

Airway Clearance Requirements Among Patients With Mechanical Ventilation and Artificial Airways: An Overview

Jane M. Braverman, Ph.D.

Employed by Hill-Rom

Author Profile

Dr. Jane Braverman has experience in laboratory, academic, and technological medicine. She has worked as a medical technologist in basic research at the University of Minnesota and in clinical laboratory medicine at Twin Cities hospitals. After earning her Ph.D., she served as assistant professor in the Department of the History of Medicine at the University of Minnesota. Dr. Braverman, as research analyst/writer for Hill-Rom, has contributed to a variety of research endeavors and developed white papers, case studies, and other supporting materials.

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A mechanical ventilator is a device designed to augment or replace spontaneous respiration. It is used for recovery from an acute respiratory crisis and/or for long-term therapy for patients with chronic hypoventilation. For those patients for whom noninvasive ventilation is not appropriate, placement of an artificial airway is a necessary component of both short- and long-term mechanical ventilation (MV). For short-term use, endotracheal tubes are preferred. For long-term use, a tracheostomy is required. A tracheostomy may also be employed without the use of a ventilator to bypass the upper airway and maintain airway patency, to reduce the risk of aspiration, and to aid in clearing secretions from the central airways.^{1,2}

In those patients who cannot be maintained adequately with noninvasive therapies, tracheostomy and/or MV may provide life-sustaining support. Mechanical ventilation facilitates alveolar ventilation and pulmonary gas exchange and assists thoraco-diaphragmatic dynamics.

Most commonly, MV is provided in the intensive care unit. The majority of patients receive short-term MV therapy until the cause of respiratory crisis resolves or death occurs, often within several days. Patients requiring long-term or permanent MV are referred to as chronic ventilator-assisted individuals (VAIs) or sometimes, when MV use must be permanent, as ventilator-dependent individuals.³

As MV technology continues to improve, a significant number of long-term MV patients are receiving treatment in extended care facilities, residential treatment centers, or in their homes,⁴ thereby reducing hospital stays and costs. With the use of tracheostomies, management outside the hospital is now recognized as a feasible, cost-effective alternative.^{5,6} However, because of the complexity of such care, the technical needs and medical issues associated with non-hospital MV utilization present major challenges to health care management teams.⁷

As the ventilator assumes the work of breathing, alterations occur in respiratory physiology, including changes in intrathoracic pressure, ventilation distribution, and cardiac output.⁸ Significant pulmonary risks are associated with the use of both artificial airways and mechanical ventilation,⁹ including, most commonly, aspiration and pneumonia. Bronchoconstriction can also occur, complicating the management of patients with underlying chronic obstructive pulmonary disease or asthma. Retention of lower airway secretions, common among VAIs, leads to atelectasis and poor gas exchange and provides an ideal medium for colonization by aspirated bacteria.

Although risks vary according to underlying pathology, patients who require an artificial airway and/or MV share the following risks of secretion retention and consequent pulmonary complications:

- impaired airway clearance;
 - infection risks associated with bypassed upper airways; and
 - immobility and associated risks of pulmonary compromise.
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Impaired airway clearance mechanisms

Impaired airway clearance is both an indication for and a consequence of an artificial airway.¹⁰ Frequently, translaryngeal intubation and/or tracheostomy are performed to clear secretions retained in the central airways.¹¹ However, the presence of such an apparatus can also cause mucosal inflammation and significant mucus hypersecretion,¹² predisposing to infection and encouraging atelectasis and hypoxia. When there is coexisting airflow obstruction or neuromuscular weakness, the work of breathing may be dramatically increased.

Moreover, the placement of an artificial airway bypasses the upper airway and the proximal trachea. As a

consequence, certain physiological airway clearance functions are lost or impaired.

