CHAPTER

14

Acute Respiratory Failure

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INTRODUCTION

Acute respiratory failure (ARF) occurs in many disease states. It may be the patient's primary problem or a complicating factor in other conditions. This chapter reviews the pathophysiology of ARF, several common causes, and the nursing care involved in the treatment of these patients.

ACUTE RESPIRATORY FAILURE

Definition

ARF is defined as a state of altered gas exchange in which the respiratory system fails in either oxygenation or carbon dioxide (CO_2) elimination. These failures are classified into one of two categories: type 1 hypoxemic or oxygenation failure, or type II hypercapnic or ventilatory failure. Hypoxemic or oxygenation failure is characterized by an abnormal arterial blood gas (ABG) value obtained with the patient breathing room air; partial pressure of oxygen (O_2) in arterial blood (PaO_2) is less than 60 mm Hg, and the partial pressure of carbon dioxide (CO_2) level ($PaCO_2$) is normal or low. Hypercapnic or ventilatory failure is characterized by a $PaCO_2$ of greater than 50 mm Hg with a pH of less than 7.30.[4] ARF differs from chronic respiratory failure in the length of time necessary for it to develop. ARF occurs rapidly over minutes to hours, with little time for physiological compensation. Chronic respiratory failure develops over time and allows the body's compensatory mechanisms to activate. ARF and chronic respiratory failure are not mutually exclusive. ARF may occur when a person who has chronic respiratory failure develops a sudden respiratory infection or is exposed to other types of stressors. This is referred to as acute-on-chronic respiratory failure. Obese individuals may also be at a greater risk for respiratory failure (see box, "Bariatric Considerations").

BARIATRIC CONSIDERATIONS

Several pathophysiological mechanisms may contribute to respiratory failure in obese individuals:

Hypoxemic Respiratory Failure
- Increase in PAO_2/PaO_2 gradient secondary to ventilation/perfusion imbalance associated with hyperperfusion and airway closure/collapse

Hypercapneic Respiratory Failure
- Decrease in compliance
- Increase in resistance
- Diminished respiratory strength
- Respiratory muscle fatigue
- Diaphragm dysfunction
- Altered ventilator pattern

Coexisting Conditions
- Sleep apnea-hypopnea syndrome
- Chronic obstructive pulmonary disease

Aggravating Conditions
- Supine position
- Rapid eye movement (REM) sleep

Adapted from Rabec C, Ramos P, Veale D. Respiratory complications of obesity. *Archivos de Bronconeumologia.*, 2011;47:252-261.

Pathophysiology

Failure of Oxygenation

Failure of oxygenation is present when the PaO_2 cannot be adequately maintained. Five generally accepted mechanisms that reduce PaO_2 and create a state of hypoxemia are (1) hypoventilation, (2) intrapulmonary shunting, (3) ventilation-perfusion mismatching, (4) diffusion defects, and (5) decreased barometric pressure (Figure 14-1). Decreased barometric pressure, which occurs at high altitudes, is not addressed in this text. Nonpulmonary conditions such as decreased cardiac output and low hemoglobin level may also result in tissue hypoxia.

Hypoventilation. In the normal lung, the partial pressure of alveolar O_2 (PAO_2) is approximately equal to the arterial O_2 (PaO_2). Alveolar ventilation refers to the amount of gas that enters the alveoli per minute. If the alveolar ventilation is reduced because of hypoventilation, the PAO_2 and the PaO_2 are reduced. Factors that may lead to hypoventilation include a drug overdose that causes central nervous system depression, neurological disorders that cause a decrease in the rate or depth of respirations, and abdominal or thoracic surgery leading to shallow breathing patterns secondary to pain on inspiration. Hypoventilation also produces an increase in the alveolar CO_2 level because the CO_2 that is produced in the tissues is delivered to the lungs but is not released from the body.

Intrapulmonary shunting. In normally functioning lungs, a small amount of blood returns to the left side of the heart without engaging in alveolar gas exchange. This is referred to as the physiological shunt. If, however, a larger amount of blood returns to the left side of the heart without participating in gas exchange, the shunt becomes pathological and a decrease in the PaO_2 occurs.[4] The condition exists when areas of the lung that are inadequately ventilated are adequately perfused (see Figure 14-1). The blood, therefore, is shunted past the lung and returns unoxygenated to the left side of the heart. Causes of shunting include atrial or ventricular septal defects, atelectasis, pneumonia, and pulmonary edema.[3]

As the shunt worsens, the PaO_2 continues to decrease. This cause of hypoxemia cannot be effectively treated by solely increasing the fraction of inspired O_2 (FiO_2) because the increased oxygen is unable to reach the alveoli. Treatment is directed toward opening the alveoli and improving ventilation.

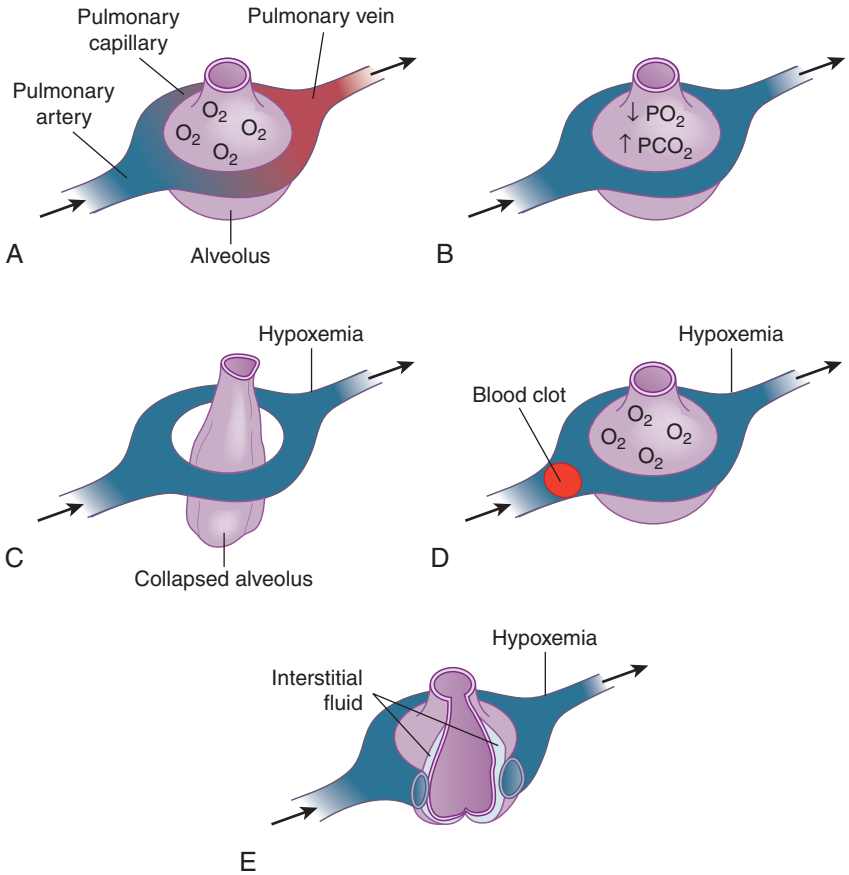

FIGURE 14-1 Pulmonary causes of hypoxemia. **A,** Normal alveolar-capillary unit. **B,** Hypoventilation causes an increased $PaCO_2$ and decreased PaO_2. **C,** Shunt. **D,** Ventilation-perfusion mismatch resulting from pulmonary embolus. **E,** Diffusion defect due to increased interstitial fluid.

Ventilation-perfusion mismatch. Gas exchange in the lungs is dependent upon the balance between ventilated areas of the lung (ventilation) receiving blood flow (perfusion). The rate of ventilation ($\dot{V}$) usually equals the rate of perfusion ($\dot{Q}$), resulting in a ventilation-to-perfusion ($\dot{V}/\dot{Q}$) ratio of 1.0. If ventilation exceeds blood flow, the $\dot{V}/\dot{Q}$ ratio is greater than 1.0; if ventilation is less than blood flow, the $\dot{V}/\dot{Q}$ ratio is less than 1. Both of these conditions are examples of $\dot{V}/\dot{Q}$ mismatch. In respiratory failure, $\dot{V}/\dot{Q}$ mismatch is the most common cause of hypoxemia and can often be corrected by increasing the FiO_2.[3] $\dot{V}/\dot{Q}$ mismatch can occur in conditions such as pneumonia or pulmonary edema when obstructed airways inhibit ventilation (and perfusion is normal), or in the case of pulmonary embolism when a clot in the pulmonary circulation obstructs perfusion.[3]

Diffusion defects. Diffusion is the movement of gas from an area of high concentration to an area of low concentration. In the lungs, O_2 and CO_2 move between the alveoli and the blood by diffusing across the alveolar-capillary membrane. The alveolar-capillary membrane has six barriers to the diffusion of O_2 and CO_2: surfactant, alveolar epithelium, interstitial fluid, capillary endothelium, plasma, and red blood cell membrane. Under normal circumstances, O_2 and CO_2 diffuse across the alveolar-capillary membrane in 0.25 seconds. The distance between an alveolus and a pulmonary capillary is usually only one or two cells thick. This narrowness of space facilitates efficient diffusion of O_2 and CO_2 across the cell membrane.

In respiratory failure, the distance between the alveoli and the capillaries may be increased by the accumulation of fluid in the interstitial space (see Figure 14-1). Changes in capillary perfusion pressure, leakage of plasma proteins into the interstitial space, and destruction of the capillary membrane contribute to the buildup of fluids around the alveolus. Fibrotic changes in the lung tissue itself, such as those seen in chronic obstructive pulmonary disease (COPD), may also contribute to a reduction in the diffusion capacity of the lung. As this capacity is reduced, PaO_2 is the first parameter affected and hypoxemia results. Because CO_2 is more readily diffusible than O_2, hypercapnia is a late sign of diffusion defects.

Low cardiac output. Adequate tissue oxygenation depends on a balance between O_2 supply and demand. The mechanism for delivering O_2 to the tissues is cardiac output. A normal cardiac output results in the delivery of 600 to 1000 mL/min of O_2, which generally exceeds the normal amount of O_2 needed by the tissues. If the cardiac output decreases, less oxygenated blood is delivered. To maintain normal aerobic metabolism in low cardiac output states, the tissues must extract increasing amounts of O_2 from the blood. When this increase in extraction can no longer compensate for the decreased cardiac output, the cells convert to anaerobic metabolism. This results in the production of lactic acid, which depresses the function of the myocardium and further lowers cardiac output.

Low hemoglobin level. Approximately 95% of the body's O_2 is transported to the tissues bound to hemoglobin. Each gram of hemoglobin can carry 1.34 mL of O_2 when all of its O_2 binding sites are completely filled. Oxygen saturation (SaO_2) refers to the percentage of O_2 binding sites on each hemoglobin molecule that are filled with O_2. The hemoglobin of a healthy person breathing room air is about 95% saturated. If a patient's hemoglobin level is less than normal, the O_2 supply to the tissues may be impaired and tissue hypoxia can occur. An alteration in hemoglobin function (i.e., carbon monoxide poisoning or sickle cell disease) can also decrease O_2 delivery to the tissues.

Tissue hypoxia. The final step in oxygenation is the use of O_2 by the tissues. Anaerobic metabolism occurs when the tissues cannot obtain adequate O_2 to meet metabolic needs. In addition, some conditions such as cyanide poisoning may leave the tissues unable to use O_2 despite normal O_2 delivery.[26] Anaerobic metabolism is inefficient and results in the accumulation of lactic acid. The point at which anaerobic metabolism begins to occur is not known and may vary with different organ systems. The effects of tissue hypoxia vary with the severity of the hypoxia but may result in cellular death and subsequent organ failure.

Failure of Ventilation

$PaCO_2$ is the index used to evaluate ventilation. When ventilation is reduced, $PaCO_2$ is increased (hypercapnia). When ventilation is increased, $PaCO_2$ is reduced (hypocapnia). Hypoventilation and $\dot{V}/\dot{Q}$ mismatching are the two mechanisms responsible for hypercapnia. Hypercapnia greatly increases cerebral blood flow. The patient may appear restless and anxious, and may demonstrate slurred speech and a decreased level of consciousness.

Hypoventilation. Hypoventilation is the cause of respiratory failure that occurs in patients with central nervous system abnormalities, neuromuscular disorders, drug overdoses, and chest wall abnormalities (see Figure 14-1). In hypoventilation, CO_2 accumulates in the alveoli and is not blown off. Respiratory acidosis occurs rapidly before renal compensation can occur. Mechanical ventilation may be necessary to support the patient until the initial cause of the hypoventilation can be corrected.

Ventilation-perfusion mismatch. Because the upper and lower airways do not play a part in gas exchange, the volume of inspired gas that fills these structures is referred to as physiologic dead space. This dead space is normally 25% to 30% of the inspired volume. A major mechanism for the elevation of $PaCO_2$ is an increase in the volume of dead space in relation to the entire tidal volume. Dead space increases when an area that is well ventilated has reduced perfusion and no longer participates in gas exchange.

