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A Comprehensive Review of Ventilator-Associated Pneumonia: The Nurse's Role in Prevention and Management

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ABSTRACT

Ventilator-associated pneumonia (VAP) is a prevalent and serious nosocomial infection in intensive care units (ICUs), affecting mechanically ventilated patients. It is characterized by clinical signs of systemic infection, changes in sputum properties, and the identification of a causative microorganism post-intubation. VAP is categorized as early-onset or late-onset based on the timing of onset after intubation, with each type associated with different causative pathogens. The pathophysiology of VAP involves colonization of the respiratory and digestive tracts, followed by microaspiration of secretions. Diagnosis relies on the presence of new pulmonary infiltrates, respiratory decline, fever, and productive cough, along with microbiological evidence. Risk factors for VAP include host-related factors (e.g., immunosuppression, COPD), device-related factors (e.g., endotracheal tubes), and personnel-related factors (e.g., lapses in hygiene). Management of VAP focuses on appropriate antibiotic therapy tailored to individual risk factors and the timing of onset. Nurses play a crucial role in preventing VAP through evidence-based interventions such as stress ulcer prophylaxis, oral care with chlorhexidine, hand hygiene, use of endotracheal tubes with subglottic aspiration, maintaining proper endotracheal tube cuff pressure, and positioning patients in a semi-upright position. Implementing a comprehensive VAP prevention bundle has been shown to significantly reduce VAP incidence, antibiotic usage, and improve patient outcomes.

Keywords: Nurses, Vap, Ventilator-Associated Pneumonia

Introduction

Ventilator-associated pneumonia (VAP) is defined as pneumonia that develops at least 48 hours after the initiation of mechanical ventilation via endotracheal intubation. VAP is characterized by clinical manifestations of systemic infection, such as fever and alterations in white blood cell counts, alongside changes in the properties of sputum and the identification of a causative microorganism post-intubation (Kalil et al., 2016). It represents the second most prevalent hospital-acquired infection in intensive care units (ICUs) and is the leading nosocomial infection among mechanically ventilated patients (Kalanuria et al., 2014). The highest risk of developing VAP occurs within the initial five days of mechanical ventilation, with an incidence rate of 3%, and the average time from intubation to the onset of VAP is approximately 3.3 days. According to data published by the International Nosocomial Infection Control Consortium in 2013, the overall VAP rate is 13.6 cases per 1000 ventilator days. Research conducted by Chen et al. and Haghighi et al. indicated that the incidence of VAP varies widely, ranging from 9% to 69% across 1000 ventilator days (Chen et al., 2016; Haghighi et al., 2017).

Globally, the prevalence of VAP in ICUs is reported to range from 9% to 28%, with mortality rates between 24% and 70%, which is consistent with data from Turkey (Hunter, 2012; Öner Cengiz, 2019). The individual incidence of VAP is influenced by factors such as patient demographics, specific risk factors, and the hospital environment. National data from Turkey's 2017 National Hospital Infections Surveillance Network reveal that VAP rates per 1000 ventilator days in adult ICUs of university hospitals vary by specialty: 10.7 in anesthesiology and reanimation ICUs,

15.2 in brain surgery ICUs, 19.2 in thoracic diseases ICUs, 11.3 in internal medicine ICUs, 10.9 in neurology ICUs, and 7.4 in thoracic surgery ICUs. Notably, approximately 50% of all antibiotics administered in ICUs are used for the treatment of VAP (Hunter, 2012).

VAP is categorized based on the timing of onset after tracheal intubation. Early-onset VAP, which occurs within the first four days, is typically associated with antibiotic-sensitive pathogens. In contrast, late-onset VAP develops after four days of intubation and is often caused by highly resistant bacteria (Kalanuria et al., 2014). This classification is crucial for identifying the active pathogens responsible based on the timing of VAP onset and for guiding antibiotic selection in treatment. Pathogens commonly implicated in early-onset VAP include Staphylococcus aureus, Streptococcus pneumoniae, and Haemophilus influenzae, while late-onset VAP is frequently attributed to hospital-associated pathogens such as Pseudomonas aeruginosa, methicillin-resistant Staphylococcus aureus (MRSA), Klebsiella species, and Acinetobacter baumannii. The causative microorganisms can originate from the patient's endogenous flora, visitors, healthcare staff, other patients, or environmental sources.

