

Clinical Foundations

*A Patient-focused Education Program
for Respiratory Care Professionals*

**Free
Continuing Education
for Respiratory
Therapists (CRCE)
and Nurses (CE)**
See Page 12

Advisory Board

Janet Boehm MS, RRT
Director, Clinical Education
Youngstown State University
Youngstown, OH

Richard Branson MS, RRT, FAARC
Associate Professor of Surgery
University of Cincinnati College of Medicine
Cincinnati, OH

Richard Kallet MSc, RRT, FAARC
Clinical Projects Manager
University of California
Cardiovascular Research Institute
San Francisco, CA

Donna Hamel RRT, FAARC
Clinical Research Coordinator
Duke University Health Systems
Raleigh-Durham, NC

Neil MacIntyre MD, FAARC
Medical Director of Respiratory Services
Duke University Medical Center
Durham, NC

Tim Myers BS, RRT-NPS
Pediatric Respiratory Care
Rainbow Babies and Children's Hospital
Cleveland, OH

Tim Op't Holt EdD, RRT, AEC, FAARC
Professor, Department of Respiratory Care
and Cardiopulmonary Sciences
University of Southern Alabama
Mobile, AL

Ruth Krueger Parkinson MS, RRT
Protocol/PI Coordinator
Sioux Valley Hospital
Sioux Valley, SD

Helen Sorenson MA, RRT, FAARC
Assistant Professor, Dept. of Respiratory Care
University of Texas Health Sciences Center
San Antonio, TX

www.clinicalfoundations.org

Visit Clinical Foundations online at
www.clinicalfoundations.org
Archives • Free CRCEs

Pathogens Associated with the Intensive Care Unit Environment – Considerations for the Respiratory Therapist

by John Davies, MA RRT FAARC

Ventilator-associated pneumonia (VAP) is the most common nosocomial pulmonary infection and accounts for a significant proportion of the 1.7 million infections and 99,000 associated deaths each year in American hospitals. Methicillin-resistant *Staphylococcus aureus* (MRSA) and vancomycin-resistant enterococcus (VRE) are of growing concern due to their ease of transmission via medical devices and hospital personnel. Other pathogens causing VAP include *Staphylococcus aureus*, *Streptococcus pneumoniae* and *Hemophilus influenzae*, *Pseudomonas aeruginosa*, *Acinetobacter* species and *Enterobacter* species. A clear diagnosis of VAP requires a combination of findings from clinical signs, chest X-ray, and analysis of bronchial alveolar lavage. Although there are several natural defense systems against infection, these are often breached through the use of indwelling devices, such as catheters, and inadequately sterilized mechanical ventilation equipment, such as endotracheal tubes, ventilator circuits, humidifiers, medication nebulizers and suction catheters. Methods to prevent VAP include effective handwashing with antimicrobial preparations, education of hospital staff, thorough sterilization of equipment, and implementation of protocols such as the VAP bundle. Modified equipment is now available to limit the likelihood of infection.

Panel Discussion: Ventilator-Associated Pneumonia and the Role of the Respiratory Therapist

Moderator: Harvey E. Marshall, MD

Panelists: Stephen Kantrow MD

Ruben Restrepo MD, RRT, FAARC

Kathleen Arias MS, CIC

Ventilator-associated pneumonia (VAP) is a significant cause of morbidity and mortality in the ICU setting. As the healthcare professional who is primarily responsible for care of the ventilated patient, it is essential that the respiratory therapist understand the factors that lead to the development of VAP and employ interventions such as the use of specialized endotracheal tubes and implementation of VAP bundles that are proven to decrease its incidence. Identification of VAP is not a straightforward task and antibiotic treatment guidelines are highly variable. Through a better understanding of the likely microbial pathogens, the respiratory therapist can also assist with VAP diagnosis and treatment. A team-oriented approach is required to improve VAP clinical outcomes. In this panel, 3 experts discuss strategies for preventing and treating VAP.

Pathogens Associated with the Intensive Care Unit Environment – Considerations for the Respiratory Therapist

John Davies, MA, RRT, FAARC

Many potentially harmful pathogens exist throughout the hospital, especially in intensive care units (ICUs). Patients in these areas are generally sicker and tend to have compromised immune systems. Along with contact transmission, these patients have many other routes for pathogen transmission into the body. For example, indwelling catheters and endotracheal tubes can act like a superhighway for bacterial invasion. Fortunately, several strategies have been devised to help prevent ventilator-associated pneumonia (VAP) in the hospital setting. Guidelines created by the Society for Healthcare Epidemiology of America (SHEA) outline reasons for concern, identify strategies to prevent and combat VAP, and provide recommendations for implementing, monitoring, and measuring performance of VAP programs.¹⁻² Respiratory therapists are key players in the fight against VAP, but in order to be effective, they must stay current on emerging guidelines and recommendations. In this article, we discuss common pathogens associated with ICU infections and how these infections can be prevented, with a special emphasis on the role of the respiratory therapist.

Nosocomial pathogens in the ICU commonly affect the urinary tract, the bloodstream or the pulmonary system. The main organisms producing bloodstream and urinary tract infections include *Staphylococcus aureus* (Gram posi-

According to the Centers for Disease Control and Prevention (CDC), healthcare-associated infections account for an estimated 1.7 million infections and 99,000 associated deaths each year in American hospitals alone.

tive) and *Enterobacter* species (Gram negative). Other Gram negative pathogens that cause urinary and bloodstream infections include *Acinetobacter*, *Serratia*, *Klebsiella*, *Pseudomonas aeruginosa* and *Escherichia coli*. These microorganisms generally gain access to the host through indwelling catheters, such as foles, CVCs and PICCs.

Ventilator-associated pneumonia is the most common nosocomial pulmonary infection³ and is divided into 2 categories: Early and late onset. Early onset VAP occurs 48-96 hours after intubation. Pathogens causing early onset VAP

include *S. aureus* (Methicillin-sensitive), *Streptococcus pneumoniae* and *Hemophilus influenzae*, as well as other less common agents. In general, these pathogens are susceptible to antibiotic therapy. Late onset VAP is usually caused by antibiotic-resistant organisms such as *P. aeruginosa*, Methicillin-resistant *S. aureus* (MRSA), *Acinetobacter* species and *Enterobacter* species (Table 1). The microorganisms responsible for producing VAP may differ depending on patient population as well as duration of stay in the ICU. However a meta-analysis involving 24 different studies (1689 episodes) found that the most common pathogens were *P. aeruginosa* (24.4%), *S. aureus* (20.4%, both Methicillin-sensitive and resistant strains), *Enterobacter* species (14.1%, *E. coli* and *Proteus* species were the most prevalent in this group), *H. influenzae* (9.8%) and *Streptococcus* species (8.0%).⁴

How important is it to prevent the spread of nosocomial infections? According to the Centers for Disease Control and Prevention (CDC), healthcare-associated infections account for an estimated 1.7 million infections and 99,000 associated deaths each year in American hospitals alone. A 2002 report identified a total of 394,288 ICU-associated infections³. Pneumonia and urinary tract infections accounted for 26% of this total while bloodstream and surgical site infections accounted for 21% and 7%, respectively. Today MRSA is of particular concern in acute care settings due to its ease of transmission by contact and its potent resistance to antibiotics, rendering many of the accepted treatment regimens useless. While antibiotic therapy is out of the scope of this discussion, it is important to bear in mind that antibiotic resistance imparts a new dimension of therapeutic complexity.