Assessment

Respiratory assessment and evaluation of gas exchange are discussed in depth in Chapter 9. Assessment of the patient with ARF begins with the neurological system. Changes in mental status resulting from hypoxia and hypercapnia begin with anxiety, restlessness, and confusion and may progress to lethargy, severe somnolence, and coma.

The respiratory assessment continues with observing the rate, depth, and pattern of respiration. In response to hypoxemia, compensatory mechanisms produce tachypnea and an increase in tidal volume. As these compensatory mechanisms fail, respirations become shallow. A decrease in respiratory rate is an ominous sign. Use of accessory muscles and sternal retractions are a cause for concern as they indicate respiratory muscle fatigue. By auscultation, the nurse assesses the adequacy of airflow and the presence of adventitious breath sounds. The presence of a cough and the amount and characteristics of any sputum production are noted.

A thorough cardiac assessment provides information about the heart's ability to deliver O_2 to the tissues. The patient must be closely monitored for changes in blood pressure, heart rate, and cardiac rhythm. ARF initially causes tachycardia and increased blood pressure. As ARF progresses, it may lead to dysrhythmias, angina, bradycardia, hypotension, and cardiac arrest. The nurse should evaluate peripheral perfusion by assessing pulses for strength and bilateral equality. The skin is assessed for a decrease in temperature and the presence of cyanosis or pallor, which are additional indicators of poor perfusion.

The patient's nutritional status must be evaluated because this is an important factor in maintaining respiratory muscle strength. The nurse looks for recent weight loss, muscle wasting, nausea, vomiting, abdominal distention, and skin turgor quality.

It is important to assess the patient's psychosocial status. This includes identifying the patient's significant others and their role in the family structure. An understanding of the patient's educational level, socioeconomic background, spiritual beliefs, and cultural or ethnic practices is important in determining an educational plan for discharge and future self-care.

Serial chest x-rays and pulmonary function tests provide important assessment information. Laboratory studies that are essential for the patient with respiratory failure include the following: electrolytes, which determine adequate muscle function; hemoglobin and hematocrit to evaluate the blood's O_2 carrying capacity; and ABG measurements to assess gas exchange and acid-base balance. Noninvasive monitoring such as pulse oximetry (SpO_2) provides information about the patient's oxygenation, whereas continuous end-tidal CO_2 monitoring provides information about the patient's ventilation.

Effects of Aging

Many age-related factors increase the older adult's risk for developing ARF. Physiological changes may make identifying the signs and symptoms of ARF more difficult in the elderly. The most common early sign of hypoxemia in the elderly is a change in mental status, such as confusion or agitation. These changes are often mistaken for dementia or a normal sign of advancing age[21,46] (see box, "Geriatric Considerations").

GERIATRIC CONSIDERATIONS

PHYSIOLOGICAL CHANGES	NURSING IMPLICATIONS
Calcification of costal and sternal cartilage	Decreased chest wall mobility
Osteoporosis	Increased functional residual capacity and residual volume
Spinal degeneration	Decreased tidal volume, vital capacity, and forced expiratory volume
Kyphosis	Decreased chest wall mobility and restricted ventilation
Flattening of diaphragm	Increased work of breathing
Decline in muscle mass	Respiratory muscle fatigue
Diminished cough reflex	Ventilation/perfusion mismatch
Decreased mucociliary clearance	Early airway collapse
Decline in surfactant production	Increased risk of atelectasis and pneumonia
Decreased effectiveness of immune system	Increased susceptibility to infection
Thickening of alveolar-capillary membrane	Ventilation/perfusion mismatch
Decreased pulmonary blood flow	Ventilation/perfusion mismatch

Based on data from El Sohl AA, & Ramadan FH. Overview of respiratory failure in older adults. *Journal of Intensive Care Medicine.* 2006;21(6), 345-351; and Muir J, Lamia B, Molano C, & Cuvelier A. Respiratory failure in the elderly. *Seminars in Respiratory and Critical Care Medicine.* 2010;31(5), 634-646.

Because of age-related decreases in chemoreceptor and central nervous system function, older adults have a lower ventilatory response to hypoxia and hypercapnia. In addition, hypoxia in the elderly may not produce the same compensatory increases in heart rate, stroke volume, and cardiac output that are seen in younger adults. This may be due to preexisting

cardiac disease or the effects of cardiac medications such as digoxin or beta-blockers. Increasing age can also lead to a slower response to O_2 therapy, making early identification and treatment of hypoxia essential in this population. Finally, normal PaO_2 levels decrease with age, but aging does not produce alterations in $PaCO_2$. For this reason hypercapnia and a falling pH are causes for concern.

Interventions

The goals of treating patients with ARF are fivefold and include (1) maintaining a patent airway, (2) optimizing O_2 delivery, (3) minimizing O_2 demand, (4) treating the cause of ARF, and (5) preventing complications.

Maintaining a Patent Airway

Some causes of acute respiratory failure such as COPD, cardiogenic pulmonary edema, pulmonary infiltrates in immunocompromised patients, and palliation in the terminally ill may be effectively treated with noninvasive positive-pressure ventilation (NPPV).[49] However, if a patient is unable to maintain a patent airway, intubation and mechanical ventilation may be required for treatment. (Refer to Chapter 9 for nursing care related to NPPV and mechanical ventilation.)

Optimizing O_2 Delivery

Optimizing O_2 delivery can be achieved in many ways, depending on the needs of the patient. The first is to provide supplemental O_2 via nasal cannula or face mask to maintain the PaO_2 above 60 mm Hg or the SaO_2 above 90%.[4] Higher PaO_2 values are indicated in cases of severe tissue hypoxia, low flow states, or deficiencies in O_2 carrying capacity.[3] If supplemental O_2 is ineffective in raising PaO_2 levels, noninvasive or invasive mechanical ventilation is indicated (see box, "Clinical Alert: Acute Respiratory Failure"). Patients are positioned for comfort and to enhance $\dot{V}/\dot{Q}$ matching. Some patients who are alert and dyspneic are able to oxygenate more effectively in the semi-Fowler to high Fowler position. Patients with unilateral lung disease should be positioned on their side with the better functioning "good" lung down. This allows gravity to perfuse the lung that has the best ventilation. Other methods to optimize O_2 delivery include red blood cell transfusion to ensure adequate hemoglobin levels to transport O_2, and enhancing cardiac output and blood pressure to deliver sufficient O_2 to the tissues.

Minimizing O_2 Demand

Decreasing the patient's O_2 demand begins with providing adequate rest. Unnecessary physical activity is avoided in the patient with ARF. Agitation, restlessness, fever, sepsis, and patient-ventilator dyssynchrony must be addressed because they all contribute to increased O_2 demand and consumption.

! CLINICAL ALERT

Acute Respiratory Failure

CONCERN	SYMPTOMS	NURSING ACTIONS
Respiratory muscle fatigue	Diaphoresis Nasal flaring Tachycardia Abdominal paradox Muscle retractions Intercostal Suprasternal Supraclavicular Central cyanosis	**Improve O_2 delivery:** Administer O_2 Ensure adequate cardiac output and blood pressure Correct low hemoglobin Administer bronchodilators **Decrease O_2 demand:** Provide rest Reduce fever Relieve pain and anxiety Decrease work of breathing Position patient for optimum gas exchange and perfusion Prepare for possible intubation and mechanical ventilation
Cerebral hypoxia and carbon dioxide narcosis from increased CO_2 retention	Lethargy Somnolence Coma Respiratory acidosis	Maintain airway patency Prepare for possible intubation and mechanical ventilation

Treating the Cause of ARF

While the patient's hypoxia is being treated, efforts must be made to identify and reverse the cause of the ARF. Specific interventions for acute respiratory distress syndrome (ARDS), COPD, asthma, pneumonia, and pulmonary embolism are detailed later in this chapter.

Preventing Complications

Finally, the critical care nurse must be alert to the potential complications that the patient with ARF may encounter. Preventive measures must be taken to prevent the complications of immobility, adverse effects from medications, fluid and electrolyte imbalances, development of gastric ulcers, and the hazards of mechanical ventilation.

Nursing Diagnoses

Several nursing diagnoses must be considered in the care of a patient with ARF and are discussed in the "Nursing Care Plan for a Patient with Acute Respiratory Failure." Expected outcomes include adequate organ and tissue oxygenation, and effective breathing and adequate gas exchange.

NURSING CARE PLAN

for a Patient with Acute Respiratory Failure*

NURSING DIAGNOSIS

Impaired Spontaneous Ventilation related to hypoventilation, respiratory muscle fatigue, bronchospasm, infection, inflammation, central nervous system depression

PATIENT OUTCOMES

Adequate ventilation

- Ventilatory demand decreased
- Respiratory distress absent
- Respirations unlabored at a rate of 12 to 16 breaths per minute
- Arterial blood gases WNL

NURSING INTERVENTIONS	RATIONALES
• Assess respiratory status every 1 to 2 hours, including breath sounds, breathing pattern, rate, depth, and rhythm respirations	• Assess for respiratory distress; changes in breath sounds may indicate fluid in the airways (crackles), accumulation of mucus (rhonchi), or airway obstruction (wheezes)
• Monitor for dyspnea and signs of respiratory distress	• Indicate worsening of condition
• Assess for restlessness or change in level of consciousness	• Assess for signs of hypoxemia
• Position patient in semi-Fowler position (45 degrees) or position in which breathing pattern is most comfortable	• Promote maximal air exchange and lung expansion
• If patient has lung pathology, position for maximal gas exchange; place the "good" lung down	• Increase perfusion to the good lung and facilitate gas exchange
• Assist with activities; provide patient with periods of rest	• Reduce oxygen consumption and demands
• Administer medications to increase airflow as prescribed; evaluate their effectiveness	• Decrease airway resistance secondary to bronchoconstriction
• Give oxygen therapy or maintain mechanical ventilation	• Correct hypoxemia
• Monitor ABGs	• Assess for worsening hypoxemia and/or increasing $PaCO_2$; assess response to treatments
• If patient is mechanically ventilated, sedate according to goals for patient; avoid oversedation	• Facilitate gas exchange and mechanical ventilation; oversedation prolongs time on mechanical ventilation and its associated risks

NURSING DIAGNOSIS

Risk for Ineffective Airway Clearance related to inability to cough, presence of endotracheal tube, thick secretions, fatigue.

PATIENT OUTCOMES

Effective airway clearance

- Airway clear of secretions
- Lung sounds clear

NURSING INTERVENTIONS	RATIONALES
• Assess lung sounds	• Rhonchi may be audible with accumulation of secretions
• Change patient's position every 2 hours	• Mobilize secretions
• Encourage patient to cough and deep breathe	• Improve lung capacity and facilitate gas exchange
• Suction (nasotracheal or endotracheal) as determined by patient assessment	• "As needed" suctioning prevents damage to the airway from the suctioning procedure
• Provide adequate humidification with supplemental oxygen or mechanical ventilation	• Prevent drying of secretions and facilitate secretion removal
• Assess amount, color, consistency of secretions	• Indicate need for humidification and/or signs of infection

*Please see Chapter 9 for Nursing Care Plan for the Mechanically Ventilated Patient.