VAP contributes to significant challenges in ICU settings worldwide, including high mortality rates, extended hospital stays, increased antibiotic usage, and consequently, elevated healthcare costs among adult patients undergoing endotracheal intubation (Kaş Güner & Kutlutürkan, 2021).

Pathophysiology

To effectively implement VAP prevention strategies, nurses must have a thorough understanding of its pathophysiology. VAP is classified into two types based on the duration between the initiation of mechanical ventilation (MV) and the onset of pneumonia: early-onset and late-onset VAP (Oliveira et al., 2014). Early-onset VAP occurs 48 to 96 hours after intubation and is primarily associated with antibiotic-susceptible organisms such as Staphylococcus aureus, Streptococcus pneumoniae, Haemophilus influenzae, Proteus species, Serratia marcescens, Klebsiella pneumoniae, and Escherichia coli. Late-onset VAP develops after 96 hours of intubation and is typically linked to antibiotic-resistant pathogens, including Pseudomonas aeruginosa, methicillin-resistant Staphylococcus aureus (MRSA), Acinetobacter species, and Enterobacter species (Augustyn, 2007).

According to the Centers for Disease Control and Prevention (CDC), patients with VAP may present with an elevated body temperature, leukopenia, the sudden appearance of purulent sputum, apnea, tachypnea, nasal flaring, chest wall retractions, grunting, wheezing, rales, rhonchi, and coughing (Peña-López et al., 2022). The pathophysiological process of VAP involves colonization of the respiratory and digestive tracts, followed by microaspiration of secretions from the upper and lower airways. VAP occurs when bacteria colonize the pulmonary parenchyma or lower respiratory tract in mechanically ventilated patients. This can result from the aspiration of secretions or the use of contaminated equipment. Pathogens may spread from various sources, including the oropharynx, sinus cavities, nares, dental plaque, gastrointestinal tract, patient-to-patient contact, and ventilator circuits, ultimately leading to bacterial colonization of the lungs (Coffin et al., 2008; Kunis & Puntillo,

2003). Inhalation of colonized bacteria from these sources elicits an active host immune response, culminating in the development of VAP.

The presence of an endotracheal tube provides a direct pathway for colonized bacteria to access the lower respiratory tract. Upper airway and oral secretions accumulate above the cuff of the tube, forming a biofilm along its surface. This biofilm, rich in bacteria, can be dislodged and transmitted to the lungs through ventilator-induced breaths, saline instillation into the tube, suctioning, coughing, or tube repositioning. Endotracheal tubes disrupt the natural barrier between the upper airway and trachea, bypassing the normal defenses and allowing bacteria to infiltrate the lower respiratory tract. This interference reduces the body's ability to filter and humidify air, compromises the cough reflex, and impairs mucociliary clearance (Kunis & Puntillo, 2003; Morehead & Pinto, 2000). Furthermore, endotracheal tubes serve as a site for bacterial adhesion in the trachea, increasing mucus production and secretion. This disrupts natural host defense mechanisms, heightening the risk of bacterial colonization and subsequent aspiration.

Microaspiration or macroaspiration of oropharyngeal or gastric fluids is a critical step in VAP development (Jam Gatell et al., 2012). The stomach can act as a reservoir for bacteria, and aspiration of gastric contents is another potential cause of VAP. Mechanically ventilated patients often have nasogastric or orogastric tubes for enteral feeding, medication administration, or gastric decompression. These tubes compromise the gastroesophageal sphincter, increasing gastrointestinal reflux and facilitating bacterial translocation to the oropharynx, where colonization of the upper airway occurs. Enteral feedings further elevate gastric pH and volume, promoting bacterial colonization and aspiration, which may lead to infection (Ferrer & Artigas, 2002).