MRSA was first isolated in the United States in 1968. By the early 1990s, MRSA accounted for 20% to 25% of *S. aureus* isolates from hospitalized patients.⁵ In 1999, MRSA accounted for

>50% of *S. aureus* isolates from ICU patients in the National Nosocomial Infection Surveillance (NNIS) system; in 2003, 59.5% of *S. aureus* isolates in NNIS ICUs were MRSA⁶. A study in 2007 estimated that 94,360 invasive MRSA infections occurred in the United States in 2005 and that these infections were associated with death in 18,650 cases.³ It was also estimated that bacteremia was the result in 76% of the MRSA infections while pneumonia was 16%³.

Vancomycin-resistant *enterococcus* (VRE) shares a commonality with MRSA in its ease of transmission through contact and in antibiotic resistance. A similar rise in prevalence has occurred with VRE. From 1990 to 1997, the prevalence of VRE in enterococcal isolates from hospitalized patients increased from <1% to approximately 15%.⁷ VRE accounted for almost 25% of enterococcus isolates in NNIS ICUs in 1999 and 28.5% in 2003.^{6,8}

Accurate data on the epidemiology of VAP are limited by the lack of standardized criteria for its diagnosis. Generally speaking, VAP can be defined as the inflammation of lung parenchyma caused by infectious agents that were not present in the lower respiratory tract at the outset of mechanical ventilation. Definitive diagnosis of VAP is difficult and is usually based on systemic signs of infection, new or worsening infiltrates on chest x-ray, and bacterial evidence of parenchymal infection. Bacterial evidence is usually obtained from bronchial alveolar lavage fluid.⁹ Tracheal aspirates are more often than not inconclusive for use as a diagnostic tool. However, sampling of the upper airway secretions may help predict the microorganisms involved in VAP.¹⁰ Differences in VAP rates among institutions can be due to the types of patients seen as well as procedural anomalies. Certainly, institutions whose populations are more at-risk (e.g. immunocompromised and/or critically ill patients) could potentially have higher VAP rates. Institutional factors could also affect the perceived VAP

The risk of developing pneumonia increases from 3 to 10 times when the patient is intubated and receiving mechanical ventilation.

rates. These factors include surveillance strategy, diagnostic techniques, and microbiology and laboratory procedures. A study looking at the comparison of 3 clinical definitions of VAP with autopsy findings showed poor correlation.¹¹ The etiology of VAP itself can also be variable, depending on the time of onset, duration of hospitalization, population studied and hospital setting.⁴

Hospital-acquired infections are associated with increases in ICU and hospital length-of-stay, costs, morbidity and mortality.¹² The risk of developing pneumonia increases from 3 to 10 times when the patient is intubated and receiving mechanical ventilation.⁴ In particular, the reported mortality attributable to VAP is in the neighborhood of 20% to 30% higher than with the underlying disease alone.⁴ The prognosis is worse when VAP is caused by gram-negative rather than gram-positive bacillae because the latter are susceptible to antibiotic therapy. Organisms that have developed antibiotic resistance are far more virulent. This is especially true with MRSA.

VAP risk factors can be associated with the patient (age, chronic lung disease and acute respiratory distress syndrome), the environment (lack of adherence to hand washing, improper disinfection of medical equipment, inappropriate patient position and in-

adequate isolation procedures) or the device (endotracheal tubes, humidification devices and ventilator circuits).

A normal respiratory tract has a variety of defense mechanisms such as anatomic barriers (glottis and larynx), cough reflexes, mucociliary lining and a phagocytic system consisting of macrophages and polymorphonuclear leukocytes (neutrophils). Patients with an endotracheal tube are at a disadvantage in that their upper airway defenses are bypassed, weakening their natural defenses.

Pneumonia results from microbial invasion of the normally sterile lower respiratory tract. An infection implies that the patient's defense mechanism has been activated and is directed at the pathogen. The lung, and in particular the lower respiratory tract, has a multifaceted system of defense. One of the most important components is the recruitment of neutrophils. The sequence of events associated with infection consists of initial interaction between bacteria and alveolar epithelial cells and macrophages. Neutrophil chemoattractants and cytokines are released as a result. Cytokines upregulate the expression of cell adhesion molecules on capillary endothelia and help mediate the migration of neutrophils into the alveolar spaces to battle the invaders. The neutrophils then induce a necrotic cellular death. Ultimately, a massive cellular death can lead to extensive lung injury.

Pathogenesis of VAP can include any of the following: aspiration of oropharyngeal organisms, inhalation of aerosolized bacteria, hematogenous spread and seeding from the gastrointestinal tract. Oropharyngeal and gastrointestinal organisms can seep around the endotracheal tube cuff. Inhalation of bacteria can occur from a contaminated ventilator circuit, humidifier or in-line medication nebulizers. Hematogenous spread involves transmission from a distal site infection.

Routes of pathogen transmission to the patient are related to either in-

dwelling devices, devices associated with mechanical ventilation or personnel. Indwelling devices include items such as catheters (IV, urinary, arterial lines), feeding tubes and endotracheal tubes. Bacteria can adhere to these artificial surfaces resulting in the formation of biofilms. This occurs because a conditioning layer of host proteins gets deposited on the surface of the catheter when it is inserted. Bacteria become attached by receptor-ligand binding. The bacteria then produce an exopolymer (biofilm) which protects them from the host immune system. Bacteria encased in a biofilm are more resistant to antimicrobial therapy. Once the biofilm is broken, the encased bacteria will flood into the urinary system, bloodstream or lungs.

Devices related to transmission with regards to mechanical ventilation include endotracheal tubes, ventilator circuits, humidifiers, medication nebulizers and suction catheters. The mere presence of an endotracheal tube provides the accessible route for pathogens to enter the lower respiratory tract. As with catheters, biofilms can form in and around endotracheal tubes. A study using an electron microscope showed that in 25 endotracheal tubes, 96% had partial bacterial colonization and 84% were completely coated with bacteria.¹³ Microaspiration of secretions from the upper respiratory tract is facilitated by leakage around the endotracheal tube cuff, impaired swallowing and oral defense mechanisms, and supine positioning.⁴

Upper airway, oral secretions and gastric juices contain many of the pathogens responsible for producing VAP. The mean prevalence of oral colonization with VAP-associated pathogens was 63% with the most common of these being *P. aeruginosa* and *Enterobacter* species along with gram-positive cocci such as *S. aureus*.¹⁴

Contamination of ventilator circuits, humidifiers, nebulizers and suction catheters can all lead to transmission

Upper airway, oral secretions and gastric juices contain many of the pathogens responsible for producing VAP.

of pathogens to the lower respiratory tract, either directly or in the form of an aerosolized bacterial cloud. The use of a contaminated suction catheter will introduce pathogens directly, as will inadvertent spilling of circuit condensate into the tracheobronchial tree. (This can happen with simple procedures such as turning or repositioning the patient.)

Personnel-related issues include not employing universal precautions (hand washing gloves, gowns) and disinfectant procedures. Why is this important? The terms colonization and infection have been used to describe bacterial populations within a host. Colonization refers to the presence of a bacterial pathogen with no host response. That is, the bacteria are present but the patient has not mounted an immune response. A patient can be colonized but not infected. This is more commonly seen outside of the ICU environment where the degree of illness is less severe. Healthcare workers may even be colonized with some of these pathogens but, in the presence of a healthy immune system, infection is kept in check. However, colonized individuals represent a threat to compromised patients so efforts must be made to prevent transmission. Lack of education in regards to pathogen transmission and/or lack of adherence to protocols (staffing or supervision issues) have the potential to create spikes in ICU infection rates.