Continued

NURSING CARE PLAN

for a Patient with Acute Respiratory Failure—cont'd

NURSING DIAGNOSIS

Risk for Infection related to underlying illness/disease process, endotracheal intubation

PATIENT OUTCOMES

Absence of infection

- Normal temperature
- White blood cell count WNL
- Chest x-ray normal
- Negative cultures of sputum and bronchial aspirates

NURSING INTERVENTIONS	RATIONALES
• Monitor temperature every 4 hours, more frequently if elevated	• Fever may be first sign of infection
• Monitor white blood cell count	• Rising count indicates body's response to combat pathogens
• Assess amount, color, consistency of secretions	• Assess for infection
• Monitor results of cultures of sputum and/or bronchial specimens	• Assess need for antibiotic and appropriate antibiotic coverage
• Elevate the head of bed to at least 30 degrees	• Reduce the risk of aspiration and ventilator-associated pneumonia
• Provide oral care every 2 to 4 hours and as needed; brush teeth every 12 hours; consider chlorhexidine gluconate (0.12%) oral rinse every 12 hours	• Reduce bacterial growth and colonization of oropharyngeal secretions; promotes patient comfort

NURSING DIAGNOSIS

Anxiety related to inability to speak, situational crises, uncertainty, fear of death, and lack of control

PATIENT OUTCOMES

Anxiety decreased or absent

- Vital signs WNL
- Relaxed facial expression and body movements, and normal sleep patterns
- Usual perceptual ability and interactions with others

NURSING INTERVENTIONS	RATIONALES
• Monitor for signs of anxiety: increased heart rate, blood pressure, respiratory rate, muscle tension, inappropriate behaviors	• Anxiety is highly individualized response to life events; signs must be recognized to provide interventions
• Develop trusting relationship by using calm, consistent, and reliable behaviors	• Encourage communication and enhance feelings of safety
• Always introduce yourself and all unfamiliar persons to the patient and explain why they are there	• Uncertainty and lack of predictability contribute to feelings of anxiety
• Provide nurturing environment; allow the patient some control over decision making	• Increase sense of independence and normality
• Provide a means of communication (e.g., nonverbal, yes/no, picture charts, pencil/paper)	• Assist in meeting patient's needs and reduce anxiety
• Teach relaxation techniques (e.g., the use of slow rhythmic breathing during stressful periods)	• Enhance coping and improve physiological response
• Reassure patient of staff member's presence and prompt interventions as needed	• Assist in meeting needs and reducing anxiety
• Allow family member to remain at bedside to decrease isolation	• Provide a sense of security and familiarity; facilitate communication

NURSING CARE PLAN

for a Patient with Acute Respiratory Failure—cont'd

NURSING DIAGNOSIS
Risk for Impaired Skin Integrity related to bed rest and altered metabolic state

PATIENT OUTCOMES
Skin intact

NURSING INTERVENTIONS	RATIONALES
• Assess skin every shift for areas of breakdown	• Identify problems and promote preventive interventions
• Keep patient's skin clean and dry	• Decrease the risk of skin breakdown
• Reposition every 2 hours; if unable to manually turn patient because of hemodynamic instability, consider continuous lateral rotation or kinetic therapy with pressure relief mattress with continued skin assessment every 2 hours	• Reduce pressure on bony prominences

NURSING DIAGNOSIS
Risk for Ineffective Family Coping related to knowledge deficits of family members

PATIENT OUTCOMES
Family integrity maintained
- Family members verbalize educational needs and fears
- Family members feel comfortable asking questions related to patient's prognosis

NURSING INTERVENTIONS	RATIONALES
• Assess family unit and coping behaviors	• Allow for anticipatory care and guidance to help family unit maintain support and coping strategies
• Assist family to identify roles to maintain family integrity	• Positive feedback from one family member can reinforce a behavior of another member
• Assist family members to verbalize fears and distress	• Promote effective communication
• Answer questions; explain procedures, equipment, changes in patient's condition, and outcomes to family members in a sensitive manner	• Establish a trusting relationship; reduce anxiety
• Inform family of resources available to them such as chaplain and psychiatric liaison	• Enhance the use of services that may assist family
• Initiate multiprofessional conferences with family to provide information and make decisions regarding ongoing treatment	• Establish trust with all health care team and encourages compliance with treatments

ABGs, Arterial blood gases; *HOB,* head of bed; *$PaCO_2$,* partial pressure of carbon dioxide in arterial blood; *WNL,* within normal limits.
Based on data from Gulanick M and Myers JL. *Nursing Care Plans: Diagnoses, Interventions, and Outcomes,* 7th ed. St. Louis: Mosby, 2011.

RESPIRATORY FAILURE IN ACUTE RESPIRATORY DISTRESS SYNDROME

Definition

ARDS was originally described in 1967 as an acute illness manifested by dyspnea, tachypnea, decreased lung compliance, and diffuse alveolar infiltrates on chest x-ray studies. The syndrome was observed in young adult patients after trauma who developed shock, required excessive fluid administration, or both. Autopsy results revealed that pathological heart and lung findings were similar to those described in infant respiratory distress syndrome.

In 1994, the American-European Consensus Conference recommended a definition of ARDS as a subset of acute lung injury. The definition included three criteria: PaO_2/FiO_2 ratio less than 200, bilateral infiltrates on chest x-ray, and pulmonary artery occlusion pressure (PAOP) less than 18 mm Hg or no clinical evidence of left atrial hypertension.[27]

The 1994 definition of ARDS was revised in 2012 by a multi-society consensus panel, and is termed the Berlin definition.[65a] Criteria for ARDS include 1) acute onset within one week of clinical insult; 2) bilateral pulmonary opacities not explained by other conditions; and 3) altered PaO_2/FiO_2 ratio. The PAOP requirement was removed from the definition. Severity is determined by the PaO_2/FiO_2 ratio, and PEEP or CPAP requirements of $\geq$ 5 cm H_2O: 1) Mild ARDS—201-300 mm Hg; 2) Moderate—101-200 mm Hg; and 3) Severe—$\leq$ 100 mm Hg.

Etiology

Several possible causes of ARDS are listed in Box 14-1, and are categorized into direct and indirect factors. However,

BOX 14-1 POSSIBLE CAUSES FOR ACUTE RESPIRATORY DISTRESS SYNDROME

Direct Causes
- Aspiration of gastric contents
- Diffuse pneumonia
- Fat embolism
- Near-drowning
- Neurogenic pulmonary edema
- Oxygen toxicity
- Pulmonary contusion
- Multisystem trauma (chest and/or lung injury)
- Radiation (chest)

Indirect Causes
- Sepsis
- Multisystem trauma (without chest and/or lung injury)
- Cardiopulmonary bypass
- Anaphylaxis
- Disseminated intravascular coagulation
- Drug overdose
- Eclampsia
- Fractures, especially of the pelvis or long bones
- Leukemia
- Transfusion-related acute lung injury
- Pancreatitis
- Thrombotic thrombocytopenic purpura

certain risk factors have a higher associated frequency of ARDS, and the presence of two or more factors increases the risk. The most common risk factors or disease processes associated with ARDS are sepsis, pneumonia, trauma, and aspiration of gastric contents. These four risk factors are believed to account for approximately 85% of all ARDS cases, with sepsis being the most common cause. Approximately one third of hospitalized patients who aspirate gastric contents develop ARDS. In addition, critically ill patients with a history of chronic alcoholism are at an increased risk of developing ARDS. Other causes with significant incidences are multiple transfusions including fresh frozen plasma and platelets, fat embolism, ischemia reperfusion, and pancreatitis.[27] Acute lung injury (mild ARDS with Berlin definition) is the most common cause of mortality related to transfusions with an incidence of about 1 for every 5000 transfusions, and a mortality of 6% to 23%. The syndrome has been named TRALI (transfusion-related acute lung injury) with defined criteria.[38]

The mortality rate for patients with diagnosed ARDS has been improving over the last decade. The 28-day mortality rate is reported to be 25% to 30%.[71] In the Berlin definition, mortality is estimated based on ARDS severity: mild, 27%; moderate, 32%; and severe, 45%.[65a] As more individuals survive ARDS, prevention of long-term disabilities must be a priority of care. One study of patients who survived ARDS revealed that although lung volume and pulmonary function were normal by 6 months, functional disability persisted 1 year after discharge.[29] Another study reported that nearly half of ARDS survivors had significant neurocognitive impairment and a decrease in quality of life that persisted for at least 2 years.[32]

Pathophysiology

ARDS is characterized by acute and diffuse injury to the lungs, leading to respiratory failure. It is a two-phase condition including the acute exudation response phase and the late phase of fibroproliferation. The acute response is a systemic inflammatory reaction secondary to direct or indirect lung injury. Initial injury causes damage to the pulmonary capillary endothelium, which activates massive aggregation of platelets and formation of intravascular thrombi. The platelets release serotonin and a substance that activates neutrophils. Other inflammatory factors such as endotoxin, tumor necrosis factor, and interleukin-1 are also activated. Neutrophil activation causes release of inflammatory mediators such as proteolytic enzymes, toxic O_2 products, arachidonic acid metabolites, and platelet-activating factors. The release of these mediators damages the alveolar-capillary membrane, which leads to increased capillary membrane permeability. Fluids, protein, and blood cells leak from the capillary beds into the alveoli, resulting in pulmonary edema. Pulmonary hypertension occurs secondary to vasoconstriction caused by the inflammatory mediators. The pulmonary hypertension and pulmonary edema lead to $\dot{V}/\dot{Q}$ mismatching. The production of surfactant is stopped, and the surfactant present is inactivated.[7,27]

During the acute phase of ARDS, damage to the alveolar epithelium and vascular endothelium occurs. The damaged cells become susceptible to bacterial infection and pneumonia. The lungs become less compliant, resulting in decreased ventilation. A right-to-left shunt of pulmonary blood develops, and hypoxemia refractory to O_2 supplementation becomes profound. The work of breathing increases.[7]

The late phase of ARDS is the fibroproliferation stage. As ARDS proceeds over time (greater than 24 to 48 hours), a fibrin matrix (hyaline membrane) forms. After approximately 7 days, fibrosis obliterates the alveoli, bronchioles, and interstitium. The lungs become fibrotic with decreased functional residual capacity and severe right-to-left shunting. The inflammation and edema become worse with narrowing of the airways. Resistance to airflow and atelectasis increase.

The inflammatory mediators responsible for lung damage also cause harm to other organs in the body, often resulting in multiple organ dysfunction syndrome. The pathophysiology of ARDS is outlined in Figure 14-2.

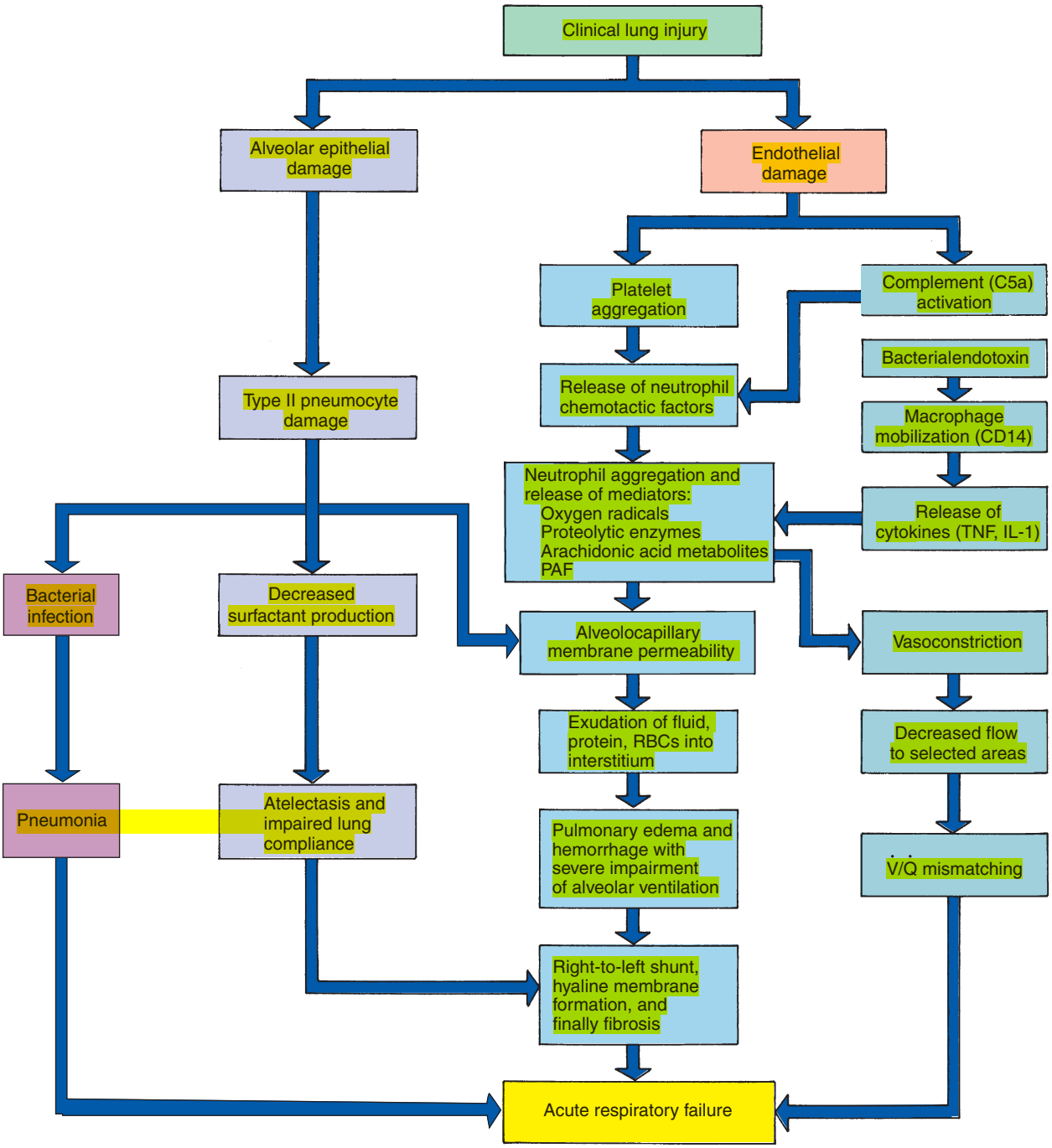

FIGURE 14-2 Pathogenesis of acute respiratory distress syndrome (ARDS). *TNF,* Tumor necrosis factor; *IL-1,* interleukin-1; *PAF,* platelet-activating factor; *RBCs,* red blood cells. (From McCance KL, Huether SE. *Pathophysiology: The Biologic Basis for Diseases in Adults and Children*. 6th ed. St. Louis: Mosby; 2010.)