Diagnosis

Despite the clinical importance of VAP, no definitive gold standard exists for its diagnosis, which may delay timely management. According to Anita Rae Modi et al. and updated guidelines, diagnosing VAP requires the simultaneous presence of the following criteria: new pulmonary infiltrates on chest imaging, respiratory decline, fever, and a productive cough (Modi & Kovacs, 2020). If chest imaging does not reveal new pulmonary infiltrates, the likelihood of VAP decreases, prompting the consideration of alternative causes of respiratory deterioration in hospitalized patients (Kalil et al., 2016). It is worth noting that in the early stages of pneumonia, only minimal inflammatory signs may be visible on radiographs, necessitating repeat imaging in patients with strong clinical suspicion, such as those exhibiting altered sputum production, cough, or fever. Furthermore, diagnostic criteria generally include at least two or three of the following: fever exceeding 38°C or hypothermia below 36.5°C; changes in sputum volume or characteristics, including increased suctioning needs; new or worsening cough; respiratory issues such as dyspnea, tachypnea, or apnea; abnormal pulmonary auscultation findings like rales, bronchial breath sounds, wheezing, or rhonchi. Additional criteria include deteriorating gas exchange after a period of stability or improvement, bradycardia or tachycardia indicating

hemodynamic changes, and elevated serum biomarkers such as C-reactive protein, procalcitonin, or leukocytosis (Chang & Schibler, 2016).

The challenge lies in the limited specificity of these clinical criteria when used independently, as they often overlap with sepsis symptoms or complications from other underlying conditions common in patients on prolonged mechanical ventilation (Chang & Schibler, 2016). Additionally, the clinical manifestation of VAP can be significantly influenced by the concurrent administration of medications in critically ill patients.

The CDC (Centers for Disease Control and Prevention) guidelines are commonly employed for diagnosing VAP in both pediatric and adult populations. Other diagnostic frameworks include the Great Ormond Street Hospital (GOSH) criteria and the Clinical Pulmonary Infection Score (CPIS). All these approaches require radiographic evidence of pneumonia, but the combination of clinical criteria and their interpretations varies. The challenges with these frameworks include reliance on subjective clinical symptoms and the need for radiographic evidence of lung infiltrates, which may not always be present in all cases (Iosifidis et al., 2018).

Blood cultures are recommended for detecting pathogens when respiratory cultures yield inconclusive results, as well as for identifying additional non-respiratory infections. These are valuable in differential diagnosis (Modi & Kovacs, 2020).

Obtaining sputum cultures is often limited by the patient’s ability to produce a sample. For those unable to provide sputum, alternative techniques such as endotracheal aspiration (preferred) or bronchoscopy with bronchoalveolar lavage may be employed. While invasive methods can provide more accurate data, they may cause patient discomfort and are generally reserved for immunocompromised patients or those not responding to standard therapies (Feinsilver et al., 1990; Kalil et al., 2016).

PCR testing is increasingly utilized to identify pathogens responsible for VAP. A nasal swab for Staphylococcus aureus demonstrates a high negative predictive value for methicillin-resistant Staphylococcus aureus (MRSA) colonization and aids in antibiotic stewardship by safely discontinuing anti-MRSA therapy when results are negative (Parente et al., 2018). Nasopharyngeal swabs for respiratory viral panels are particularly useful during influenza seasons, helping to identify viral infections and guide appropriate therapy. PCR testing, with its high sensitivity, allows for rapid bacterial detection in sputum or tracheal aspirate samples. Results, typically available within hours, should be interpreted alongside clinical findings, as they may yield false positives due to airway colonization by non-pathogenic bacteria. Quantitative PCR results that measure bacterial or viral load are particularly useful for correlating findings with clinical symptoms.

Procalcitonin testing helps differentiate between bacterial and viral infections, identifies possible co-infections, and assists in determining therapy duration. While clinical judgment is sufficient to initiate antibiotics, procalcitonin-guided discontinuation of antibiotic therapy has been associated with reduced mortality and treatment costs (Lam et al., 2018).