Multiple factors can contribute to the spread of infection in the ICU. Proper identification of these factors and the adoption of effective safeguards need

to be in place to effectively barricade pathogen transmission. Effective hand washing is probably the most important factor in terms of infection spread.¹⁵ The use of soap or antimicrobial solutions can drastically reduce pathogenic spread from healthcare worker to patients. Gowns are generally not used when caring for every patient. However, they should be employed as part of a universal precaution initiative for patients infected with antibiotic resistant pathogens. Education and surveillance systems help inform clinicians on how to operate safely in the ICU environment and identify trends in either success or failure of protocols. Specific prevention of VAP is also multifaceted and includes prudent usage of antimicrobials (to reduce the possibility of resistance), keeping respiratory equipment and accessories properly cleaned, and scrupulous hand washing.

Many institutions employ what is termed the “VAP bundle” to help reduce the incidence of VAP. Components of the bundle include: 1) Avoidance of endotracheal intubation, if possible; and minimizing the duration of mechanical ventilation, 2) The use of orotracheal and orogastric tubes to prevent hospital-acquired sinusitis, 3) Avoidance of heavy sedation and neuromuscular blockade, 4) Endotracheal tube cuff pressure > 20 cm H₂O, 5) Prevention of ventilator circuit condensate in the lower respiratory tract, 6) Head of the bed raised 30-45 degrees, 7) Oral care and, 8) Hand hygiene.

NIV is associated with a decreased duration of mechanical ventilation and ICU stay.¹⁶ Daily assessments for spontaneous breathing and extubation will help reduce the amount of time with an endotracheal tube in place and length of time on a mechanical ventilator. Avoidance of heavy sedation and neuromuscular blockade will help decrease the time on the ventilator. Inline suction units prevent breaks in the circuit which help reduce the incidence of bacterial introduction as well as alveolar

de-recruitment. Keeping the same in-line suction catheter in place will result in secretion pooling; but these secretions would consist only of the patient's own bacterial flora. Using a different catheter for each suction encounter will heighten the chances of contamination from the surrounding environment. It has also been recently suggested that saline instillation before tracheal instillation may reduce the incidence of VAP though the induction of a cough and possible washout of the biofilm layer.¹⁷ Keeping the endotracheal tube cuff inflated to a pressure >20 cm H₂O will slow the incidence of microaspiration of oral pathogens from around the cuff into the lower respiratory tract. Endotracheal tubes with subglottic suction have the potential to minimize pooling of secretions on the cuff. However, difficulties with keeping the suction lumen clear in the presence of thick, tenacious secretions and prolapse of the tracheal mucosa into the subglottic suction port



Figure 1. OSMO (Teleflex Medical)

Table 1. Causative organisms of ventilator-associated pneumonia

EARLY ONSET	LATE ONSET
<i>Staphylococcus aureus</i> (Meth sensitive)	<i>Pseudomonas aeruginosa</i>
<i>Streptococcus pneumoniae</i>	MRSA
<i>Hemophilus influenzae</i>	<i>Acinetobacter</i> species
<i>Proteus</i> species	<i>Enterobacter</i> species
<i>Serratia</i> species	
<i>Klebsiella pneumoniae</i>	
<i>Escherichia coli</i>	

sometimes hamper their effectiveness.¹⁸ Also, these tubes tend to have a larger outer diameter for the same size inner lumen, so potential hazards exist with the intubation procedure itself. Damage to the tracheal wall and resultant mucociliary transport compromise can add to the risk of VAP.

Modification to endotracheal tubes themselves hold some promise in reducing the rate of VAP. A newly developed cuff material appears to have potential to also reduce microaspiration. The Kimberly-Clark Microcuff Adult Endotracheal Tube incorporates this type of cuff. It consists of microthin polyurethane which may create a better seal by minimizing channel openings within folds formed when the cuff is inflated. Further research needs to be done with this device. Another potential option is the use of endotracheal tubes lined with silver nitrate.¹⁹ The silver nitrate coating interferes with the creation of the bacterial biofilm. This may result in the delay of onset and/or a decrease in the severity of VAP. An example of this type of endotracheal tube is the Agento IC developed by C.R. Bard, Inc. However, these types of endotracheal tubes tend to be more expensive, so cost assessment must be taken into account. As with the new cuff material, more research is needed to determine if these theoretical improvements translate into VAP rate reductions.

Protocols regarding changing of the ventilator circuit have evolved significantly over the years. It is no longer recommended that ventilator circuits be changed on a routine basis, but rather they should be changed when visibly

soiled to prevent the spread of contaminants pooled in the circuit.²⁰ Also, in an effort to reduce the number of ventilator circuit disconnects, the SHEA guidelines recommend that the circuit remain closed during condensate removal to reduce the possibility of cross contamination.² As was the case with the endotracheal tubes, new technology is being developed to reduce the number of ventilator circuit disconnects due to excessive condensate. An example is the OSMO™ from Teleflex Medical. (Figure 1). The theory behind the OSMO is that it incorporates a unique media that promotes the condensation of water vapor from exhaled gas. OSMO is designed to reduce circuit condensation, thereby reducing clinician intervention. Another innovation is the use of in-line nebulizers with a 1-way tee valve connector that allows for draining of the medication cup without breaking the circuit. Draining will prevent pooling and possible contamination that would get delivered along with the medication in aerosol form. The same can be said of the active humidifier. Although it is not recommended that humidifiers be changed on a regular basis, they should get changed if there are any signs of visible contamination.

Heat Moisture Exchangers (HMEs) have been suggested as preventative measures to stop the spread of VAP. Evidence suggests that the use of HMEs and active heated humidifiers are comparable in terms of VAP.⁴ However, HMEs have the potential of getting clogged with secretions and increasing the work of breathing. In addition, the need to remove the HME to administer



Figure 2. Gibeck Humid-Flo HME device (Teleflex Medical)

regular aerosol treatments increases the risk for bacterial invasion and alveolar de-recruitment. New advances are being made with HMEs as well. One such device is the Gibeck® Humid-Flo® HME from Teleflex Medical (figure 2). It is designed to remain in the circuit even in the event of aerosolized medication delivery. It employs a rotating collar that can open a separate channel for medication delivery. It has 2 channels, one for passive humidification and a second for medication delivery. The collar merely gets rotated to select the intended channel and a circuit disconnect is not needed. By reducing the number of circuit disconnects, the potential exists to reduce VAP rates.

There are a variety of pathogens capable of producing infections in compromised hosts in the ICU environment. Some of them, in particular MRSA and VRE, are readily transmitted by contact and increasing with alarming frequency. Many of these infections are preventable. Attention must be paid to routes of transmission and programs developed to help block these routes in order to reduce the frequency of ICU infections and the associated costs and morbidity.

References

1. Yokoe DS, Mermel LA, Anderson DJ, et al. A compendium of strategies to prevent healthcare-associated infections in acute care hospitals. *Infect Control Hosp Epidemiol* 2008;29:S12-S21.

There are a variety of pathogens capable of producing infections in compromised hosts in the ICU environment. Some of them, in particular MRSA and VRE, are readily transmitted by contact and increasing with alarming frequency.