Assessment

Assessment of a patient with ARDS is collaborative. A key clinical finding that is often diagnostic of ARDS is a lung insult (direct or indirect) followed by respiratory distress with dyspnea, tachypnea, and hypoxemia that does not respond to O_2 therapy and PEEP. Initial signs of ARDS include restlessness, disorientation, and change in the level of consciousness. Pulse and temperature may be increased. Chest x-ray studies are usually normal in the initial stage.

As ARDS progresses and the PaO_2 decreases, dyspnea becomes severe. Intercostal and suprasternal retractions are often present. Other signs may include tachycardia and central cyanosis. The $PaCO_2$ continues to decrease, resulting in respiratory alkalosis. Hypocapnia and hypoxemia do not respond to increasing levels of supplemental O_2. Patients developing ARDS frequently need their noninvasive supplemental O_2 increased until it is at the maximum level, with little effect on the PaO_2. Metabolic acidosis caused by lactic acid buildup often results, and is confirmed by serum lactate level determinations. The metabolic imbalances are a result of a low $\dot{V}/\dot{Q}$ ratio and a deteriorating PaO_2/FiO_2 ratio. As the ARDS progresses, crackles, rhonchi, and bronchial breath sounds are audible as fluid moves into the airways. Initially, the chest x-ray shows bilateral patchy infiltrates that have a "ground glass appearance." As ARDS worsens, the chest x-ray shows complete opacity, sometimes referred to as a "whiteout." The cardiac silhouette is normal.[62]

Pulmonary mechanics show a decrease in lung volume, especially functional residual capacity, and a decrease in static and dynamic compliance. Peak inspiratory pressures rise, indicating a decrease in compliance.

Once ARDS is diagnosed, important assessment data that are used to guide treatment include hemodynamic measurements, ABGs, mixed venous blood gases, breath sounds, serial chest x-ray studies, computerized tomography (CT), complete blood cell count with differential, blood and sputum cultures, and fluid and electrolyte values. Metabolic and nutritional needs, and psychosocial needs of the patient and family, must also be assessed.

Interventions

Achieving adequate oxygenation is the primary goal in the treatment of ARDS. Other treatments are primarily supportive.

Oxygenation

Patients with ARDS generally require intubation and mechanical ventilation. Selection of ventilator settings is based on lung-protective strategies that attempt to achieve adequate oxygenation while minimizing the risks of ventilator-associated complications. Lung-protective strategies consist of low tidal volume (V_T), low end-inspiratory plateau pressure, FiO_2 at nontoxic levels (less than 0.60), and positive end-expiratory pressure (PEEP) (Table 14-1). Large clinical studies have shown reduced mortality and complications with the use of low V_T. The target V_T recommended is 6 mL/kg of predicted ideal body weight (calculated from sex and height) (see Table 14-1). Actual body weight should not be used. The body weight may change secondary to accumulation of body fluid, but the size of the lungs does not change. The V_T may be reduced to 4 to 5 mL/kg to maintain the end-inspiratory plateau pressure at 30 cm H_2O or less. These lower volumes and plateau pressures prevent the alveoli from overdistending and minimize shearing. The respiratory acidosis that occurs secondary to the low V_Ts can be controlled by increasing the ventilator respiratory rate in a stepwise manner generally to an upper limit of 35 breaths per minute. The $PaCO_2$ should be kept within a permissive hypercapnia range of 50 to 70 mm Hg, and the pH maintained between 7.30 and 7.45[3,22,27] (see box, "Evidence-Based Practice").

EVIDENCE-BASED PRACTICE

Mechanical Ventilation in ARDS

Problem

Patients with acute respiratory distress syndrome (ARDS) require mechanical ventilation and other support to ensure adequate oxygenation and ventilation.

Clinical Question

What are the best practices for mechanical ventilation for patients with ARDS?

Evidence

In this systematic review and meta-analysis, the authors analyzed three trials with data from 2299 patients. They compared outcomes of higher (greater than 12 cm H_2O) versus lower (traditional 5 to 12 cm H_2O) of positive end-expiratory pressure (PEEP) in patients with acute lung injury or ARDS. They concluded that mechanical ventilation with higher levels of PEEP significantly improved survival in patients with ARDS only. Patients with acute lung injury did not show improvement and high PEEP levels may actually be harmful in this population.

Implications for Nursing

Nurses must collaborate with respiratory therapists and the intensivists in determining the best management of patients with ARDS. Protocols for ventilator management that include low tidal volumes may assist in implementation of practices that improve patient outcomes. With the revised Berlin definition of ARDS, new management protocols may be developed specific to the three levels of severity. Some providers are concerned that ventilation with low tidal volumes is associated with patient discomfort, tachypnea, and hypercapnia. The nurse can assist by assessing the patient regularly for these potential outcomes of low–tidal volume ventilation and provide appropriate sedation.

Level of Evidence

A—Meta-analysis

Reference

Briel M, et al. Higher vs lower positive end-expiratory pressure in patients with acute lung injury and acute respiratory distress syndrome. *Journal of the American Medical Association*. 2010;302, 865-873.

TABLE 14-1 MECHANICAL VENTILATION PROTOCOL FOR ACUTE RESPIRATORY DISTRESS SYNDROME (NHLBI, NIH)

Definition:

1. Acute onset
2. PaO_2/FiO_2 ≤300 mm Hg, ALI (Berlin definition mild ARDS); PaO_2/FiO_2 ≤200, ARDS (Berlin definition moderate to severe ARDS) (referred to as P/F ratio)
3. Bilateral (patchy, diffuse, or homogeneous) infiltrates consistent with pulmonary edema
4. No clinical evidence of left atrial hypertension

Ventilator Setup and Adjustment:

1. Calculate predicted body weight (PBW)
   Males = 50.0 + 2.3 (height [inches] − 60)
   Females = 45.5 + 2.3 (height [inches] − 60)
2. Select any ventilator mode
3. Set initial V_T to 8 mL/kg PBW
4. Reduce V_T by 1 mL/kg at intervals ≤2 hours until V_T = 6 mL/kg PBW
5. Set initial rate to approximate baseline VE (not >35 breaths/min)
6. Adjust V_T and RR to achieve pH and plateau pressure goals

Oxygenation Goal: PaO_2 55-80 mm Hg or SpO_2 88%-95%

Lower PEEP/higher FiO_2 Recommendations:

FiO_2	0.3	0.4	0.4	0.5	0.5	0.6	0.7	0.7
PEEP cm H_2O	5	5	8	8	10	10	10	12
FiO_2	0.7	0.8	0.9	0.9	0.9	1.0		
PEEP cm H_2O	14	14	14	16	18	18-24		

Higher PEEP/lower FiO_2 Recommendations:

FiO_2	0.3	0.3	0.3	0.3	0.3	0.4	0.4	0.5
PEEP	5	8	10	12	14	14	16	16
FiO_2	0.5	0.5-0.8	0.8	0.9	1.0	1.0		
PEEP	18	20	22	22	22	24		

Plateau Pressure Goal: ≤30 cm H_2O

Check Pplat, at least every 4 hours and after each change in PEEP or V_T

1. **If Pplat >30 cm H_2O:** decrease V_T by 1-mL/kg steps (minimum, 4 mL/kg)
2. **If Pplat <25 cm H_2O:** V_T <6 mL/kg, increase V_T by 1 mL/kg until Pplat >25 cm H_2O or V_T = 6 mL/kg
3. **If Pplat <30 cm H_2O and breath stacking occurs:** may increase V_T in 1-mL/kg increments (maximum, 8 mL/kg)

pH Goal: 7.30-7.45

Acidosis Management: (pH <7.30)

1. **If pH 7.15-7.30:** Increase RR until pH >7.30 or $PaCO_2$ <25 mm Hg (maximum RR, 35 breaths/min)
2. **If pH <7.15:** Increase RR to 35 breaths/min; V_T may be increased in 1-mL/kg steps until pH >7.15 (Pplat target may be exceeded).

Alkalosis Management: (pH >7.45)

1. Decrease ventilator breaths/min rate if possible

I:E Ratio Goal: Duration of inspiration ≤ duration of expiration

ALI, Acute lung injury; *ARDS,* acute respiratory distress syndrome; *FiO_2,* fraction of inspired oxygen; *H_2O,* water; *I:E,* inspiration-to-expiration ratio; *$NaHCO_3$,* sodium bicarbonate; *NHLBI,* National Heart, Lung, and Blood Institute; *NIH,* National Institutes of Health; *PaO_2,* partial pressure of oxygen in arterial blood; *$PaCO_2$,* partial pressure of carbon dioxide in arterial blood; *Pplat*, peak plateau pressure; *PEEP,* positive end-expiratory pressure; *P/F,* PaO_2/FiO_2 (oxygenation index); *RR,* respiratory rate; *SaO_2,* arterial oxygen saturation; *SpO_2,* arterial oxygen saturation via pulse oximeter; *VT*, tidal volume; *VE,* minute ventilation.

Adapted from the NIH NHLBI *ARDS Clinical Network Mechanical Ventilation Protocol Summary.* Retrieved on May 27, 2012, from http://www.ardsnet.org/system/files/Ventilator%20Protocol%20Card.pdf.

Patients with ARDS require significant support to achieve and maintain arterial oxygenation. High levels of FiO_2 may be required for short periods while aggressively working to reduce the FiO_2 to the lowest level that maintains the PaO_2 above 60 mm Hg. To prevent O_2 toxicity, the goal is to maintain the PaO_2 with levels of FiO_2 at 0.60 or below.

Ventilatory support typically includes PEEP to restore functional residual capacity, open collapsed alveoli, prevent collapse of unstable alveoli, and improve arterial oxygenation.[22] The National Heart, Lung, and Blood Institute ARDS network developed a protocol for PEEP application based on amount of FiO_2 requirements (see Table 14-1). Studies have shown that higher PEEP resulted in improved survival without increase in the incidence of pneumothorax in ARDS patients only; decreased mortality was not seen in patients with acute lung injury.[8]

When using high levels of PEEP, the nurse must assess for potential adverse effects. PEEP increases intrathoracic pressure, potentially leading to decreased cardiac output. Excessive pressure in stiff lungs increases peak inspiratory and plateau pressures, which may result in barotrauma and pneumothorax. Treatment of a pneumothorax requires prompt insertion of a chest tube. A patient receiving high levels of PEEP therapy should be monitored every 2 to 4 hours, and after every adjustment in the PEEP setting, for changes in respiratory status such as increased respiratory rate, worsening adventitious breath sounds, decreased or absent breath sounds, decreased SpO_2, and increasing dyspnea.

A few unconventional modes of mechanical ventilation are used to treat ARDS when patients are unable to be oxygenated with standard modes of ventilation. These modes include high-frequency oscillatory ventilation; pressure-controlled, inverse-ratio ventilation; and airway pressure release ventilation. These modes often improve alveolar ventilation and arterial oxygenation while decreasing the risk of lung injury. None have been successful enough to be considered standard therapy (see Chapter 9).

Sedation and Comfort

Patients with ARDS routinely receive sedation to promote comfort and sleep/rest, alleviate anxiety, prevent self-extubation or harm, and ensure adequate ventilation. A major adverse effect of undersedation is breathing dyssynchrony between the patient and ventilator. Ventilator dyssynchrony causes inadequate gas exchange and increases the patient's risk for ventilator-induced lung injury.[24,44]

Oversedation can also lead to long-term sequelae such as delirium. The amount of sedation used must be monitored carefully to achieve predetermined end points or goals (see Chapter 5). Sedation goals are based on the patient's response to therapy and are determined through a collaborative effort between the physician, clinical pharmacist, and the critical care nurse. Regular assessment and documentation of response to therapy with a validated sedation assessment scale along with a validated delirium assessment scale are essential.[34,58]

Therapeutic paralysis with a neuromuscular blocking agent may be required to completely control ventilation and promote adequate oxygenation. Patients who require unconventional modes of mechanical ventilation often need neuromuscular blockade because these modes are uncomfortable for the patient and provide an unnatural means of respiration.[10] Use of neuromuscular blocking agents require careful consideration and monitoring because of the increased risk of prolonged myopathy (see Chapter 5). However, a recent article showed that early administration of neuromuscular blocking agents in patients with severe ARDS improved survival, increased the time off the ventilator, and did not increase muscle weakness in this subset population.[55]

Prone Positioning

Patients with ARDS who do not respond to standard treatment may benefit from prone positioning. Turning the patient to the prone position *(proning)* alters the $\dot{V}/\dot{Q}$ ratio by shifting perfusion from the posterior bases of the lung to the anterior portion with improved ventilation. Proning also removes the weight of the heart and abdomen from the lungs, facilitates removal of secretions, improves oxygenation, and enhances recruitment of airways.[63,67] Proning should be considered when the PaO_2/FiO_2 ratio falls below 100, other lung recruitment strategies have been maximized, and/or the pulmonary status continues to deteriorate. Once turned to the prone position, the optimal duration of therapy is up to 24 hours daily, with therapy continuing until the improvement in oxygenation is maximized.[4,67]

Turning the patient to the prone position is a cumbersome procedure requiring involvement of several healthcare professionals to ensure the patient's safety. Care must be taken to prevent dislodging the ETT and other tubes and lines. Several commercial devices are available to assist in turning the patient such as the Vollman Prone Positioner (Hill-Rom Services Corp.) and specialized proning beds.