The common pathogens responsible for VAP include Pseudomonas species, Staphylococcus aureus, and various Gram-negative bacilli, with the proportions

differing between ICUs and pediatric ICUs (PICUs) (Vijay et al., 2018). The etiological agents also vary with the timing of onset. Early-onset VAP, occurring within four days of mechanical ventilation, is primarily caused by Staphylococcus aureus, Streptococcus pneumoniae, and Haemophilus influenzae. In contrast, late-onset VAP, developing after more than five days, is associated with Pseudomonas species, Acinetobacter species, and enteric Gram-negative bacilli. A prospective cohort study in North India identified the most frequently isolated VAP pathogens in descending order as Acinetobacter, Pseudomonas, Klebsiella, Enterobacter, and Escherichia coli (Liu & Zhang, 2021).

Risk factors

All intubated patients receiving ventilatory support are at risk of developing VAP. These risk factors can be classified into three categories: host-related, device-related, and personnel-related factors.

Host-Related Risk Factors

Host-related risk factors encompass preexisting medical conditions such as immunosuppression, chronic obstructive pulmonary disease (COPD), and acute respiratory distress syndrome (ARDS). Additional factors include the patient's body positioning, advanced age, level of consciousness, the frequency of intubations, blood transfusion, and the use of medications such as sedatives and antibiotics (Cason et al., 2007). A decreased level of consciousness compromises cough and gag reflexes, thereby increasing the risk of aspiration and subsequently VAP (Zeitoun et al., 2003).

Device-Related Risk Factors

Device-related factors include the use of an endotracheal tube, ventilator circuits, and nasogastric or orogastric tubes. When patients are positioned supine, there is an elevated risk of pulmonary aspiration as secretions tend to accumulate above the endotracheal tube cuff. If cuff pressure is inadequately maintained, these pooled secretions can result in micro aspiration or bacterial leakage into the trachea. Similarly, nasogastric and orogastric tubes disrupt the gastroesophageal sphincter, leading to reflux and increasing the likelihood of VAP (Fulbrook & Mooney, 2003).

Personnel-Related Risk Factors

Personnel-related factors involve lapses in hygiene and the use of protective equipment. Improper hand hygiene, failure to change gloves between patient interactions, and not wearing appropriate personal protective equipment when antibiotic-resistant bacteria are present contribute significantly to the risk of VAP. For example, inadequate handwashing during procedures like suctioning or ventilator circuit manipulation increases the probability of cross-contamination. Failure to adhere to handwashing protocols or change gloves between patients has been directly linked to higher incidences of VAP. Moreover, not using protective equipment when antibiotic-resistant pathogens are detected exacerbates the risk of cross-contamination among patients (Kollef, 2004; Tablan et al., 2004, p. 2).

Management

The management of VAP presents a critical yet challenging task for healthcare teams in the ICU. It relies on the interplay between the infective agent, the host's immune response, and the antimicrobial therapy utilized. Pulmonary infection develops when pathogens infiltrate the lungs. However, a comprehensive and proactive strategy has been developed for managing VAP, incorporating updates on local epidemiology, daily evaluation of VAP cases, utilization of diagnostic tools, and assessment of host responses through clinical and biochemical markers. Two key steps recommended in the management of VAP are etiologic diagnostic testing and the prompt initiation of antibiotic therapy (Diaz et al., 2009).

Antibiotic Management

For the initial treatment of VAP, the selection of appropriate antibiotics must be tailored to each patient, taking into consideration individual risk factors for multidrug-resistant (MDR) pathogens and the timing of disease onset. In addition, the spectrum of antimicrobial activity, appropriate dosing, pharmacokinetics, and potential adverse effects of the chosen antibiotics should be meticulously evaluated (American Thoracic Society & Infectious Diseases Society of America, 2005).