2. Coffin SE, Klompas M, Classen D. Strategies to prevent ventilator-associated pneumonia in acute care hospitals. *Infect Control Hosp Epidemiol* 2008;29:S31-S40.
3. Klevins RM, Edwards JR, Richards JL. Estimating Health Care-Associated Infections and Deaths in U.S. Hospitals, 2002. *Public Health Reports* March-April 2007;122:160-166.
4. Chastre J, Fagon JY. Ventilator-associated Pneumonia. *Am J Respir Crit Care Med* 2002;165:867-903.
5. Boyce JM, Jackson MM, Pugliese G, et al. Methicillin-resistant *Staphylococcus aureus* (MRSA): a briefing for acute care hospitals and nursing facilities. The AHA Technical Panel on Infections Within Hospitals. *Infect Control Hosp Epidemiol* 1994;15:105-115.
6. NNIS System. National Nosocomial Infections Surveillance (NNIS) System Report, data summary from January 1992 through June 2003, issued August 2003. *Am J Infect Control*. 2003;31(8):481-98.
7. Jones, R. N. Resistance Patterns Among Nosocomial Pathogens. *Chest* 2001;119:397S-404S.
8. Fridkin SK, Edwards JR, Courval JM, et al. The effect of vancomycin and third-generation cephalosporins on prevalence of vancomycin-resistant enterococci in 126 U.S. adult intensive care units. *Ann Intern Med* 2001;135:175-183.
9. Swanson JM, Wood GC, Croce MA. Utility of Trauma 2008; 65:1271-1277.
10. Pirrachio R, Mateo J, Raskine L, et al. Can bacteriological upper airway samples obtained at intensive care unit admission guide empiric antibiotherapy for ventilator-associated pneumonia? *Crit Care Med* 2009;37:2559-2563.

Med 2009;37:2559-2563.

11. Tejerina E, Esteban A, Fernandez-Segoviano, et al. Accuracy of clinical definitions of ventilator-associated pneumonia: Comparison with autopsy findings. *J Crit Care* 2009; Article in press. Published online 10 July 2009. <http://www.jccjournal.org/article/PIIS088394410900121X/abstract>.
12. Safdar N, Dezfulian C, Collard HR, et al. Clinical and Economic Consequences of Ventilator-Associated Pneumonia: A Systematic Review. *Crit Care Med* 2005;33:2184-2193.
13. Sottile FD, Marrie TJ, Prough DS, et al. Nosocomial pulmonary infection: possible etiologic significance of bacterial adhesion to endotracheal tubes. *Crit Care Med* 1986;14:265-270.
14. Brennan MT, Bahrani-Mougeot F, Fox PC, et al. The role of oral microbial colonization in ventilator-associated pneumonia. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 2004;98:665-672.
15. Pittet D, Boyce JM. Revolutionising hand hygiene in health-care settings: Guidelines revisited. *Lancet Infect Dis* 2003;3:269-270.
16. Antonelli M, Conti G, Rocco M, et al. A comparison of noninvasive positive-pressure ventilation and conventional mechanical ventilation in patients with acute respiratory failure. *N Engl J Med* 1998; 339(7):429-435.
17. Caruso P, Denari S, Ruiz SA, et al. Saline instillation before tracheal suctioning decreases the incidence of ventilator-associated pneumonia. *Crit Care Med* 2009;37:32-38.
18. Dragoumanis CK, Vretzakis GI, Papaioannou VE, et al. Investigating the failure to aspirate subglottic secretions with the evac endotracheal tube. *Anesth Analg* 2007;105:1083-1085.
19. Olson ME, Harmon BG, Kollef MH. Silver-coated endotracheal tubes associated with reduced bacterial burden in the lungs of mechanically ventilated dogs. *Chest* 2002;121:863-870.
20. Hess DR. AARC Evidence-based clinical practice guidelines: Care of the ventilator circuit and its relation to ventilator-associated pneumonia. *Respir Care* 2003;48:869-879.

John Davies, MA, RRT, FAARC is a registered respiratory therapist and Clinical Research Coordinator at the Duke University Medical Center, Durham, North Carolina. John's research interests include ventilation techniques, the distribution of nebulizer medication in lung transplant patients, body weight and tidal volume calculation, and other aspects of respiratory care. He has published a number of papers in the literature and has presented at several medical meetings. He also lectures on mechanical ventilation at the Duke University Medical Center. John lives in Cary, North Carolina.

Panel Discussion: Ventilator-Associated Pneumonia and the Role of the Respiratory Therapist

Moderator:

Harvey E. Marshall, MD

Assistant Professor of Medicine

Division of Pulmonary, Allergy,
and Critical Care Medicine

Duke University

Panelists:

Stephen Kantrow MD

Ruben Restrepo MD, RRT, FAARC

Kathleen Arias MS, CIC

The diagnosis of VAP requires a thorough understanding of clinical signs and the microbial pathogens involved. Prevention and treatment also requires education, care in handling medical devices, and appropriate use of antibiotics. In this panel discussion, we ask 3 experts to discuss issues related to the differential diagnosis of VAP, how one should choose antibiotics, what to do if respiratory tract cultures are negative, endotracheal tube modifications that impede infections, use of the VAP bundle, and other methods for decreasing the likelihood of VAP.

What clinical criteria should be used to define ventilator-associated pneumonia (VAP) and what is the difference between VAP and tracheobronchitis?

Kantrow: VAP is suspected in patients with a new infiltrate on chest radiograph more than 2 days after intubation when at least one clinical feature of infection (fever, leukocytosis or purulent sputum) is present. These criteria are sensitive for VAP, however, confirming the presence

of pneumonia with confidence is more difficult. Cultures of the lower respiratory tract that yield pathogenic organisms in large quantity support the diagnosis of VAP. Negative lower respiratory cultures in the absence of new antibiotics in the past 3 days make this diagnosis less likely. Cultures may be obtained by sampling distal airway purulence or air-spaces or can be obtained from a more proximal airway site via endotracheal aspiration.¹ Infection may also occur in the trachea and bronchi without obvious involvement of the airspaces. In the setting of fever, leukocytosis and purulence in the airway without radiographic evidence of pneumonia, the diagnosis of nosocomial tracheobronchitis should be considered. Cultures of endotracheal aspirates are typically positive for bacterial pathogens in patients with this infection, and antibiotic therapy is associated with improved clinical outcomes.²

Restrepo: VAP is a nosocomial pneumonia that develops more than 48 hours after endotracheal intubation.³ The clinical criteria used to support or suspect the diagnosis of VAP includes new or progressive pulmonary infiltrate with fever, leukocytosis, and purulent sputum. Additionally, the patient may appear tachypneic, hypoxemic, or in need for more ventilatory support. Clinical suspicion of VAP requires radiologic and microbiological confirmation. Ventilator-associated tracheobronchitis (VAT) is a relatively new term to define an intermediate process between tracheal colonization and VAP.^{2,4-6} Discriminating between VAT and VAP can be challenging. VAT should be suspect-

ed in intubated patients with clinical signs of lower respiratory tract infection (fever, leukocytosis, and purulent sputum), with a Gram stain demonstrating the presence of microorganisms and polymorphonuclear leukocytes in the absence of a new or progressive infiltrate on the chest radiograph. Since VAT may appear to be an important risk factor for VAP, early detection of respiratory colonization by monitoring tracheal aspirates is critical in the initiation of targeted antibiotic therapy. The most common pathogens for VAT and VAP include *Pseudomonas aeruginosa*, followed by *Acinetobacter baumannii*, *Enterobacter* spp, and methicillin-resistant *Staphylococcus aureus* (MRSA).⁷⁻¹⁰