Potential complications from the prone position are gastric aspiration, peripheral nerve injury, pressure ulcers, corneal ulceration, and facial edema. Gastric tube feedings are turned off for 1 hour or aspirated before turning the patient to reduce the risk of aspiration. Proper body alignment must be maintained while the patient is in the prone position to decrease the risk of nerve damage. Pillows and foam support equipment are used to prevent overextension or flexion of the spine and reduce weight-bearing on bony prominences. Protective pads are used at the shoulders, iliac crest, and knees to decrease alterations in skin integrity and peripheral nerve damage. To avoid peripheral nerve injury and contractures of the shoulders, the arms are positioned carefully and repositioned often. A moisture barrier is applied to the patient's entire face to protect the skin from the massive amount of drainage from the mouth and nose. Absorbent pads, an emesis basin, or both, can be placed to capture the excessive oral and nasal drainage. The eyes must be protected to prevent direct ocular pressure caused by facial edema. The eyes are lubricated and taped shut to prevent corneal drying and abrasions.[67]

Fluids and Electrolytes

Conservative fluid management, including diureis, is the goal for patients with ARDS, resulting in reduced mortality, improved lung function, shorter length of mechanical ventilation, and fewer critical care unit days.[27,59] Patients who are hypotensive or hypovolemic should however, receive aggressive fluid resuscitation until the condition has resolved. The use of colloids along with diuretics has been shown to be effective in hypoproteinemic patients only.[28,69]

Nutrition

The goal of nutritional support is to provide adequate nutrition to meet the patient's level of metabolism and reduce morbidity[17] (see Chapter 6). Several studies have evaluated the effects of a specialized enteral nutritional formula enriched with eicosapentaenoic acid (EPA), gamma-linolenic acid (GLA), and elevated antioxidants (EPA/GLA) in the treatment of ARDS. The studies have demonstrated reduced mortality, improved oxygenation secondary to reduced pulmonary inflammation, and fewer days of mechanical ventilation.[56]

Pharmacological Treatment

Despite clinical studies of various medications, no pharmacological agents are considered standard therapy for ARDS. Furosemide with albumin is advocated when the patient's protein level is low. The combination has resulted in improved oxygenation and reduced time receiving mechanical ventilation. Corticosteroids administration should be considered in patients with severe ARDS and before day 14.[59,64] Studies with inhaled nitric oxide have not shown improvements in survival or duration of mechanical ventilation; however, it is used as rescue therapy for severe refractory hypoxemia. The early use of cisatracurium, a neuromuscular blocking agent, during the first 48 hours in patients with severe ARDS may improve some some outcomes.[55] Several new and different drugs are being studied, including statins and granulocyte-macrophage colony-stimulating factor.[59]

Psychosocial Support

The onset of ARDS and its long recovery phase result in stress and anxiety for both the patient and the family. The patient may also experience feelings of isolation and dependence because of the length of the recovery phase. Healthcare team members must always remember to provide a warm, nurturing environment in which the patient and family can feel safe. A therapeutic environment includes taking the time to explain procedures, equipment, changes in the patient's condition, and outcomes to the patient and family members. Allowing the patient to participate in the planning of care and to verbalize fears and questions may help reduce stress and anxiety. In the intubated patient, communication is impaired, which increases the patient's sense of isolation. The isolation and accompanying depression can be minimized by encouraging a family member to stay with the patient and displaying personal items from home, such as photographs of loved ones.

ACUTE RESPIRATORY FAILURE IN CHRONIC OBSTRUCTIVE PULMONARY DISEASE

Pathophysiology

COPD is a progressive disease characterized by airflow limitations that are not fully reversible. These airflow limitations are associated with an abnormal inflammatory response to noxious particles or gases.[47] COPD is characterized by a mixture of small airway disease (obstructive bronchiolitis) and parenchymal destruction (emphysema). COPD is a preventable disease with treatment goals of decreasing symptoms and reducing exacerbations. Its incidence and impact on chronic morbidity and mortality are increasing. COPD is the fourth leading cause of death in the United States after cardiac disease, cancer, and stroke. The primary cause of COPD is tobacco smoke, and smoking cessation is the most effective intervention to reduce the risk of developing COPD and stop disease progression.[47] Other contributing factors to the development of COPD include air pollution, occupational exposure to dust or chemicals, and the genetic abnormality alpha$_1$-antitrypsin deficiency.[20]

The primary pathogenic mechanism in COPD is chronic inflammation, which may be both direct injury to the airway and also lead to systemic effects.[53] Exposure to inhaled particles leads to airway inflammation and injury. The body repairs this injury through the process of airway remodeling, which causes scarring, narrowing, and obstruction of the airways. Destruction of alveolar walls and connective tissue results in permanent enlargement of air spaces. Increased mucus production results from enlargement of mucus-secreting glands and an increase in the number of goblet cells. Areas of cilia are destroyed, contributing to the patient's inability to clear thick, tenacious mucus. Structural changes in the pulmonary capillaries thicken the vascular walls and inhibit gas exchange. Systemic inflammation also causes direct effects on peripheral blood vessels and may be a concomitant factor in the association of cardiovascular disease in these patients. Table 14-2 outlines the physiological changes that result from COPD.

ARF can occur at any time in the patient with COPD. These patients normally have little respiratory reserve, and any condition that increases the work of breathing worsens $\dot{V}/\dot{Q}$ mismatching. Common causes of ARF in patients with COPD are acute exacerbations, heart failure, dysrhythmias, pulmonary edema, pneumonia, dehydration, and electrolyte imbalances.

Assessment

The hallmark symptoms of COPD are dyspnea, chronic cough, and sputum production. The diagnosis is confirmed by postbronchodilator spirometry that documents irreversible airflow limitations.[47] These pulmonary function tests show an increase in total lung capacity and a reduction in forced expiratory volume over 1 second (FEV_1). Functional residual capacity is increased as a result of air trapping.

TABLE 14-2 PATHOLOGICAL AND PHYSIOLOGICAL CHANGES IN CHRONIC OBSTRUCTIVE PULMONARY DISEASE

PATHOLOGICAL CHANGES	PHYSIOLOGICAL CHANGES
Mucus hypersecretion	Sputum production
Ciliary dysfunction	Retained secretions Chronic cough
Chronic airway inflammation	Expiratory airflow limitation
Airway remodeling	Terminal airway collapse Air trapping Lung hyperinflation
Thickening of pulmonary vessels	Poor gas exchange with hypoxemia and hypercapnia Pulmonary hypertension Cor pulmonale (right ventricular enlargement and heart failure)

By the time the characteristic physical findings of COPD are evident on physical examination, a significant decline in lung function has occurred. The chest will be overexpanded, or barrel-shaped, because the anteroposterior diameter increases in size. Respiration may include the use of accessory muscles and pursed-lip breathing. Clubbing of the fingers indicates long-term hypoxemia. Lung auscultation usually reveals diminished breath sounds, prolonged exhalation, wheezing, and crackles. ABG results show mild hypoxemia in the early stages of the disease, and worsening hypoxemia and hypercapnia as the disease progresses. Over time, as a compensatory mechanism, the kidneys increase bicarbonate production and retention (metabolic alkalosis) in an attempt to keep the pH within normal limits.

Exacerbations of COPD often result in a change in the patient's baseline dyspnea and an increase in sputum volume. Changes in the character of the sputum may signal the development of a respiratory infection. Additional symptoms may include anxiety, wheezing, chest tightness, tachypnea, tachycardia, fatigue, malaise, confusion, fever, and sleeping difficulties. Wheezing indicates narrowing of the airways. Retraction of intercostal muscles may occur with inspiration, and exhalation is prolonged through pursed lips. The patient is generally more comfortable in the upright position. Tachycardia and hypotension may result from reduced cardiac output.

ABG monitoring is a sensitive indicator of the respiratory status of the patient with COPD. It is important to know the patient's baseline ABG values to detect changes that indicate ARF. The patient with COPD usually has baseline ABG results that show a normal pH, a moderately low PaO_2 in the range of 60 to 65 mm Hg, and an elevated $PaCO_2$ in the range of 50 to 60 mm Hg (compensated respiratory acidosis). When ARF ensues, the pH decreases, the $PaCO_2$ increases, and the PaO_2 often decreases, resulting in respiratory acidosis and tissue hypoxia. An additional indicator is a change in the patient's mental status and signals an immediate evaluation (see box, "Clinical Alert: Chronic Obstructive Pulmonary Disease").

! CLINICAL ALERT

Chronic Obstructive Pulmonary Disease

During an acute exacerbation of chronic obstructive pulmonary disease, the risk of death is highest in patients with a low PaO_2, respiratory acidosis, significant comorbidities, and the need for ventilatory support.

Interventions

Box 14-2 outlines the care of patients with stable COPD. These interventions should be individualized to reduce risk factors, manage symptoms, limit complications, and enhance the patient's quality of life. When a patient has an acute exacerbation, the goals of therapy are to provide support during the episode of acute failure, to treat the triggering event, and to return the patient to the previous level of functioning.

BOX 14-2 TREATMENT OF STABLE CHRONIC OBSTRUCTIVE PULMONARY DISEASE

- Reduce exposure to airway irritants
- Counseling/treatment for smoking cessation
- Remain in air-conditioned environment during times of high air pollution
- Influenza and pneumococcal vaccinations
- Inhaled bronchodilators (short-acting, long-acting, or combination)
- Inhaled glucocorticosteroids (for severe disease and repeated exacerbations)
- Pulmonary rehabilitation program with exercise training
- Long-term administration of oxygen more than 15 hours/day (for severe disease)

Oxygen

The most important intervention for acute exacerbation of COPD is to correct hypoxemia. O_2 should be administered to achieve a PaO_2 greater than 60 mm Hg or an SaO_2 greater than 90%.[47,53] However, delivering high concentrations of O_2 in an attempt to raise the PaO_2 above 60 mm Hg will not significantly raise the SaO_2 and may also blunt the COPD patient's hypoxic drive. This can diminish respiratory efforts and further increase CO_2 retention. Oxygen should be titrated slowly and incrementally along with reevaluation of arterial blood gases to monitor both O_2 and CO_2 levels.

Bronchodilator Therapy

Table 14-3 lists commonly administered bronchodilator agents. Short-acting, inhaled beta$_2$-agonists cause bronchial smooth muscle relaxation that reverses bronchoconstriction. They are primarily administered via a nebulizer or a metered-dose inhaler with a spacer. The dosage and frequency vary, depending on the delivery method and the severity of bronchoconstriction. Adverse effects are dose related and are more common with oral or intravenous administration compared with inhalation. Adverse effects include tachycardia, dysrhythmias, tremors, hypokalemia, anxiety, bronchospasm, and dyspnea. Beta$_2$-agonists should be administered cautiously in patients with cardiac disease. Long-acting beta$_2$-agonists are effective in controlling stable COPD, but their onset of action is too long to be useful in the rapid treatment of acute exacerbations. They are administered by inhalation using a metered-dose inhaler or dry powder inhaler.

Anticholinergics may also be administered to treat bronchoconstriction. They are indicated for patients who are not immediately responsive to tolerate beta$_2$-agonists and may be used in combination. The use of methylxanthines for acute exacerbation is controversial and requires the monitoring of trough blood levels to maintain therapeutic concentrations.[47] Cardiac side effects may be seen in addition to central nervous system stimulation that may lead to headache, restlessness, and seizures. The use of expectorants, mucolytic agents, and chest physical therapy has not been found to be effective in the management of COPD exacerbations.

Corticosteroids

Administration of oral or intravenous corticosteroids for a period of 7 to 10 days to decrease airway inflammation is beneficial in the management of an acute exacerbation of COPD.[47] Common adverse effects of steroid therapy include hyperglycemia and an increased risk of infection. There may also be an unexplained association between steroid use in the critically ill and the development of skeletal muscle neuromyopathy.

