COMMENTARY

Shifting the Paradigm: Preventing More Than Infection

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In the field of infection control, our work is focused on how we prevent infection on a day-to-day basis. For years, we have endeavored to prevent infections secondary to urinary catheters, central venous catheters, and ventilators, among many other devices and conditions. Controversy has always surrounded surveillance versus the clinical definitions applied; however, ventilator-associated pneumonia (VAP) definitions have been so subjective and cumbersome to record that in 2013, a new definition algorithm was implemented. The new algorithm broadly identifies Ventilator-associated events (VAEs) under which several subcategories exist, including: ventilator-associated conditions (VACs), infection-related ventilator-associated conditions (IVACs), and probable ventilator-associated pneumonia (PVAPs).[1–12]

Historically, infection prevention programs have used one of several bundled care programs as a strategy to prevent VAP. However, demonstrating consistent efficacy of such bundled care has been severely limited by the subjective nature of the VAP definition.[13] With the implementation of the new definitions, the standard bundle is inadequate because the prevention initiatives must now include a broad scope of respiratory complications beyond infections.[1,3,6,7,12,14–18]

Ventilator-associated events are more objective and can be automated for detection because they are determined by specific changes in the fraction of inspired oxygen (FiO_2) and positive end expiratory pressure (PEEP). Therefore, many other noninfectious conditions associated with mechanical ventilation may trigger a VAE: atelectasis, acute respiratory distress syndrome (ARDS), fluid overload resulting in pulmonary edema or pleural effusions, transfusion-related lung injury, traumatic injury to the chest, barotrauma or volume-related trauma from the ventilator, pneumothorax and pulmonary emboli, among others.[1–7,9,11,12,15,17–20]

The inclusion of the noninfectious causes has resulted in much scrutiny of the new definitions. Ventilator-associated events are associated with increased costs, duration of mechanical ventilation, ICU length of stay and mortality; however, there are discrepancies between the new and old definitions in the detection of VAP.[2–12,14–16,18,19] Whether or not the VAE algorithm identifies VAP as well as the old remains controversial, particularly because PVAP comprises only ~10%–30% of VAE cases.[2,3,9–12,15,17–19] If definitions of VAP were poor to start with, comparisons will be fraught with variability: the data are only as good as the initial definition of a case.

Ventilator-associated events have also been touted as a possible quality metric given their association with outcomes and even antimicrobial utilization, but considering the risk of future punitive action for high VAE rates, the question of preventability has moved to the forefront of the conversation.[2,4,7,10,12,14,17,18] Can VAE's be prevented?

Preventable VAEs have been reported at varying rates in different studies ranging up to ~50% (actual and potential prevention combined). Thus, there is clearly opportunity to prevent some, albeit likely not all, VAEs.[3–5,10,14,18–20]

In the study "Ventilator Bundle Compliance and Risk of Ventilator-Associated Events," Harris et al[1] addressed whether or not decreased VAP bundle compliance increased the risk of VAEs in a matched, single-center, case-control study of adults. Ventilator bundle success with decreased VAP and overall compliance was previously established via an electronic dashboard in the medical record and consisted of the following elements: elevation of the head of the bed, daily assessment for spontaneous breathing trials with completion if appropriate, assessment of agitation via the Richmond agitation sedation scale, oral care (brushing teeth, oral chlorhexidine swabs, and hypopharyngeal suctioning), and prophylaxis against both stress ulcers and deep vein thrombosis.