- Impairment of the mucociliary system: Bypassed airways eliminate the humidifying, warming and filtering function of the upper airway. Breathing dry air significantly reduces mucus transport velocity.¹³ It is not known whether the effect of decreased humidification on mucus mobility is a result of impairment of the mucociliary transport system, moisture-related changes in mucus viscosity and elasticity, or both.^{14,15} However, regardless of causes, increased viscosity and hyperproduction of mucus impairs ciliary function and exceeds the capacity for mucociliary transport.^{16,17}

- Impairment of the cough reflex: Artificial airway placement may compromise the cough reflex.¹⁸ Effective cough requires: inspirations sufficient to expand the airways and allow airflow to reach distal secretions; glottis closure and contraction of expiratory muscles to increase intrathoracic pressure; and sudden opening of the glottis to create a high air flow rate. Bypassing the glottis diminishes or eliminates its role in producing a forceful expulsion of air and secretions. Additionally, the reduced diameter and rigidity of an artificial airway increases airway resistance and contributes to restrictive lung disease, characterized by a reduced ability to exercise to maintain bellows function, lung volume, and aerobic capacity. Restrictive lung disease reduces the ability to generate airflow sufficient to produce an effective cough.^{19,20} When secretion viscosity rises and elasticity falls, or when airway resistance is increased, ineffective cough may actually cause retrograde movement of secretions to the lung periphery or aspiration to the contralateral lung.^{21,22,23}
- Structural damage to airway tissue: An artificial airway may result in both acute traumatic injuries to airway tissue and long-term physiological responses to a foreign irritant.²⁴ The intubation procedure itself may precipitate laryngeal edema, inflammation, ulceration, or hematoma. High cuff pressures may induce mucosal ischemia and pressure necrosis of tracheal tissue. Risk factors associated with long-term or permanent tracheostomy include tracheal erosion, glottic and/or tracheal stenosis, tracheal dilation, and tracheomalacia.²⁵ Artificial airway may also be associated with denuded epithelium and ciliary damage.²⁶

Infection risks associated with bypassed airways

In the United States, pneumonia, second only to urinary tract infections²⁷ as a common nosocomial infection, is the leading cause of death from community-acquired infection in hospitals.²⁸ Pulmonary infection is a common complication in patients treated with endotracheal intubation and mechanical ventilation. A recent international prevalence study involving more than 1,000 intensive care units indicated that ventilator-associated pneumonia (VAP) is responsible for approximately 90% of infections in patients receiving assisted ventilation.²⁹ Although the incidence of opportunistic infection varies greatly according to a number of underlying and contributing factors, the average rate of infection is approximately 25%.^{30,31} The risk increases with the duration of invasive airway therapy.³² Although debate

exists regarding the mortality attributable to VAP among other causes of death, a substantial proportion of deaths result from VAP. Best estimates suggest that the crude mortality rate of VAP is 50%, with attributable mortality accounting for about 30% of total mortality.³³

Inpatients with bypassed upper airways, the potential for infection is enhanced by a number of factors including:

- bypass of the filtering function of the upper airway;
- introduction of bacteria during aspiration;
- impairment of mucociliary transport;
- hypersecretion of airway mucus;
- introduction of bacteria during intubation or suctioning;
- contaminated tubing, ventilator circuits, etc.; and
- patterns of antibiotic use.

The presence of an artificial airway impairs normal airway protective reflexes. Endotracheal tubes prevent the aspiration of sizable particles, but they may permit pharyngeal secretions to enter the trachea via the interstices of the balloon cuff, resulting in tracheal colonization and increased risk of serious pulmonary infection.³⁴

Aspiration can have dire consequences, especially for debilitated patients with underlying pulmonary disease. Deleterious consequences include chemical pneumonitis,³⁵ bacterial pneumonitis, mechanical obstruction, and transient hypoxia.