Antibiotics

Antibiotic therapy is recommended when dyspnea is accompanied by increased sputum volume and purulence, or if mechanical ventilation is needed. Infections are commonly caused by *Haemophilus influenzae, Streptococcus pneumoniae,* and *Moraxella catarrhalis.*[47] Multiple drug-resistant bacterial infections are common in COPD exacerbations, and antibiotic selection should be based on local bacterial resistance patterns and on sensitivity reports from sputum cultures.[52]

TABLE 14-3 PHARMACOLOGY

Bronchodilators

MEDICATION	MECHANISM OF ACTION	ADVERSE EFFECTS/NURSING IMPLICATIONS
Beta$_2$-agonists (short-acting) Albuterol Bitolterol Fenoterol Pirbuterol Terbutaline	Bronchial smooth muscle relaxation; relief of acute symptoms	Tremor, anxiety, bronchospasm, dyspnea, tachycardia, dysrhythmias, palpitations, hypertension, hypokalemia, throat irritation
Beta$_2$-agonists (long-acting) Salmeterol Formoterol	Bronchial smooth muscle relaxation; long-term prevention of symptoms	Same as for short-acting beta$_2$-agonists; do not use to treat acute exacerbations
Anticholinergics Ipratropium bromide Oxitropium bromide Tiotropium bromide	Inhibit action of acetylcholine, causing bronchial smooth muscle relaxation	Dry mouth, bitter taste, dizziness, bronchoconstriction, palpitations; lower incidence of tachycardia than beta$_2$-agonists; avoid contact with eyes
Methylxanthines Theophylline Aminophylline	Phosphodiesterase inhibitor	Tremor, tachycardia, dysrhythmias, CNS stimulation (headache, seizures, restlessness), nausea, vomiting; do not crush sustained-release capsules; monitor trough levels

CNS, Central nervous system.

Ventilatory Assistance

Patients with ARF from a COPD exacerbation benefit from early treatment with NPPV. Unlike invasive mechanical ventilation that requires insertion of an ETT or a tracheostomy, NPPV assists the patient's respiratory efforts by delivering positive airway pressure through a nasal, oronasal, or full face mask.

Contraindications to NPPV include respiratory arrest, hemodynamic instability, thick or copious secretions, a change in mental status or uncooperative, extreme obesity, burns, and head or facial trauma/surgery.[49] Studies on the use of NPPV in COPD exacerbations have shown a decrease in the need for intubation, lower mortality rates, a decreased critical care length of stay, and a decrease in the occurrence of health care–acquired pneumonia.[40,49]

Intubation and invasive mechanical ventilation are indicated in those patients who, despite aggressive therapy, develop significant mental status changes, severe dyspnea and respiratory muscle fatigue, respiratory acidosis, significant hypoxemia, or hypercapnia.

In the late stages of severe COPD, patients often report that their quality of life deteriorates because of severe activity limitations and comorbid conditions. Decisions regarding the use or avoidance of intubation, mechanical ventilation, cardiopulmonary resuscitation, and other forms of life support should be made by the patient in conjunction with the patient's family and physician before ARF occurs. Critical care nurses are in an ideal position to facilitate discussions about advance directives and to answer questions for the patient and significant others.

ACUTE RESPIRATORY FAILURE IN ASTHMA

Pathophysiology

Asthma is a chronic inflammatory disorder of the airways. The inflammation causes the airways to become hyperresponsive when the patient inhales allergens, viruses, or other irritants (Box 14-3). Episodic airflow obstruction results because these irritants cause bronchoconstriction, airway edema, mucus plugging, and airway remodeling[48,60] (Figure 14-3). Air trapping, prolonged exhalation, and $\dot{V}/\dot{Q}$ mismatching with an increased intrapulmonary shunt occur. The airflow limitations in asthma are largely reversible. When asthma is controlled, symptoms and exacerbations should be infrequent.

BOX 14-3 ASTHMA TRIGGERS

Inhalant Allergens
- Animals
- House-dust mites
- Cockroaches
- Indoor fungi
- Outdoor allergens

Occupational Exposure
- Organic and inorganic dusts
- Chemical agents
- Fumes

Irritants
- Tobacco smoke
- Indoor/outdoor pollution
- Fumes: perfumes, cleaning agents, sprays

Other Factors Influencing Asthma Severity
- Viral respiratory infections
- Rhinitis/sinusitis
- Gastroesophageal reflux disease
- Exercise
- Sensitivity: aspirin, other nonsteroidal antiinflammatory drugs, sulfites
- Topical and systemic beta-blockers

Assessment

Symptoms of asthma exacerbation are wheezing, dyspnea, chest tightness, and cough, especially at night or in the morning. The patient initially hyperventilates, producing respiratory alkalosis. As the airways continue to narrow, it becomes more difficult for the patient to exhale. Peak expiratory flow readings will be less than 50% of the patient's normal values. The lungs become overinflated and stiff, which further increases the work of breathing. Nursing assessment will reveal tachypnea, tachycardia, pulsus paradoxus greater than 25 mm Hg, agitation, possible use of accessory muscles, and suprasternal retractions. A severe asthma exacerbation, previously referred to as status asthmaticus, occurs when the bronchoconstriction does not respond to bronchodilator therapy, and ARF ensues. The patient experiences fatigue from the severe dyspnea, cough, and increased work of breathing. Hypercapnia, hypoxia, and respiratory acidosis develop, and cardiac output decreases as a result of a decreased venous return that is related to increased intrathoracic pressures (see box, "Clinical Alert: Asthma").

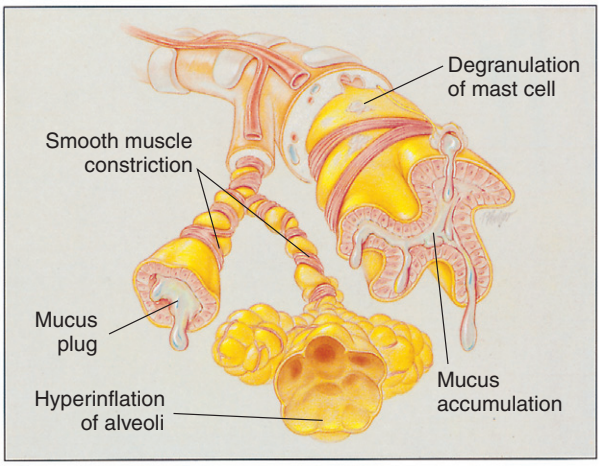

FIGURE 14-3 Airway obstruction caused by asthma. Bronchial asthma: thick mucus, mucosal edema, and smooth muscle spasm causing obstruction of small airways. (From McCance KL, Huether SE. *Pathophysiology: The Biologic Basis for Diseases in Adults and Children*. 6th ed. St. Louis: Mosby; 2010.)

! CLINICAL ALERT

Asthma

Signs of impending acute respiratory failure in a patient with severe asthma include:

- Breathlessness at rest and the need to sit upright
- Speaking in single words; unable to speak in sentences or phrases
- Lethargy or confusion
- Paradoxical thoracoabdominal movement
- Absence of wheezing ("silent chest") indicating no air movement and respiratory muscle fatigue
- Bradycardia
- Respiratory acidosis and hypoxemia with $PaCO_2$ higher than 45 mm Hg and PaO_2 less than 60 mm Hg

Interventions

Mild exacerbations of asthma can be managed by the patient at home with the use of short-acting beta$_2$-agonists to treat bronchoconstriction (see Table 14-3). Treatment of acute, severe exacerbations of asthma requires O_2 therapy, repeated administration of rapid-acting inhaled bronchodilators, and systemic steroid administration (Table 14-4). Most patients respond well to treatment, but some may need intubation and mechanical ventilation. Because of severe airflow obstruction, these patients are at risk for developing dynamic lung hyperinflation (auto-PEEP), lung injury from barotrauma, and hemodynamic compromise.[9] Precise management of mechanical ventilation is required to enhance outcomes and prevent complications. In cases that are refractory to standard treatment, oxygenation may be improved by delivering a mixture of helium and O_2 (heliox) to the lungs. Because helium is less dense than O_2, it enhances gas flow through the constricted airways and may improve oxygenation.[9]

During a patient's recovery from a severe asthmatic event, the critical care nurse should focus efforts on teaching the patient asthma management techniques because patient and family education is essential for achieving asthma control. Persons with asthma are taught how to implement environmental controls to prevent symptoms, understand the differences between medications that relieve and control symptoms, properly use inhaler devices, and monitor their level of asthma control.[48] A written action plan and goals of treatment mutually determined by the patient and the healthcare provider helps patients to achieve asthma control and assists with early identification and treatment of exacerbations.

ACUTE RESPIRATORY FAILURE RESULTING FROM PNEUMONIA

Definition and Etiology

Pneumonia is the leading cause of death from infection in the United States and a common cause of acute respiratory failure.[43,50,68] Pneumonia is a lower respiratory tract infection with a variety of risk factors. Classification of the pneumonia is important because of the likely organism and treatment (Table 14-5). Populations that are at increased risk include the elderly, alcoholics, tobacco smokers, and those with lung or heart disease, head injury, malignancies, renal or liver failure, diabetes mellitus, splenic dysfunction, or any conditions with immunosuppression.

TABLE 14-4 EMERGENCY TREATMENT OF SEVERE ASTHMA

THERAPY	PURPOSE	GOALS
Oxygen via nasal cannula or face mask	Correct hypoxemia	Maintain SpO_2 ≥90%
Inhaled rapid-acting $beta_2$-agonists via nebulizer (continuous); followed by intermittent on-demand therapy	Relieve airway obstruction caused by bronchoconstriction	Achieve PEF >70% of predicted or personal best; normalizing/improving ABGs; respiratory rate <30 breaths/min without use of accessory muscles
Inhaled anticholinergics (added to $beta_2$-agonist therapy)	Relieve bronchoconstriction	Relieve sensation of dyspnea; patient able to complete full sentences without breathlessness
Systemic corticosteroids (orally or intravenous)	Reverse airway inflammation	Improve lung sounds; prevent intubation

ABGs, Arterial blood gases; *PEF,* peak expiratory flow; *SpO_2,* arterial oxygen saturation by pulse oximetry.

TABLE 14-5 PNEUMONIA DEFINITIONS AND COMMON INFECTIOUS CAUSES

PNEUMONIA	CRITERIA	LEADING INFECTIOUS CAUSES
Community-acquired pneumonia (CAP)	Pneumonia that develops outside the hospital in patients who have either not been hospitalized or living in a long-term care facility for more than 2 weeks	*Streptococcus pneumoniae* *Haemophillus influenzae* *Mycoplasma pneumoniae* *Chlamydophila pneumonia* *Legionella*
Healthcare-acquired pneumonia (HCAP)	Develops in patients who were hospitalized in an acute care hospital for 2 or more days within 90 days of the infection; resided in a long-term care facility; received intravenous antibiotic therapy, chemotherapy, or wound care within the past 30 days; or had hemodialysis at a hospital or clinic	*Staphylococcus aureus* *Pseudomonas aeruginosa* *Enterobacter species* *Acinetobacter baumannii* *Klebsiella pneumoniae* *Escherichia coli* *Candida* species *Klebsiella oxytoca* Coagulase-negative staphylococci *Enterococcus* species
Hospital-acquired pneumonia (HAP)	Pneumonia that develops in hospitalized patients which occurs more than 48 hours after hospital admission excluding any infection incubating at the time of admission	
Ventilator-associated pneumonia (VAP)	Pneumonia that develops in patients which develops 48 hours or more after intubation	

Based on data from Mandell LA, Wunderkink RG, Anzueto A, Bartlett JG, et al. Infectious disease society of America/American thoracic society concensus guidelines on the management of community-acquired pneumonia in adults. *Clinical Infectious Disease,* 2007;44, S27-S72; Hidron AL, Edwards JR, Patel J, Horan TC, et al. Antimicrobial-resistant pathogens associated with healthcare-associated infections: annual summary of data reported to the national healthcare safety network at the centers for disease control and prevention, 2006-2007. *Infection Control and Hospital Epidemiology,* 2008;29(11), 996-1011.

Pathophysiology

For pneumonia to occur, enough organisms must accumulate in the lower respiratory tract to overwhelm the patient's normally competent defense mechanisms. The lower respiratory tract is usually a sterile environment and protected by the filtration, warming and filtering of air through the upper airway, closure of the epiglottis, cough and sneezing reflexes, mucocilliary clearance, and alveolar macrophages. The major routes of entry for these organisms include aspiration of gastric or oropharyngeal secretions, inhalation of aerosols or particles, and hematogenous spread from another infected site into the lungs. The normal bronchomucociliary clearance mechanism is overwhelmed by the organism, causing a large influx of phagocytic cells along with exudate into the airways and alveoli. This inflammatory response leads to a ventilation perfusion mismatch resulting in dyspnea, hypoxemia, fever, and leukocytosis.[7]

The pathogens responsible for pneumonia vary depending on the type (community versus hospital-acquired) and are also based on the environmental factor or cause[30,42] (see Table 14-5). The pathogens include bacterial, viral, and fungal causes along with multidrug-resistant organisms, such as methicillin-resistant *Staphylococcus aureus* (MRSA). The most common bacterial causes of CAP are *streptococcus* and *pneumococcus* infection. Pneumococcal pneumonia, is preventable by receiving the pneumococcal vaccination[12] (Table 14-6). The most common viral cause is influenza. The influenza vaccine has been shown to reduce the incidence of pneumonia by 53%, and routine vaccination annually is recommended for all persons older than 6 years in the United States.[11,50] The composition of the vaccine changes yearly based on projected viruses for that season. As an example, influenza A or H1N1 vaccine was added to the 2010 vaccination in response to the 2009 H1N1 pandemic. Fungal causes of pneumonia are uncommon unless the patient is immunocompromised.

