Ventilator-associated pneumonia is ubiquitous and troublesome

Hospital-acquired infections are a source of increased morbidity and mortality in patients admitted for all severities of disease. Patients requiring intensive care are at significantly increased risk owing to the nature of their illness and the need for invasive procedures that include mechanical ventilation, vascular access, and drainage tubes of all kinds. Ventilator-associated pneumonia (VAP) is one of the most common manifestations of hospital-acquired infection and is associated with increased intensive care and hospital stay, as well as increased mortality.[2]

Mazwi et al.[3] have added a useful description of VAP from an intensive care unit (ICU) in the developing world. They demonstrated a moderate rate of VAP, in line with figures from other parts of the world (16.4 per 1 000 ventilator days), with global figures ranging between 9.0 and 18.0 per 1 000 ventilator days.[4] Later-onset VAP was associated with the isolation of multidrug-resistant organisms and significantly increased mortality. This study was conducted between March 2013 and December 2015, after the time when global awareness of VAP increased and various care bundles were introduced that have been associated with a decreased prevalence of VAP.[4] This was also shortly before the current problems of increasingly drug-resistant organisms with carbapenem-resistant Enterobacteriaceae and Acinetobacter species.

VAP remains a difficult problem for intensivists. A number of ICU-related events can mimic pneumonia, and colonisation of patients with organisms is extremely common. These make diagnosis difficult, with no universally accepted definition for VAP. One of the most widely used clinical approaches uses clinical suspicion of pneumonia with an infiltrate on the chest radiograph plus any one of leucocytosis, fever >38.3°C, or purulent tracheobronchial secretions.[5] This method lacks precision, with an autopsy study showing that it had sensitivity of 69% and specificity of 75%.[6] The Clinical Pulmonary Infection Score[7] was similarly imprecise, with sensitivity of 77% and specificity of 42%. Adding detection of organisms may increase the diagnostic precision, but this often requires invasive investigation with lavage and quantitative culture.[8] Although more rapid detection and determination of resistance patterns of significant organisms by novel technologies that include polymerase chain reaction multiplex panels are potentially useful, studies to date have not demonstrated major improvements in outcome compared with conventional techniques.[9]

The lack of specificity for the diagnosis of VAP has been of concern over the years, partly because of the lack of an appropriate gold standard other than histopathology. The Centers for Disease Control attempted to formalise a surveillance definition of VAP in 2012.[10] Acknowledging the wide differential diagnosis of pulmonary-based complications in ventilated patients, a ventilator-associated event (VAE) surveillance definition was formulated. The first level was ventilator-associated condition (VAC), marked by a deterioration in oxygenation (need for an increase in the fraction of inspired oxygen or positive end-expiratory pressure 48 hours after stability has been achieved). However, VAC can include many ICU-related events, but the suspicion of infection (new fever or leucocytosis) coupled with starting a new antimicrobial agent elevated the grading to infection-related VAC, which could then be classified as possible or probable VAP depending on the findings on microbiological investigation.[10]

Despite the logical nature of this surveillance definition, a number of studies have shown that it too has problems, and a recent meta-analysis suggested that the VAE approach missed up to 50% of cases of VAP with overall sensitivity <50%, although specificity reached 80%.[11] The lack of precision of the VAE surveillance approach for the diagnosis may have implications for epidemiology and intervention studies, and the discordance between VAE and VAP at the clinical level needs to be recognised.[12] Consensus diagnostic criteria are still lacking, which makes comparison of VAP incidence rates between institutions and nations difficult.[3]

The increasing burden of multidrug-resistant organisms and the mortality and morbidity associated with VAP make prevention paramount. The many processes involved in management of critically ill patients make VAP likely, and considerable attention has been paid to various interventions and bundles of care to prevent VAP. There has been considerable success with these approaches, with a marked decline in VAP rates, although zero VAP may not be achievable.[13] Mazwi et al.[14] discuss prevention of VAP as an essential component of management. A number of interventions have been suggested as part of prevention strategies and bundles, some of which, such as venous thromboembolism prophylaxis, may reduce VAC but not VAP. A particular area of concern related to VAP prevention in less well-resourced countries is ICU staffing levels, with an increased staff-to-patient ratio associated with an increased incidence of VAP, particularly late-onset VAP.[14] Effective VAP prevention approaches to date have included non-pharmacological measures related to endotracheal tube management and patient positioning. Shortening the duration of intubation using sedation and weaning policies[15] and avoiding intubation by the use of non-invasive ventilation or high-flow nasal cannula oxygen administration are effective, as are semi-recumbent positioning and subglottic secretion drainage.[16,17] Pharmacological measures, including selective oral or digestive tract decontamination, have been effective in some areas, but are often difficult to implement and are costly.[18] The most important and cost-effective recommendation for prevention of all hospital-acquired infections remains hand hygiene.[19]

Richard I Raine, MB ChB, MMed (Med), FCP (SA) a
Department of Pulmonology, Division of Medicine, Groote Schuur Hospital and University of Cape Town, South Africa
richard.raine@uct.ac.za