Most episodes of hospital-acquired pneumonia (which may be antibiotic resistant) are thought to derive from aspiration of pathogenic oropharyngeal bacteria into the distal bronchi.³⁶ The premise that nosocomial pneumonia in intubated patients develops from aspiration of colonized oropharyngeal or tracheal secretions is supported by studies showing that upper airway colonization usually precedes such infection.^{37,38} The very high rate of endotracheal tube colonization may represent a persistent source of organisms causing pneumonia.³⁹

In patients with impaired mucociliary clearance, secretions retained in the lower airways provide an ideal environment for colonizing organisms. Aspirated bacteria proliferate in the lower airways, stimulating a host inflammatory response. If the rate of bacterial proliferation exceeds the rate of host inflammatory clearance, the inflammatory response spreads to the contiguous bronchiole wall, leading to bronchiolitis, and may proceed to involve the alveoli and other interstitial tissues, leading to bronchopneumonia.⁴⁰ Patients who have impaired mechanical, humoral, or cellular lung defense mechanisms are especially vulnerable.

Aspiration of colonized gastric contents may also play a role in the etiology of pneumonia among patients with artificial airways. Although the role of gastric colonization and subsequent gastric aspiration as a risk factor for VAP remains controversial, most investigators agree that significant aspiration of contaminated gastric contents into the lower airway can lead to the development of VAP and may induce lung injury.^{41,42,43} Although gross aspiration of large volumes of gastric contents is uncommon with artificial airways, the presence of invasive tubing can impair swallowing and esophageal motility, increasing the risk of aspiration.^{44,45} Gastroesophageal aspiration may result in laryngitis, recurrent bronchitis, pneumonitis, and bronchiectasis. It may also lead to chemical pneumonia, increased secretion production, bronchospasm, and hypoxia.⁴⁶

Pathogens may also be introduced directly during the intubation procedure during routine suctioning, or via contaminated tubing, ventilator circuits, etc.

Overzealous use of antibiotics, especially broad-spectrum agents, predisposes to the development of antibiotic-resistant infections.⁴⁷

The pathogenesis of pneumonia associated with artificial airways and MV has been investigated extensively in the last decade.^{48,49} The roles of tracheal contamination from infected sinus and contaminated gastric secretions, bacterial adhesins and receptors, and other factors have received detailed attention.^{50,51,52} Despite numerous studies, debate continues concerning the pattern and sequence of endotracheal tube colonization in intubated and MV patients. However, regardless of the precise sequence of events, significant colonization typically involves the lower part of the respiratory tract.⁵³

Immobility

Physical exercise is an important component of normal airway clearance, increasing mucus clearance by as much as 41% compared to quiet breathing at rest.⁵⁴ For those peripheral airways not effectively cleared by coughing, exercise is the most important component of the clearing mechanism.

Mechanically ventilated individuals with tracheostomies typically manifest both impaired mobility and absent or impaired cough function. As a consequence, they are prone to significant secretion retention and become vulnerable to bacterial colonization and a host of associated pulmonary complications, including recurrent infection leading to tracheitis, bronchitis, bronchiectasis, impaired gas exchange, and tissue damage.

Indications for airway clearance therapy

Impaired mucociliary clearance is both an indication for and a consequence of the use of artificial airways. Artificial airways bypass important components of the normal airway clearance system and are irritants, which promote mucus hypersecretion and impair mucociliary clearance.

Poor secretion clearance disrupts the defense mechanisms of airway epithelium.

Retained airway secretions lead to:

- Airway obstruction, inhibiting O₂/CO₂ exchange
- Ineffective cough, resulting from mucus accumulation in the airways
- Accumulation of particulate matter, including pathogens

In individuals with bypassed upper airways, recurrent respiratory tract infections exacerbate lower respiratory tract secretion retention, frequently resulting in a vicious cycle of pneumonia, pulmonary atelectasis and respiratory failure.⁵⁵ During such events, existing pulmonary deficits are worsened by bronchial mucus plugging and further weakening and fatigue of respiratory muscles. Such episodes initiate a downward spiral involving recurrent or refractory pneumonia, emergency hospitalizations and, eventually, fatal respiratory failure.

Prophylactic and therapeutic airway clearance is a very important component in optimizing outcomes for this patient population.

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