Arias: The clinical diagnosis of VAP is difficult and is complicated by the lack of a standard diagnostic protocol for identifying pneumonia. VAP is generally defined as nosocomial, or health-care associated, pneumonia (i.e. was not clearly present or incubating at the time of admission) in a patient on mechanical ventilation for >48 hours.³ Criteria used to identify VAP have been noted by Drs. Restrepo and Kantrow. However, because these findings are nonspecific they cannot be used alone to definitively establish the presence of VAP. Additional clinical signs used to identify pneumonia include dyspnea, rales, and worsening gas exchange. It should be noted that criteria used for surveillance (i.e. to calculate VAP incidence rates) often differ from criteria used to clinically diagnose and treat a patient for VAP. Many hospitals use the definitions of the CDC's National Healthcare Safety Network (NHSN) for surveillance purposes, however, these criteria were not developed for use as a clinical definition.¹¹ For instance, in the NHSN surveillance definition for VAP, there is no minimum period of time that the ventilator must be in place in order for pneumonia to be considered ventilator-associated. In other words, in the NHSN surveillance system, pneumonia is reported as VAP if

the patient was intubated and ventilated at the time of or within 48 hours before the onset of pneumonia. The ATS/IDSA guidelines for the management of adults with pneumonia note that tracheobronchitis should be considered when fever, leukocytosis, purulent sputum, and a positive culture of a sputum or tracheal aspirate are present without a new lung infiltrate. While many of the signs of tracheobronchitis mimic those of VAP, a major difference is the absence of an infiltrate in tracheobronchitis.

In a patient with VAP, what factors should guide the choice of antibiotics?

Kantrow: Empiric therapy is recommended for patients suspected to have VAP. Timely collection of respiratory sample for microbiology is important, and antibiotic therapy should not be delayed until culture results are available.³ Pathogens to consider when choosing antibiotics for VAP include *Pseudomonas aeruginosa*, *Klebsiella pneumoniae* and *Acinetobacter* species. Antibiotics likely to be effective against resistant gram negative organisms should be administered initially, with consideration given to specific organisms endemic in the intensive care unit or previous patient location (e.g. ESBL-expressing *Klebsiella* or pan-resistant *Acinetobacter* species). Initial antibiotic therapy should also provide appropriate coverage for resistant gram positive organisms, especially methicillin-resistant *Staphylococcus aureus* (MRSA). Early onset VAP is more likely than later onset VAP to be caused by drug susceptible bacteria. However, additional risk factors for resistant pathogens should be sought to determine appropriate antibiotic therapy. Recent antibiotic therapy or hospitalization within the past 90 days, immune suppression, nursing home residence, chronic dialysis, and wound care all increase the likelihood of harboring multidrug resistant pathogens at the time of admission. Local patterns of nosocomial infection and antimicrobial resistance are often unique and should

Early onset VAP is more likely than later onset VAP to be caused by drug susceptible bacteria.

— Kantrow —

guide the antibiotic strategy.

Restrepo: The presence of risk factors for multidrug resistance (MDR) pathogens such as late onset disease, positive cultures, presence of an underlying disease, and antibiotic therapy in the preceding 3 months typically guide the selection of antibiotic therapy. However, knowledge of resident flora, local susceptibility and resistance rate trends in the ICU have shown to be invaluable in creating institution-specific guidelines for appropriate empiric antibiotic therapy.^{12,13} Empiric broad-spectrum, multidrug therapy is typically recommended until pretherapy results are available. If antibiotic therapy was administered prior to the diagnosis of VAP, coverage for MRSA, *Acinetobacter spp*, and *Legionella* should be considered. Once the pathogen and its susceptibility pattern are identified, the antibiotic regimen should be narrowed.

Arias: Antimicrobial selection should be guided by the likely microbial etiology of the pneumonia, the common respiratory pathogens seen in the hospital and the community, and the antibiotic susceptibility and resistance patterns in those pathogens.

If respiratory tract cultures are negative, should empiric antibiotic therapy be discontinued?

Kantrow: Respiratory tract cultures can be obtained distally at the site of lung infection with bronchoscopic or nonbronchoscopic techniques, or from

central airways via endotracheal aspiration. Samples collected by experienced operators at the site of suspected infection can establish a diagnosis of VAP and identify the etiologic agent, guiding subsequent therapy. Culture negative samples collected from the distal airways decrease the likelihood of VAP, and may allow discontinuation of antibiotic therapy, particularly when the clinical suspicion of pneumonia is not high. Endotracheal aspirates from proximal airway secretions do not distinguish between bacterial colonization, infection in the airways and pneumonia. However, when the endotracheal aspirate sample is adequate and antibiotics have not been started or changed in the past 3 days, negative cultures decrease the likelihood of VAP. If antibiotic therapy is not discontinued based upon culture results, the clinician may still use culture data to make important decisions about de-escalation. For patients with a good response to antibiotics, therapy can be narrowed as culture results become available. When there is no evidence of resistant gram negative rods such as *Pseudomonas* and *Acinetobacter* in the endotracheal aspirate, antibiotic therapy can be discontinued on day 7 or 8.¹⁴

Restrepo: Serial assessment of clinical status and culture results 48-72 hours after initiation of antibiotic therapy are the best parameters for de-escalating or discontinuing therapy. If the patient shows clinical improvement and cultures are negative, antibiotic therapy could be safely discontinued after 72 hours.^{3,15,16}

Arias: Discontinuation of empiric therapy should be based on improvement or deterioration of the patient's clinical status.

Are there modifications of the endotracheal tube that can diminish the incidence of VAP?

Kantrow: After endotracheal intubation, colonization of the endotracheal

tube and lower airway mucosa occurs as contaminated secretions from the sinuses, oropharynx and/or the stomach enter the subglottic space. Two modifications of the endotracheal tube have been found in randomized controlled trials to decrease the incidence of VAP. First, impregnation of the endotracheal tube with an antibacterial substance (silver or chlorhexidine) can decrease the biofilm formed by bacteria and subsequent inoculation of the lung.¹⁷ Second, a modified endotracheal tube with a port for continuous aspiration of subglottic secretions may limit entry of bacteria laden secretions into the lung.¹⁸ In addition, maintenance of cuff pressure greater than 20 cm H₂O in a conventional endotracheal tube can oppose inoculation of the lung from this reservoir and decrease VAP.

Restrepo: Formation of antibiotic-resistant biofilms in the inner surface of the endotracheal tube (ETT) within hours of intubation and aspiration of secretions into the lower airway play a critical role in the incidence of VAP.¹⁹ A silver-coated ETT has been designed to decrease the incidence of VAP by preventing bacterial colonization, biofilm formation, and its subsequent embolization to the lower airways.^{20,21,22} A large clinical trial recently found that patients receiving a silver-coated ETT had a statistically significant reduction in the incidence of VAP.¹⁷ Another specially designed ETT that provides continuous aspiration of subglottic secretions (CASS) decreases the risk of VAP especially in patients expected to require ventilatory support for more than 72 hours.^{18,23} Although the silver-coated ETT is radically more expensive than the standard ETT, its use has been associated with important hospital savings.²⁴ These 2 specially designed tubes, however, cost more than the standard ETT.

Is the implementation of a VAP bundle a valuable strategy? Are there any pitfalls

Formation of antibiotic-resistant biofilms in the inner surface of the endotracheal tube (ETT) within hours of intubation and aspiration of secretions into the lower airway play a critical role in the incidence of VAP.¹⁹

— Restrepo —

or weaknesses of such an approach?