For patients with early-onset VAP who have no risk factors for MDR pathogens, the currently recommended empiric antibiotic options include:

- Ceftriaxone
- Fluoroquinolones
- Ampicillin-sulbactam
- Ertapenem

For patients with VAP who present risk factors for MDR pathogens or have late-onset VAP, the initial antibiotic regimen may include:

- Antipseudomonal cephalosporins (e.g., cefepime, ceftazidime)
- Antipseudomonal carbapenems (imipenem or meropenem)
- Beta-lactam/beta-lactamase inhibitors (e.g., piperacillin-tazobactam) in combination with an antipseudomonal fluoroquinolone (e.g., ciprofloxacin) or an aminoglycoside
- Linezolid or vancomycin, especially in the presence of risk factors for methicillin-resistant *Staphylococcus aureus* (MRSA)
- Telavancin, which is specifically indicated for hospital-acquired bacterial pneumonia (HABP) or VAP caused by susceptible isolates of *Staphylococcus aureus*, including methicillin-susceptible and methicillin-resistant strains, when alternative treatments are deemed unsuitable (DiCocco & Croce, 2009).

Nurse's role in prevention of VAP development

The Healthcare Improvement Institute's care bundle is an evidence-based collection of interventions that significantly enhance patient outcomes when implemented together. The original VAP prevention bundle includes maintaining head-of-bed elevation, oral care with chlorhexidine, stress ulcer prophylaxis, deep venous thrombosis prophylaxis, and daily sedation assessments paired with spontaneous breathing trials. In 2010, this bundle was updated to incorporate subglottic aspiration and endotracheal tube cuff pressure monitoring. While the efficacy of each intervention has been questioned, evidence supports their combined effectiveness in reducing VAP incidence (Youngquist et al., 2007).

A study implementing a systematic VAP prevention bundle based on the Health Improvement Institute's methodology reported significant decreases in VAP rates, antibiotic usage, and MRSA acquisition. Despite a total package compliance rate of only 70% compared to the recommended 95%, the outcomes were markedly improved. Effective preventive strategies include limiting unnecessary stress ulcer prophylaxis, using selective digestive decontamination, providing oral care with chlorhexidine, ensuring hand hygiene, preferring oral intubation over nasal intubation, using endotracheal tubes with continuous subglottic aspiration, avoiding prolonged ventilator disconnection, minimizing unplanned extubation and reintubation, and maintaining appropriate patient positioning (Álvarez Lerma et al., 2014).

1. Stress Ulcer Prophylaxis

Stress ulcer prophylaxis is routinely applied to ICU patients, with common medications including H2 receptor antagonists, proton pump inhibitors (PPIs), and sucralfate. While these drugs prevent gastrointestinal bleeding by suppressing gastric acid secretion, they can increase gastric pH, promoting bacterial colonization and elevating the risk of VAP. To minimize this risk, unnecessary use of stress ulcer prophylaxis should be avoided (Álvarez Lerma et al., 2014). A randomized controlled trial by Bashar et al. comparing PPIs and H2 receptor antagonists in trauma patients found that PPIs were associated with a threefold increase in VAP risk compared to H2 receptor antagonists (Bashar et al., 2013). The "Manual for the Prevention of Healthcare-Related Pneumonia" advises selecting agents based on the patient's clinical condition.

2. Selective Digestive System Decontamination

Selective digestive decontamination aims to eliminate Gram-negative bacteria and fungi from the digestive tract while preserving anaerobic flora (Álvarez Lerma et al., 2014). This approach involves using antimicrobial agents like colistin, tobramycin, and amphotericin-B to prevent the colonization of pathogenic microorganisms (de Smet et al., 2009). Studies indicate that selective digestive decontamination reduces mortality by approximately 3.5%, while selective oropharyngeal decontamination reduces it by about 2.9%. However, concerns about antibiotic resistance have

prevented the routine recommendation of this practice in international guidelines (de Smet et al., 2009).

3. Oral Care with Chlorhexidine

Oral care significantly lowers VAP risk by reducing oral bacterial load, translocation, and pulmonary colonization. Ventilated patients with endotracheal tubes cannot maintain their oral hygiene, increasing biofilm formation by pathogenic bacteria (29). A meta-analysis involving 2,144 patients demonstrated that using oral antiseptics substantially reduced VAP incidence (Micek & Skrupky, 2010). Institutions are encouraged to establish oral care protocols using antiseptic solutions to minimize bacterial colonization in the oropharynx (Hua et al., 2016). Chlorhexidine gluconate is particularly effective against bacteria causing late-onset VAP. Oral care should be performed every 2–4 hours, covering teeth, cheeks, and tongue, using a 0.12% chlorhexidine solution or mouth swabs with 1.5% hydrogen peroxide (Gupta et al., 2016).