Assessment

The clinical presentation for pneumonia commonly includes fever, cough, purulent sputum, hemoptysis, dyspnea, tachypnea, pleuritic chest pain, and abnormal breath sounds. Elderly patients may present with nonspecific symptoms such as confusion, weakness, lethargy, or change in appetite.[51] Recommended diagnostic studies include a chest x-ray which may show new or progressive infiltrates. Pretreatment blood and sputum cultures should be obtained without delaying implementation of antibiotic therapy. Urinary antigen tests for *Legionella pneumophila* and streptococcus pneumonia may also be obtained.[42] Abnormal laboratory results include an elevated white blood cell count and arterial blood gases demonstrating hypoxemia and hypocapnia.[7]

TABLE 14-6 PNEUMOCCAL VACCINE RECOMMENDATIONS FOR PREVENTION OF PNEUMOCOCCAL DISEASE

- All persons between 19 and 64 years old by risk group (single dose)
- All persons 65 years of age or older (single dose if not previously received, or second dose with waiting period of 5 years between doses if received previously as high risk)

RISK GROUP	UNDERLYING MEDICAL INDICATION
Immunocompetent	Chronic heart disease (excluding hypertension) Chronic lung disease Diabetes mellitus Cerebrospinal fluid leaks Cochlear implants Alcoholism Chronic liver disease (includes cirrhosis) Cigarette smoking
Functional or anatomical asplenia	Sickle cell disease or other hemoglobinopathies Asplenia, spenic dysfunction, or splenectomy
Immunocompromised	Congenital or acquired Human immunodeficiency virus infection Chronic renal failure Hemotological and generalized malignancies Treatment with immunosuppressive drugs (including long-term systemic corticosteroids or radiation therapy) Solid organ transplantation

Modified from the Centers for Disease Control and Prevention. Updated recommendations for prevention of invasive pneumococcal disease among adults using the 23-valent pneumococcal polysaccharide vaccine. *MMWR. Morbidity and Mortality Weekly Report,* 2010;59:1102-1106.

The Centers for Medicare & Medicaid Services defined core performance measure for pneumonia that are collected by all U.S. hospitals and publicly reported at www.hospitalcompare.hhs.gov. These core measures include: (1) the first dose of antibiotics is given within 6 hours of arrival to the hospital; (2) the correct antibiotic is given; (3) blood cultures are obtained within 24 hours for all patients admitted to an ICU, with blood cultures drawn in the ED before antibiotics are given; (4) smoking cessation advice given to the patient is documented; and (5) pneumococcal and influenza vaccines are administered to appropriate candidates.

VENTILATOR-ASSOCIATED PNEUMONIA

Ventilator-associated pneumonia (VAP) is a preventable hospital-acquired infection with high morbidity and mortality. Crude mortality varies from 10% to 40% and reaches as high as 76% if the disease is caused by high-risk pathogens.[1] Ventilated patients who develop VAP have higher mortality rates and longer length of stays for both the ICU and hospital.[5] The risk for developing VAP is highest during the first 5 days of ventilation.[37] Recently reported VAP rates ranged from 0.5 cases per 1000 ventilator days in respiratory ICUs to 10.7 cases per 1000 ventilator days in burn units.[19]

Pathophysiology

The pathogenesis of VAP is depicted in Figure 14-4. Patients with an ETT are at increased risk for aspiration secondary to the natural anatomical barrier of the glottis being violated. The ETT is inserted into the trachea past the vocal cords, thereby holding the glottis in the open position and compromising its ability to prevent aspiration.[1] Sources of exogenous pathogens include contamination from healthcare personnel, ventilator and respiratory equipment, and the biofilm coating on the ETT.

Assessment

Clinical criteria for the diagnosis of VAP include a new or progressive pulmonary infiltrate along with fever, leukocytosis, and purulent tracheobronchial secretions. Cultures can be obtained via bronchoscopy, protected-specimen brush, or endotracheal aspirate, and results are reported in either quantitative or semiquantitative terms. Diagnosis is complicated by a lack of sensitive and specific criteria; there is no gold standard. Over 1500 hospitals nationally report VAP rates using the National Healthcare Safety Network (NHSN) surveillance criteria. This includes the presence of a new or persistent lung density seen on chest x-rays with two or more of the following: temperature of more than 38.5° C or less than 36.5° C, leukocyte count of more than 11,000 cells/microliter or less than 5000 cells/microliter, and the presence of purulent endotracheal secretions.[1,13] The clinical pulmonary infection score (CPIS) may also aid in diagnosis. This score combines clinical, radiographic, physiological (PaO_2/FiO_2 ratio), and microbiological information into a numerical value that predicts the presence or absence of pneumonia (Table 14-7).[1,23]

Because of the difficulty in accurately diagnosing VAP based on existing criteria, the NHSN has proposed new surveillance for ventilator-associated events, which includes VAP. Proposed changes to surveillance criteria for adult patients receiving traditional ventilation are summarized in Figure 14-5. As these

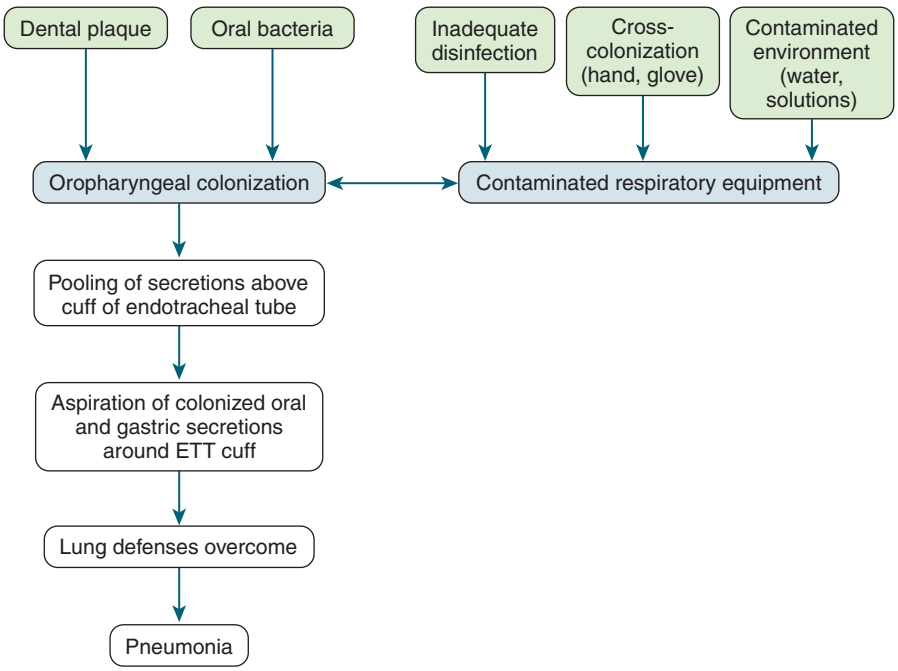

FIGURE 14-4 Role of airway management in the pathogenesis of ventilator-associated pneumonia.

TABLE 14-7 MODIFIED CLINICAL PULMONARY INFECTION SCORE

CPIS POINTS	0	1	2
Tracheal secretions	Rare	Abundant	Abundant and purulent
Chest x-ray infiltrates	No infiltrate	Diffuse	Localized
Temperature (°C)	≥36.5 and ≤38.4	≥38.5 and ≤38.9	≥39 or ≤36
Leukocyte count (per microliter)	≥4000 and ≤11,000	<4000 or >11,000	<4000 or >11,000 + band forms ≥500
PaO_2/FiO_2 ratio (mm Hg)	>240 or ARDS		≤240 and no evidence of ARDS
Microbiology	Negative		Positive

Score each section and determine total points. A score of more than 6 at baseline or after incorporating the Gram stains or culture results is suggestive of pneumonia.
ARDS, Acute respiratory distress syndrome; *CPIS,* clinical pulmonary infection score; *FiO_2,* fraction of inspired oxygen; *PaO_2,* partial pressure of oxygen in arterial blood.
From Fartoukh M, Maitre B, Honore S, Cerf C, Zahar JR, & Brun-Buisson C. Diagnosing pneumonia during mechanical ventilation: The clinical pulmonary infection score revisited. *American Journal of Respiratory and Critical Care Medicine*, 2003;*168*, 173-179.

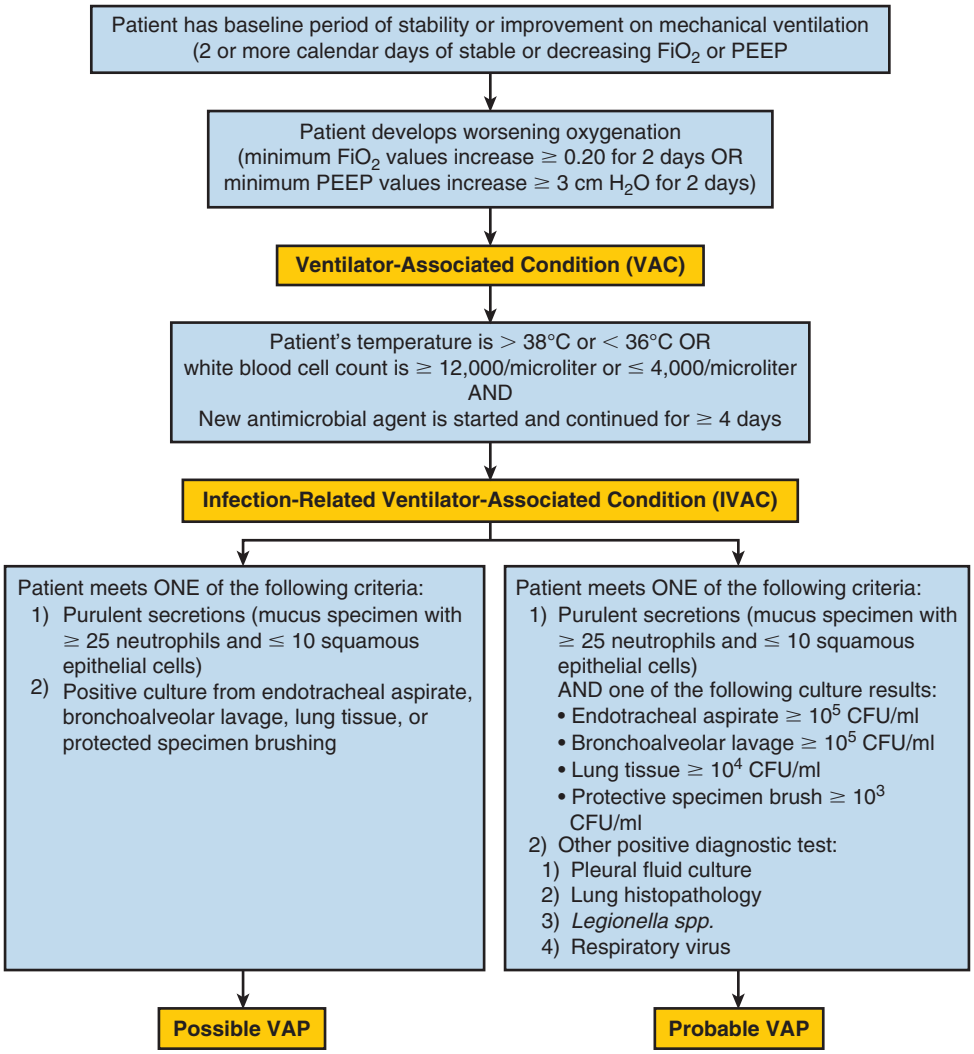

FIGURE 14-5 Proposed NHSN Surveillance Algorithm for Ventilator-Associated Events. (Modified from Centers for Disease Control and Prevention. *Improving Surveillance for Ventilator-Associated Events in Adults,* 2012. Retrieved May 28, 2012 from http://www.cdc.gov/nhsn/PDFs/vae/CDC_VAE_CommunicationsSummary-for-compliance_20120313.pdf.)

criteria are evolving at time of publication, the reader is encouraged to monitor the NHSN website for updated information.