Kantrow: A VAP bundle is a systems-based approach to implementing clinical strategies with established benefit to decrease the incidence of VAP. Implementation of a VAP bundle is likely to increase adherence to best available practices. Challenges include the fact that there is frequently not a consensus about the strength of evidence and cost effectiveness of interventions placed within the bundle. Future studies may demonstrate that interventions currently held to be beneficial are not actually helpful, even while they are widely implemented. For this reason, it is important to adapt the systems based approach over time with new data. A pitfall to assessing the impact of bundle implementation is that the targeted outcome measure may be susceptible to bias in reporting. That may be the case for VAP, where confirmatory testing is not consistently performed.

Restrepo: The VAP bundle is an evidence-based strategy that can be credited for a dramatic reduction of VAP rates in the ICU over the last few

years.²⁵ However, lack of a clear definition of the components of the bundle and adequate staffing may hamper the implementation of a protocol to reduce VAP. The Institute for Healthcare Improvement (IHI) Ventilator bundle contained 4 evidence-based practices to improve outcomes of patients requiring mechanical ventilation, semi-recumbent position, daily 'sedation vacation' and daily extubation readiness assessments, peptic ulcer disease prophylaxis, and deep venous thrombosis. Except for daily awakening and implementation of spontaneous breathing trials, the other strategies do not allow early liberation from mechanical ventilation. However, implementation of the IHI bundle was associated with a reduction on VAP incidence and many institutions adopted it as a VAP bundle. Other common listed patient care practices to reduce VAP include CASS, and oral hygiene; however, they have not been routinely identified as part of the Ventilator bundle.

Although head bed elevation appears relatively simple to implement, multiple studies have found low compliance.^{26,27} The CASS may be applied to all intubated patients. However, the ETT needs to be available when the patient is first intubated so reintubation is avoided, and it also requires additional suction regulators to be setup. While oral hygiene improves patient comfort and has no side effects, it is labor intensive and its compliance greatly depends on adequate staffing of the ICU. Reduction in ventilator circuit changes is associated with reduction of overall costs but no strong correlation has been found between circuit replacement frequency and VAP rates. Earlier weaning reduces risks associated with mechanical ventilation including VAP. Its benefit in reducing VAP incidence is then limited to those patients who can be weaned shortly after being intubated. It is also labor intensive for it requires close monitoring of a patient with lighter sedation and possibly more prompt to experiencing anxiety

and self extubation. While the evidence behind the use of histamine type-2 antagonists or sucralfate as well as the use of DVT prophylaxis to reduce VAP incidence are still controversial, they should be retained as components of the Ventilator Bundle.²⁸

Arias: Many hospitals that have implemented VAP bundles have been able to demonstrate significant reductions in VAP rates.^{28,29} Many hospitals have incorporated into their practice bundles additional evidence based strategies aimed at preventing VAP. These include: oral hygiene, removal of subglottic secretions, hand hygiene, and education of personnel who care for patients on ventilators regarding prevention of VAP.^{28,30,31} Several obstacles limit the ability to measure the potential efficacy of VAP bundles. These obstacles include: difficulty in obtaining an accurate diagnosis of VAP, the variety of definitions used to identify VAP, the inability to precisely determine the impact of each bundle component in preventing VAP, the use of different VAP bundle components in published reports, and the implementation of a variety of additional infection prevention strategies at the same time the VAP bundle is implemented.³² Since most studies of the efficacy of VAP bundles have been conducted in critical care settings, the effectiveness of implementing VAP bundles in other settings has not been determined.

What other measures, protocols, practices can the respiratory therapist employ that have been determined to/or may decrease the incidence of VAP?

Kantrow: The respiratory therapist plays a key role in several aspects of mechanical ventilation that can affect the risk of VAP. Implementation of non-invasive ventilator support in carefully selected patients can avoid endotracheal intubation and subsequent VAP. Unnecessarily prolonged mechanical ventilation carries a daily risk of infection

The respiratory therapist plays a critical role in preventing VAP by using aseptic technique to avoid contaminating medications, fluids and equipment, maintaining endotracheal tube cuff pressure above 20 cm of water, promoting early extubation, avoidance of reintubation, and use of noninvasive ventilation.

— Arias —

and can be avoided by adhering to a protocol of daily sedation interruption, respiratory assessment and spontaneous breathing trials where indicated.³³ Reintubation carries an increased risk of VAP as well, and can be avoided by carefully determining readiness for extubation and by avoiding accidental extubation.

Restrepo: Addition to the Ventilator Bundle of other evidence-based patient care practices such as oral decontamination with chlorhexidine antiseptic, and subglottic secretion drainage in patients expected to be mechanically ventilated for more than 72 hours is a very effective VAP prevention strategy.^{27,34} Other recommendations for VAP prevention include the use of the orotracheal route of intubation, the change of heat and moisture exchangers every 5-7 days

and as clinically indicated, and the use of closed endotracheal suction systems (in-line suction).³⁵ Strong involvement of respiratory therapists in educational programs has been associated with significant reductions of VAP rates.^{36,37,38}

Arias: In addition to the practices noted above, the respiratory therapist plays a critical role in preventing VAP by using aseptic technique to avoid contaminating medications, fluids and equipment, maintaining endotracheal tube cuff pressure above 20 cm of water, promoting early extubation, avoidance of reintubation, and use of noninvasive ventilation, appropriately cleaning, disinfecting and sterilizing respiratory devices, and using barrier precautions such as gloves.^{3,30,31}

References

- 1 The Canadian Critical Care Trials Group. A randomized trial of diagnostic techniques for ventilator-associated pneumonia. *NEJM* 2006;355:2619-30.
- 2 Nseir S, Ader F, Marquette CH. Nosocomial tracheobronchitis. *Curr Opin Infect Dis* 2009;22(2):148-153.
- 3 American Thoracic Society; Infectious Diseases Society of America. Guidelines for the management of adults with hospital-acquired, ventilator-associated, and healthcare-associated pneumonia. *Am J Respir Crit Care Med* 2005;171:388.
- 4 Craven DE, Chroneou A, Zias N, Hjalmarsen KI. Ventilator-associated tracheobronchitis: the impact of targeted antibiotic therapy on patient outcomes. *Chest* 2009;135(2):521-528.
- 5 Nseir S, Favory R, Jozefowicz E, et al. Antimicrobial treatment for ventilator-associated tracheobronchitis: a randomized controlled multicenter study. *Crit Care* 2008;12:R62.
- 6 Palmer LB, Smaldone GC, Chen JJ, et al. Aerosolized antibiotics and ventilator-associated tracheobronchitis in the intensive care unit. *Crit Care Med* 2008;36:2008-2013.
- 7 Torres A, Valencia M. Does ventilator-associated tracheobronchitis need antibiotic treatment? *Crit Care* 2005; 9:255-256.
- 8 Nseir S, Di Pompeo C, Pronnier P, et al. Nosocomial tracheobronchitis in mechanically ventilated patients: incidence, aetiology and outcome. *Eur Respir J* 2002;20:1483-1489.
- 9 Kampf G, Wischnewski N, Schulgen G, Schumacher M, Daschner F. Prevalence and risk factors for nosocomial lower respiratory tract infections in German hospitals. *J Clin Epidemiol* 1998;51:495-502.
- 10 Koulenti D, Lisboa T, Brun-Buisson C, et al. Spectrum of practice in the diagnosis of nosocomial pneumonia in patients requiring mechanical ventilation in European intensive care units. *Crit Care Med* 2009;37(8):2360-2368.
- 11 CDC NHSN Manual: Patient Safety Component Protocol. Ventilator associated pneumonia (VAP)