4. Hand Hygiene

Prominent signage at ICU entry points reminding staff to wash hands and wear gloves is an inexpensive yet vital measure to prevent bacterial cross-contamination between patients. Effective hand hygiene reduces bacterial colonization in the oropharynx and gastrointestinal tract. Hands should be washed for at least 10 seconds before and after patient contact. If gloves are not visibly contaminated after handling secretions or equipment, alcoholic hand antiseptics can be used (Augustyn, 2007).

5. Oral Intubation

The CDC/NHSN manual (2019) recommends orotracheal over nasotracheal intubation to reduce VAP risk, as the latter can promote aspiration of infected secretions from the nasal sinuses. Unless contraindicated, oral intubation should be prioritized (Oliveira et al., 2014).

6. Endotracheal Tubes with Continuous Subglottic Aspiration

Endotracheal tubes with continuous subglottic aspiration help mitigate VAP by preventing the accumulation of secretions above the endotracheal cuff, which can leak into the lower airways (Hua et al., 2016). Their use has been shown to reduce VAP rates by 45–50%. Utilizing specialized intubation and tracheostomy tubes that facilitate subglottic secretion removal is strongly recommended to reduce micro aspiration and subsequent infection risks.

7. Non-Invasive Mechanical Ventilation

The presence of an endotracheal tube (ETT) is identified as the most significant risk factor for VAP development, increasing the risk by 6–21 times (Oliveira et al., 2014).Non-invasive mechanical ventilation (NIMV) is recommended when feasible to prevent endotracheal intubation and reduce the risk of VAP in patients experiencing acute respiratory failure. A meta-analysis examining patients with chronic obstructive pulmonary disease (COPD) compared the effects of invasive positive-pressure mechanical ventilation (MV) and non-invasive positive-pressure

MV, showing that NIMV significantly decreased VAP incidence, mortality rates, and the length of ICU and hospital stays (Burns et al., 2013). While NIMV presents a viable alternative to invasive MV in certain clinical conditions, it is not universally applicable and cannot replace MV or endotracheal intubation in all cases (Oliveira et al., 2014). Conditions suitable for NIMV include COPD, cardiogenic pulmonary edema, acute hypoxemic respiratory failure, and respiratory failure in immunocompromised patients (Kalil et al., 2016).

8. Endotracheal Tube Cuff Pressure

Maintaining an endotracheal tube cuff pressure between 20–30 cmH_2O is a key measure in preventing VAP (Kalanuria et al., 2014). A cuff pressure lower than 20 cmH_2O may lead to gas leakage and the aspiration of subglottic secretions into the lower respiratory tract, facilitating bacterial entry. Conversely, pressures exceeding 30 cmH_2O may cause mucosal ischemia due to barotrauma (Augustyn, 2007). For this reason, continuous monitoring of endotracheal tube cuff pressure is necessary.

9. Aspiration Method

Tracheobronchial aspiration involves the removal of respiratory system secretions using a vacuum device with negative pressure in patients with ETT. This procedure can be performed using either an open or closed system. In the open system, the patient is briefly disconnected from the ventilator, and secretions are removed using a disposable suction catheter under surgical asepsis. In the closed system, a reusable suction catheter remains connected to the ventilator circuit, allowing secretions to be removed without requiring sterile gloves (Kandeel & Tantawy, 2012). A randomized controlled trial comparing open-system aspiration (n=75) and closed-system aspiration (n=66) methods found no significant differences in VAP incidence between the two techniques (Elmansoury & Said, 2017). Current VAP prevention guidelines confirm that neither method has a distinct advantage regarding pneumonia risk.

