artificial airway.**
   A. True
   B. False

7. **The AARC CPG on endotracheal suctioning recommends that the suction catheter be introduced:**
   A. 3 cm above the carina
   B. until the catheter meets resistance and then it should be withdrawn prior to apply negative pressure
   C. to the end of the endotracheal tube
   D. after normal saline is instilled directly into the trachea

8. **Only deep suctioning is associated with the risk of biofilm dislodgement into the lower airway**
   A. True
   B. False

9. **For which of the following strategies is there clinical evidence that its use reduces the impact of biofilm and could potentially reduce the incidence of VAP?**
   A. Avoidance of normal saline intratracheal instillation
   B. Shallow suctioning (to the end of the ETT)
   C. Sweeping/scraping the interior lining of the ETT
   D. Active humidification

10. **According to the latest meta-analysis, which humidification strategy reduces the incidence of VAP?**
    A. Active humidification
    B. Passive humidification
    C. Use of large volume nebulizers
    D. None of the above

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Participant's Evaluation

1. What is the highest degree you have earned? Circle one. 1. Diploma 2. Associate 3. Bachelor 4. Masters 5. Doctorate
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Upon completion of this activity, the participant will be able to:

1. Recognize the impact of humidification on biofilm formation and optimal strategies to manage airway secretions.
   Strongly Agree Strongly Disagree
   1 2 3 4 5 6
2. Explain why the Centers for Disease Control and Prevention switched from ventilator-associated pneumonia surveillance to ventilator-associated events surveillance.
   Strongly Agree Strongly Disagree
   1 2 3 4 5 6
3. List best practices to prevent ventilator-associated events.
   Strongly Agree Strongly Disagree
   1 2 3 4 5 6
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   Strongly Agree Strongly Disagree
   1 2 3 4 5 6

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3	A ☐	B ☐	C ☐	D ☐
4	A ☐	B ☐	C ☐	D ☐
5	A ☐	B ☐	C ☐	D ☐
6	A ☐	B ☐	C ☐	D ☐
7	A ☐	B ☐	C ☐	D ☐
8	A ☐	B ☐	C ☐	D ☐
9	A ☐	B ☐	C ☐	D ☐
10	A ☐	B ☐	C ☐	D ☐

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