- event. <http://www.cdc.gov/nhsn>
- 12 Beardsley JR, Williamson JC, Johnson JW, Ohl CA, Karchmer TB, Bowton DL. Using local microbiologic data to develop institution-specific guidelines for the treatment of hospital-acquired pneumonia. *Chest* 2006;130(3):787-793.
 - 13 Kuti JL, Shore E, Palter M, Nicolau DP. Tackling empirical antibiotic therapy for ventilator-associated pneumonia in your ICU: guidance for implementing the guidelines. *Semin Respir Crit Care Med* 2009;30(1):102-115.
 - 14 Chastre J, Wolff M, Fagon JY, et al. Comparison of 8 vs 15 days of antibiotic therapy for ventilator-associated pneumonia in adults: a randomized trial. *JAMA* 2003;290(19):2588-98.
 - 15 Kollef MH, Kollef KE. Antibiotic utilization and outcomes for patients with clinically suspected ventilator-associated pneumonia and negative quantitative BAL culture results. *Chest* 2005;128:2706-2713.
 - 16 Kollef MH, Morrow LE, Niederman MS, et al. Clinical characteristics and treatment patterns among patients with ventilator-associated pneumonia. *Chest* 2006;129(5):1210-1218.
 - 17 Kollef MH, Afessa B, Anzueto A, et al. Silver-coated endotracheal tubes and incidence of ventilator-associated pneumonia: the NASCENT randomized trial. *JAMA* 2008;300(7):805-813.
 - 18 Bouza E, Perez MJ, Munoz P, Rincon C, Barrio JM, Hortal J. Continuous aspiration of subglottic secretions in the prevention of ventilator-associated pneumonia in the postoperative period of major heart surgery. *Chest* 2008;134(5):938-946.
 - 19 Shah C, Kollef MH. Endotracheal tube intraluminal volume loss among mechanically ventilated patients. *Crit Care Med* 2004;32(1):120-125.
 - 20 Olson ME, Harmon BG, Kollef MH. Silver-coated endotracheal tubes associated with reduced bacterial burden in the lungs of mechanically ventilated dogs. *Chest* 2002;121(3):863-870.
 - 21 Berra L, De Marchi L, Yu ZX, Laquerriere P, Baccarelli A, Kolobow. Endotracheal tubes coated with antiseptics decrease bacterial colonization of the ventilator circuits, lungs, and endotracheal tube. *Anesthesiology* 2004;100(6):1446-1456.
 - 22 Rello J, Kollef M, Diaz E, et al. Reduced burden of bacterial airway colonization with a novel silver-coated endotracheal tube in a randomized multiple-center feasibility study. *Crit Care Med* 2006;34(11):2766-2772.
 - 23 Dezfulian C, Shojania K, Collard HR, Kim HM, Matthay MA, Saint S. Subglottic secretion drainage for preventing ventilator-associated pneumonia: a meta-analysis. *Am J Med* 2005;118(1):11-18.
 - 24 Shorr AF, Zilberberg MD, Kollef M. Cost-effectiveness analysis of a silver-coated endotracheal tube to reduce the incidence of ventilator-associated pneumonia. *Infect Control Hosp Epidemiol* 2009;30(8):759-63.
 - 25 Institute for Healthcare Improvement. <http://www.ihl.org>. [Accessed 14 August 2009].
 - 26 van Nieuwenhoven CA, Vandenbroucke-Grauls C, van Tiel FH, et al. Feasibility and effects of the semi-recumbent position to prevent ventilator-associated pneumonia: a randomized study. *Crit Care Med* 2006;34:396-402.
 - 27 Helman DL Jr, Sherner JH 3rd, Fitzpatrick TM, Callender ME, Shorr AF. Effect of standardized orders and provider education on head-of-bed positioning in mechanically ventilated patients. *Crit Care Med* 2003;31:2285-2290.
 - 28 Wip C, Napolitano L. Bundles to prevent VAP: How valuable are they? *Curr Opin Infect Dis* 2009;22(2):159-166.
 - 29 5 Million Lives Campaign. Getting Started Kit: Prevent Ventilator-Associated Pneumonia How-to Guide. Cambridge, MA: Institute for Healthcare Improvement; 2008. (www.ihl.org) [accessed August 19, 2009]
 - 30 Tablan OC, Anderson LJ, Besser R, Bridges C, Hajjeh R. Guidelines for preventing health-care-associated pneumonia, 2003: recommendations of CDC and the Healthcare Infection Control Practices Advisory Committee. *MMWR Recomm Rep* 2004; 53:1-36.
 - 31 Yokoe DS, Mermel LA, Classen, D, et al. A compendium of strategies to prevent healthcare-associated infections in acute care hospitals. *Infect Control Hosp Epidemiol* 2008;29:S12-S21.
 - 32 Blamoun J, Alfakir M, Rella M, et al. Efficacy of an expanded ventilator bundle for the reduction of ventilator associated pneumonia in the medical intensive care unit. *Am J Infect Control* 2009;37:172-175.
 - 33 Ely EW, Bennett PA, Bowton DL, Murphy SM, Florence AM, Haponik EF. Large scale implementation of a respiratory therapist-driven protocol for ventilator weaning. *Am J Respir Crit Care Med* 1999;159:439-46.
 - 34 Chan EY, Ruest A, Meade MO, Cook DJ. Oral decontamination for prevention of pneumonia in mechanically ventilated adults: systematic review and meta-analysis. *BMJ* 2007;334:889.
 - 35 Muscedere J, Dodek P, Keenan S, Fowler R, Cook D, Heyland D. Comprehensive evidence-based clinical practice guidelines for ventilator-associated pneumonia: prevention. *J Crit Care* 2008;23:126-137.
 - 36 Babcock HM, Zack JE, Garrison T, Trovillion E, Kollef MH, Fraser VJ. An educational intervention to reduce ventilator-associated pneumonia in an integrated health system: a comparison of effects. *Chest* 2004;125:2224-2231.
 - 37 Bloos F, Müller S, Harz A, et al. Effects of staff training on the care of mechanically ventilated patients: a prospective cohort study. *Br J Anaesth* 2009;103(2):232-237.
 - 38 Hawe CS, Ellis KS, Cairns CJ, Longmate A. Reduction of ventilator-associated pneumonia: active versus passive guideline implementation. *Intensive*

Stephen Phillips Kantrow, MD, is Associate Professor of Medicine, Louisiana State University Health Sciences, and Program Director, Pulmonary/Critical Care Fellowship, Louisiana State University Medical Center, New Orleans. Dr. Kantrow has authored or coauthored many papers and book chapters on topics related to pulmonary and critical care medicine and he conducts an active research program on the effects of alcohol and HIV infection on the host response. He lives in New Orleans.

Harvey E. Marshall, MD, is Assistant Professor of Medicine, Division of Pulmonary, Allergy, and Critical Care Medicine, Duke University Medical Center, Durham, North Carolina. He is also a member of the American Thoracic Society, the American College of Chest Physicians, and Alpha Omega Alpha. He is a reviewer for several major journals in the area of pulmonary and critical care medicine and conducts an active research program on the pathophysiology of lung injury and the pathogenesis and genetics of environmental asthma. Dr. Marshall has been coauthor of many articles and abstracts related to pulmonary medicine, and is a frequent invited speaker at medical meetings. He lives in Chapel Hill, North Carolina.