10. Mechanical Ventilator Breathing Circuit, Frequency of Humidifier Use, and Replacement

Frequent replacement of ventilator circuits is a risk factor that can prolong hospital stays. It is recommended to use a single respiratory circuit for patients on MV, replacing it only in cases of mechanical damage or contamination (e.g., blood, vomit, or purulent secretions) (Blanquer et al., 2011). Accumulated fluid in breathing circuits should be drained periodically, and only sterile water should be used in humidifier containers. Water in these containers should be replaced, rather than refilled, when depleted. Routine replacement of humidifier filters is unnecessary unless contamination or malfunction is evident. Heat-moisture exchanger traps (HME) are preferred over heated humidifiers unless contraindicated (Dimopoulos et al., 2013; Vallés et al., 2013).

11. Nutrition

Enteral nutrition is prioritized to prevent complications associated with parenteral nutrition, such as infections, higher costs, and fluid-electrolyte imbalances. However, nasogastric feeding may increase bacterial colonization, and aspiration risks due to increased gastric volume and pH. Nasogastric feeding has been linked to a threefold increase in VAP risk (Kahraman & Ozdemir, 2015). To minimize this risk, it is recommended to elevate the patient's head to 30–45° during feeding and monitor gastric residual volume (Uysal et al., 2012). Residual volumes of 150–200 ml should prompt temporary cessation of feeding for 1–2 hours (Keyt et al., 2014).

12. Disconnecting from the Ventilator (Weaning)

Weaning patients from MV requires close monitoring for signs of failure, such as tachypnea, tachycardia, sweating, oxygen desaturation, hypertension, or increased anxiety (Keyt et al., 2014). Prolonged MV duration heightens VAP risk, underscoring the importance of minimizing ventilation time. Spontaneous breathing trials, where patients breathe independently through an ETT connected to a T-piece, are the most common weaning method. Current guidelines recommend protocols to assess readiness for weaning, including discontinuing sedative medications, which has been shown to reduce MV duration, ICU stays, and VAP risk (Blanquer et al., 2011).

13. Prevention of Unplanned Extubation and Reintubation

Unplanned extubations frequently necessitate reintubation within 48 hours, significantly increasing VAP risk due to the likelihood of aspiration. Measures to minimize unplanned extubations include ensuring adequate ICU staffing, routinely checking the integrity of intubation tubes, and carefully planning extubation procedures (Keyt et al., 2014).

14. Half-Sitting Position

Elevating the head of the bed reduces the risk of stomach content aspiration, thereby decreasing VAP incidence. A semi-upright position (30–45°) is pivotal in VAP prevention. The supine position (0°) facilitates aspiration of contaminated gastric contents, increasing VAP incidence (Xie et al., 2019). When not contraindicated by conditions such as increased intra-abdominal pressure, maintaining a head elevation of 30–45° is recommended (Bassi et al., 2017). A randomized controlled study comparing head elevations of ≥30° and 0–10° found significantly reduced VAP rates in the ≥30° group. However, higher head elevations (≥45°) have been associated with extended weaning durations and increased pressure injuries, particularly in the sacral region (Schallom et al., 2015). Another study reported a 12.5% reduction in VAP rates in patients with 45° head elevation compared to 30°, though the difference was not statistically significant (Najafi Ghezeljeh et al., 2024). While the supine position should be avoided in ventilated patients, clinical practice guidelines suggest a head elevation of at least 30° to minimize aspiration risk (Keyt et al., 2014).

Conclusion

Ventilator-associated pneumonia (VAP) remains a significant clinical challenge, posing risks of high morbidity, mortality, and healthcare costs, particularly in intensive care settings. Its development is influenced by a complex interplay of

patient-related, device-related, and personnel-related factors. Despite advancements in diagnostic techniques and management protocols, no universal gold standard exists for VAP diagnosis, complicating timely and accurate identification. Effective management strategies rely on evidence-based approaches, including accurate etiologic diagnosis, prompt initiation of empiric antibiotic therapy tailored to the patient's risk factors, and ongoing reassessment based on clinical and microbiological findings.

Prevention remains the cornerstone of mitigating VAP incidence, with a strong emphasis on adopting comprehensive care bundles, maintaining hand hygiene, optimizing mechanical ventilation practices, and employing non-invasive mechanical ventilation whenever feasible. Interdisciplinary collaboration among healthcare professionals, regular training, and adherence to established protocols are critical to achieving better patient outcomes and minimizing the burden of VAP.

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