Compliance was assessed on days 3 and 7, and no association was detected between compliance and the development of a VAE. Because the bundle was focused on the prevention of VAP according to the old definition, an analysis of IVAC was also completed as a surrogate for VAP, and the lack of association held true for IVACs. Interestingly, chlorhexidine mouth swabs were specifically noted in all analyses to be associated with increased rates of VAE.[1]

Based on this study, compliance with previously recommended VAP bundles may not impact VAE rates. Although discouraging at first, this finding is not entirely unexpected nor does it indicate that VAE is not preventable. In fact, this outcome may provide the impetus and opportunity to increase multidisciplinary collaboration to ensure that best practices for ventilated patients are implemented. The paradigm of prevention now extends far beyond infection only and incorporates goals of optimizing the decision to pursue endotracheal

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intubation, the management of mechanically ventilated patients, and the duration of mechanical ventilation.[1]

If we consider the 4 most frequently cited underlying etiologies—pneumonia (VAP), ARDS, atelectasis, and fluid overload—as separate entities, prevention practices extend beyond the current scope of many infection prevention groups. But with evolving evidence that these entities can be prevented and thereby also prevent VAEs, perhaps it is time to reconsider the scope of practice.[6,14,19] New bundled approaches need to focus on each of these etiologies in a multifaceted approach to improvement.

Prevention strategies for VAP already exist (disregarding variances in reported efficacy), so I will not be belabor them here. However, many of the best practices regarding prevention of the noninfectious etiologies also decrease VAP through shorter durations of intubation, improved clinical status, and decreased risk factors for development of pneumonia.[6,7,12,13,16,17] Notably, management by intensivists has also been associated with improvement in VAP rates.[20]

For decades, research has focused on refining the definition of ARDS and treating ARDS; however, pharmacologic options have failed to result in significant improvement in ARDS. Improved ventilator management with low tidal volumes, optimized PEEP for each individual patient (decreasing atelectasis and thereby optimizing oxygenation capacity through pressure), and decreased overall driving pressure of each breath from the ventilator, decrease ARDS via prevention or limitation of iatrogenic lung injury.[18,20–22] Subsequently, ARDS research is moving toward a primary strategy of ARDS prevention. Such prevention requires early and effective decisions regarding how patients are managed beyond the ventilator settings, including conservative fluid and transfusion utilization.[18,21,22] ARDS prevention synergistically may help prevent VAEs because it focuses on prevention of infection, atelectasis and fluid overload with subsequent development of pulmonary edema, among other best practices for critically ill patients.[18,21,22] Furthermore, nonintensivist management of mechanically ventilated patients has also been identified as a potential risk factor for ventilator-associated conditions. Therefore, addressing who manages ventilated patient may be a critical component to decreasing complications overall.[20]

When these strategies are combined with a primary overarching goal of decreasing patient time on mechanical ventilation, prevention efforts will include the basic goals of endotracheal intubation only if needed (with extubation as soon as possible), early mobilization and sedation awaking trials (with optimization of sedation choice), paired with spontaneous breathing trials (to decrease the duration of mechanical ventilation).[4,6,7,12–14,17–21] Sedation awaking trials and spontaneous breathing trials were specifically shown to decrease VAEs in the Wake Up and Breathe Collaborative.[4]

Where do we go from here? We know that VAP bundles are not enough and that the number of VAEs that can be prevented is unclear. But arguably, the primary underlying etiologies of VAE present opportunities to decrease incidence through best practices in each area. Smarter, better models incorporating decreasing overall risks of invasive mechanical ventilation are needed, and these strategies may no longer consist of a simple bundle but rather a protocol and method to bring the best evidence-based care surrounding invasive mechanical ventilation to the bedside.[3,12,13,16–21]

Clearly, further study is needed, but the ventilator bundle research must be expanded to include concurrent use of early optimization of ventilator settings (eg, low tidal volume for all patients and adequate PEEP to prevent atelectasis), careful management of fluid status to minimize pulmonary edema, transfusion threshold compliance, early mobility and sedation awakening trials with spontaneous breathing trials combined with the elements of preexisting VAP bundles.[1,4,12–14,16–20] These goals are ambitious, and they shift our paradigm from infection prevention to optimizing overall care and outcomes among our ventilated patients. Yet, in the most simplistic form, the dogma of medicine remains the primary driver for how to prevent ventilator-associated events: we must do what is best for the patient.

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