Kathleen Meehan Arias, MS, CIC has worked in the infection prevention and control field since 1980 and is currently the Director of Arias Infec-

tion Control Consulting, LLC. She has infection prevention and control experience in a variety of settings, including acute care hospitals, long-term care, rehabilitation care, ambulatory care and industry. Among other editorial projects, Ms. Arias is the editor and an author of the APIC/Joint Commission Resources Infection Prevention and Control Workbook and the APIC Tool Kit Assessing and Developing an Infection Control Program in the Acute Care Setting. She has taught epidemiology classes at Thomas Jefferson University and at the Medical College of Philadelphia and has served on the faculties of Penn State University and the Hahnemann University College of Allied Health Sciences. Ms. Arias is a frequent speaker at local, national, and international conferences. An active member of the Association for Professionals in Infection Control and Epidemiology (APIC), she served as the 2006 APIC President.

Ruben Restrepo, MD obtained his diploma as physician and surgeon from Colombia University. Dr. Restrepo is currently Associate Professor in the Department of Respiratory Care at The University of Texas Health Science Center at San Antonio in 2005. In recognition of his contribution to the field, he was inducted as a fellow of the American Association for Respiratory Care. He is a member of the editorial board for Respiratory Care and the Open Journal of Allergy and the chair of the Clinical Practice Guidelines Steering Committee for the AARC. He has been invited to speak at national and international symposiums to both medical and respiratory audiences. He has over 60 peer-reviewed publications between manuscripts, book chapters, and abstracts.

Clinical Foundations is a serial education program distributed free of charge to health professionals. *Clinical Foundations* is published by Saxe Healthcare Communications and is sponsored by Teleflex Medical. The goal of *Clinical Foundations: A Patient-Focused Education Program for Respiratory Care Professionals* is to present clinically- and evidence-based practices to assist the clinician in making an informed decision on what is best for his/her patient. The opinions expressed in *Clinical Foundations* are those of the authors only. Neither Saxe Healthcare Communications nor Teleflex Medical make any warranty or representations about the accuracy or reliability of those opinions or their applicability to a particular clinical situation. Review of these materials is not a substitute for a practitioner's independent research and medical opinion. Saxe Healthcare Communications and Teleflex Medical disclaim any responsibility or liability for such material. They shall not be liable for any direct, special, indirect, incidental, or consequential damages of any kind arising from the use of this publication or the materials contained therein.

Please direct your correspondence to:

Saxe Healthcare Communications
P.O. Box 1282
Burlington, VT 05402
info@saxecommunications.com
Fax: 802.872.7558
© Saxe Communications 2009

Questions

- What is biofilm?**
 - Lining of a stethoscope
 - Bacteria exopolymer
 - Film on anatomy
 - None of the above
- How much more likely are mechanically ventilated patients to get pneumonia?**
 - Twice as likely
 - The same
 - 20 times
 - 3-10 times
- VRE and MRSA are spread mainly by droplet nuclei?**
 - True
 - False
- What factors contribute to differences among various institutions in VAP rates?**
 - Surveillance strategy
 - Diagnostic techniques
 - Type of ventilator used
 - a + b
- How many deaths are associated with VAP each year in the United States?**
 - 800,000
 - 2000
 - 99,000
 - 210,000
- VAP does not occur in the first 5 days of mechanical ventilation?**
 - True
 - False
- What is the name of the organization that wrote the VAP practice guidelines?**
 - OMNI
 - PETCO
 - ASHE
 - SHEA
- What is not part of the "VAP bundle"?**
 - Orotracheal vs nasotracheal tubes
 - Head of bed at 30-45 degrees
 - Use of filters on the expiratory limb of the ventilator circuit
 - Hand hygiene
- NIV and invasive mechanical ventilation have similar VAP rates?**
 - True
 - False
- Which type of white blood cell causes cellular death?**
 - Mast cell
 - Neutrophil
 - Monocyte
 - Macrophage
- What practices can the respiratory therapist employ that have been determined to/or may decrease the incidence of VAP?**
 - Implementation of non-invasive ventilator support in carefully selected patients
 - Oral decontamination with chlorhexidine antiseptic
 - Subglottic secretion drainage
 - All of the above
- Impregnation of the endotracheal tube with an antibacterial substance can decrease the biofilm formed by bacteria**
 - True
 - False

This program has been approved for 2.0 contact hours of continuing education (CRCE) by the American Association for Respiratory Care (AARC). AARC is accredited as an approver of continuing education in respiratory care.

To earn credit, do the following:

- Read all the articles.
- Complete the entire post-test.
- Mark your answers clearly with an "X" in the box next to the correct answer. You can make copies.
- Complete the participant evaluation.
- Go to www.saxetesting.com to take the test online or mail or fax the post-test and evaluation forms to address below.
- To earn 2.0 CRCEs or CEs, you must achieve a score of 75% or more. If you do not pass the test you may take it over one more time.
- Your results will be sent within 4-6 weeks after forms are received by mail or fax.
- Answer forms must be postmarked by Dec. 31, 2009 (RTs) or Aug. 26, 2011 (Nurses).
- This test can now be taken online. Go to www.saxetesting.com and log in. Upon successful completion, your certificate can be printed out immediately.** AARC members' results are automatically forwarded to the AARC for accreditation.

Please consult www.clinicalfoundations.org for current annual renewal dates.

Participant's Evaluation

The goal of this program is to educate healthcare professionals on Non-invasive Respiratory Support in the NICU

- What is the highest degree you have earned? Circle one. 1. Diploma 2. Associate 3. Bachelor 4. Masters 5. Doctorate
- Indicate to what degree the program met the objectives:

Objectives

Upon completion of the course, the reader was able to:

- Describe what constitutes ventilator-associated pneumonia.**

Strongly Agree	Strongly Disagree
1 2 3	4 5 6
- Identify opportunistic pathogens.**

Strongly Agree	Strongly Disagree
1 2 3	4 5 6
- Identify route of transmission for infection.**

Strongly Agree	Strongly Disagree
1 2 3	4 5 6
- Describe interventions that could decrease the rate ventilator-associated pneumonia.**

Strongly Agree	Strongly Disagree
1 2 3	4 5 6
- Please indicate your agreement with the following statement. "The content of this course was presented without bias of any product or drug."**

Strongly Agree	Strongly Disagree
1 2 3	4 5 6

Answers

- | | | | |
|---|---|----|---|
| 1 | A B C D | 9 | A B C D |
| | <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> | | <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> |
| 2 | A B C D | 10 | A B C D |
| | <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> | | <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> |
| 3 | A B C D | 11 | A B C D |
| | <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> | | <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> |
| 4 | A B C D | 12 | A B C D |
| | <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> | | <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> |
| 5 | A B C D | 13 | A B C D |
| | <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> | | <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> |
| 6 | A B C D | 14 | A B C D |
| | <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> | | <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> |
| 7 | A B C D | 15 | A B C D |
| | <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> | | <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> |
| 8 | A B C D | 16 | A B C D |
| | <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> | | <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> |

Name & Credentials _____

Position/Title _____

Address _____

City _____ State ___ Zip _____

Phone # _____

Fax # or email _____

AARC Membership # _____

Credit given only if all information is provided. Please PRINT clearly.