Interventions

The interventions for VAP are aimed at prevention and treatment. The prevention of VAP is a major focus of many recent safety initiatives and focuses on modification of risk factors. The Institute for Healthcare Improvement proposed a "bundle of care" for mechanically ventilated patients. Bundles are evidence-based interventions grouped together to improve outcomes. Five strategies are included in the ventilator bundle: elevation of head of bed (HOB) to at least 30 degrees, daily awakening ("sedation vacation") with assessment of the need for mechanical ventilation, prophylaxis for stress ulcers, prophylaxis for deep venous thrombosis, and daily oral care with chlorhexidine.[2,33] Strategies for prevention of VAP are summarized in Box 14-4, and an example of an oral care protocol is described in Box 14-5

BOX 14-4 PREVENTION OF VENTILATOR-ASSOCIATED PNEUMONIA

1. Effective infection control measures including staff education and hand hygiene
2. Conduct surveillance of ICU infections.
3. Implement components of IHI Ventilator Bundle.
   - Maintain head-of-bed elevation at 30 to 45 degrees.
   - Sedation interruptions and daily assessment of readiness to wean from ventilator
   - Deep vein thrombosis prophylaxis
   - Peptic ulcer disease prophylaxis
   - Daily oral care with chlorohexidine
4. Prevent transmission of microorganisms.
   - Use sterile water for use in or cleaning of respiratory equipment.
   - Change the ventilator circuit only when visibly soiled.
   - Drain condensate in ventilator circuits away from the patient.
   - Do not instill normal saline into ETT.
5. Modify host risk for infections: prevent aspiration.
   - Avoid intubation and reintubation, use noninvasive ventilation if possible.
   - Intubate patients orally.
   - Use orogastric tubes.
   - If available, use ETT that allows continuous aspiration of subglottic secretions.
   - Use sedation and weaning protocols.
6. Other prevention strategies
   - Enteral nutrition is preferred over parenteral.
   - Develop and implement a mobilization program.

ETT, Endotracheal tube; *IHI,* Institute for Healthcare Improvement. Modified from American Thoracic Society. (2005). Guidelines for the management of adults with hospital-acquired, ventilator-acquired pneumonia, and healthcare-associated pneumonia. *American Journal of Respiratory and Critical Care Medicine, 171*, 388-416; Institute of Healthcare Improvement. (2010). *Protecting 5 million lives from harm. Getting started kit: Prevent ventilator-associated pneumonia how-to guide.* Retrieved May 22, 2011, from www.ihi.org.

BOX 14-5 EXAMPLE OF AN ORAL CARE PROTOCOL

Equipment

1. Oral suction catheter
2. Soft toothbrush or suction-toothbrush
3. Toothettes, oral swab or suction-swab
4. 1.5% Hydrogen peroxide mouth rinse or toothpaste
5. Water-based mouth moisturizer
6. Oral chlorhexidine gluconate (0.12%) rinse
7. Suction source and tubing

Interventions

1. Assess intubated patients every 2 hours, before repositioning or deflating the endotracheal tube, and as needed to determine the need for removal of oropharyngeal secretions. Suction as needed.
2. Brush teeth, gums and tongue twice a day using a soft pediatric or adult toothbrush with toothpaste or cleaning solution.
3. Swab oral chlorhexidine gluconate (0.12%) over all oral surfaces for 30 seconds twice a day; suction excess.
4. Avoid brushing teeth or oral intake for 2 hours after chlorhexidine use.
5. Provide oral moisturizing to oral mucosa and lips every 2 to 4 hours.

Treatment

VAP is associated with a high risk of mortality if an appropriate antibiotic regimen is not started in a timely manner.[1] The guidelines for antibiotic use have two major goals: to provide therapy with an appropriate and adequate empirical antibiotic regimen, and to achieve the first goal without overusing and abusing antibiotics. The initial antibiotic therapy algorithm includes two groups of patients: patients with early-onset VAP without any risk factors for multidrug-resistant (MDR) pathogens, and patients with late-onset VAP or risk factors for MDR pathogens.[30] Patients with early-onset VAP without any risk factors for MDR may be placed on narrow-spectrum monotherapy based on knowledge of local microbiological data. Patients at risk for MDR pathogens require broad-spectrum therapy based on knowledge of the local hospital antibiogram. When the patient is at high risk for MDR, three antibiotics are prescribed: two drugs of different classes active against *Pseudomonas aeruginosa* and a third drug to treat methicillin-resistant *S. aureus*. The antibiotic regimens for both classifications of patients should be narrowed once the results of the quantitative cultures are known *(deescalation therapy)*. Clinical improvement takes about 3 days. If clinical improvement does not occur within 72 hours, the patient should be evaluated for noninfectious causes of the symptoms or extrapulmonary infections. If a patient receives an appropriate antibiotic regimen, the duration of therapy can be reduced to 7 to 8 days versus the traditional 14 to 21 days.[1,57]

ACUTE RESPIRATORY FAILURE RESULTING FROM PULMONARY EMBOLISM

Definition/Classification

An embolus is a clot or plug of material that travels from one blood vessel to another smaller vessel. The clot lodges in the smaller vessel and obstructs blood flow. An embolus in the pulmonary vasculature is called a pulmonary embolism (PE). The embolus may be a clot that has broken off from a deep vein thrombosis (DVT), a globule of fat from a long bone fracture, septic vegetation, or an iatrogenic catheter fragment. In pregnancy, amniotic fluid can be the cause of a PE. Most PEs originate from DVT of the lower extremities. PE and DVT are the two components of the disease process known as venous thromboembolism (VTE).[54]

PE is classified in several different ways. An initial classification may be acute or chronic. An acute PE occurs quickly and either responds to treatment, or death occurs. A chronic PE initially responds to treatment but then reoccurs. In chronic PE, small clots continue to develop and travel to the pulmonary vascular bed after treatment. Chronic PE is typically caused by a coagulopathy. A PE is also classified based on the amount of pulmonary vascular occlusion: massive, submassive, or nonmassive.[54] A massive PE obstructs 50% or more of the pulmonary vasculature or two or more lobar arteries. The clinical presentation of a massive PE may include syncope, hypotension, extreme hypoxemia, or cardiac arrest.[18,41] A *submassive* PE is usually noted on an echocardiogram as right ventricular dysfunction without hemodynamic instability. A *nonmassive* PE is not associated with right ventricular dysfunction.

BOX 14-6 RISK FACTORS FOR VENOUS THROMBOEMBOLISM

Hereditary
- Antithrombin deficiency
- Protein C deficiency
- Protein S deficiency
- Factor V Leiden
- Activated protein C resistance
- Dysfibrinogenemia
- Plasminogen deficiency

Acquired
- Reduced mobility
- Advanced age
- Cancer
- Acute medical illness
- Major surgery
- Trauma
- Spinal cord injury
- Pregnancy and postpartum
- Polycythemia vera
- Antiphospholipid antibody syndrome
- Oral contraceptives
- Hormone replacement therapy
- Heparins
- Chemotherapy
- Obesity
- Central venous catheterization
- Immobilizer or cast

Modified from Tapson VF. Acute pulmonary embolism. *New England Journal of Medicine,* 2008;358(10), 1037-1052.

Etiology

The three main mechanisms that favor the development of VTE, often referred to as Virchow triad, are (1) venous stasis, or a reduction in blood flow; (2) altered coagulability of blood; and (3) damage to the vessel walls. Specific causes of VTE are listed in Box 14-6.

Acute PE remains a cardiovascular emergency and has a high mortality rate. PE is the third highest cause of hospital mortality and is considered to be the leading cause of preventable hospital deaths in the United States. The Centers for Medicare & Medicaid Services has noted DVT and PE following total knee or hip replacements as a *never event;* an avoidable medical error.[45] Critically ill patients are also at high risk for VTE, and DVT prophylaxis is a part of the IHI ventilator bundle. PE is also the leading cause of maternal death after delivery. It occurs in 2 of every 100,000 live births.[61]

Pathophysiology

When an embolus completely or partially occludes the pulmonary artery or one of its branches, a mechanical obstruction impedes forward flow of blood. The pulmonary circulation has an enormous capacity to compensate for a PE. This compensatory mechanism results from the lung vasculature that is necessary to accommodate increased blood flow during exercise, and it is the reason many patients do not initially decompensate from a massive PE. After an embolus lodges in the pulmonary vasculature, blood flow to the alveoli beyond the occlusion is eliminated. The result is a lack of perfusion to ventilated alveoli, an increase in dead space, a $\dot{V}/\dot{Q}$ mismatch, and a decrease in CO_2 tension in the embolized lung zone. Gas exchange cannot occur. Reaction to the mechanical obstruction causes the release of a number of inflammatory mediators such as prostaglandin, serotonin, and histamine. The ensuing inflammation causes constriction of bronchi and surrounding blood vessels.[7]

Constriction in the terminal airways of the nonperfused lung zones results in alveolar shrinking and an increase in the work of breathing. The reduction in blood flow to the alveoli also results in hypoxia for the type II pneumocytes, which are responsible for the production of surfactant. Although the effects are not seen for 24 to 48 hours, the decrease in surfactant results in an unequal gas distribution, an increase in the work of breathing, and a stiffening and collapse of the alveoli. Ventilation is then shifted away from these units, thus worsening the $\dot{V}/\dot{Q}$ mismatch. Atelectasis

and shunting transpire as a result of the release of serotonin from the platelets that surround the clot. The result is peripheral airway constriction, which often involves functioning alveoli. In this situation, perfusion with inadequate ventilation occurs.[18,41,65]

The entire process may lead to pulmonary hypertension and an increase in right ventricular workload to maintain pulmonary blood flow. The right ventricle increases in size, causing a leftward shift of the septum. As the process continues, it can lead to decreased left ventricular filling and output. The patient may develop right and left ventricular failure, leading to decreased cardiac output and shock.[7]

The overall prognosis after a PE depends on two main factors. The first is the patient's underlying disease state before the PE, and the second is the appropriate diagnosis and treatment. Appropriate anticoagulation decreases mortality to less than 5%.[54,65]

Assessment

Dyspnea, hemoptysis, and chest pain have been called the "classic" signs and symptoms for a PE, but the three signs and symptoms actually occur in less than 20% of cases.[65] A PE should be suspected in any patient who has unexplained cardiorespiratory complaints and has any risk factors for VTE. A relatively common symptom of PE is the sudden onset of dyspnea. The patient may also be especially apprehensive or anxious, with a feeling of impending doom. Syncope, defined as a loss of consciousness lasting at least 2 minutes, is the presenting symptom in 10% to 15% of patients with a PE. Other common signs and symptoms of PE are chest wall tenderness, chest pain aggravated by deep inspiration, tachypnea, decreased SpO_2, tachycardia, cough, crackles, wheezing, and hemoptysis. Additional signs and symptoms that may occur are an accentuated pulmonic component of the second heart sound (S2), new-onset atrial fibrillation, fever, new-onset reactive airway disease (adult onset asthma), cyanosis, and diaphoresis.[41,54,65] Approximately 79% of patients with PE have positive evidence of DVT in their legs.

Diagnosis

D-Dimer Assay

D-Dimers are fibrin degradation products or fragments produced during fibrinolysis. The D-dimer assay is a sensitive but nonspecific test to diagnose a PE. A negative D-dimer assay has about 90% sensitivity for ruling out a PE in young patients with no comorbidities. A positive D-dimer assay can occur in a number of other conditions such as infection, cancer, surgery, pregnancy, heart failure, or kidney failure.[54,65]

Ventilation-Perfusion Scan

A $\dot{V}/\dot{Q}$ scan is a noninvasive scintigraphic lung scan that calculates pulmonary airflow and blood flow. A $\dot{V}/\dot{Q}$ scan may detect dead space from impaired perfusion of ventilated alveoli. Results of $\dot{V}/\dot{Q}$ scans are reported as low, medium, or high probability.[65]

Duplex Ultrasonography

Duplex ultrasonography is a noninvasive imaging study useful in detecting lower extremity DVT. It has a high sensitivity and specificity for DVT in the leg above the knee, but is not accurate in detecting DVT in pelvic vessels or small vessels in the calf.[54,61]

High-Resolution Multidetector Computed Tomography Angiography

High-resolution multidetector CT angiography (MDCTA; spiral CT) has become the preferred tool for detecting a PE. It is highly accurate for direct visualization of large emboli in the main and lobar pulmonary arteries. MDCTA does not always visualize small emboli in distal vessels, but a pulmonary angiogram has the same limitation.[65]

Magnetic Resonance Imaging

Magnetic resonance imaging has a sensitivity and specificity comparable to that of spiral CT, but it is rarely used to diagnose PE in critically ill patients.

Pulmonary Angiogram

A pulmonary angiogram is considered the gold standard for detecting a PE. It provides direct anatomical visualization of the pulmonary vasculature. Pulmonary angiography is an invasive procedure consisting of catheterization of the right side of the heart with contrast medium injected through the catheter into the pulmonary vascular system. MDCTA is replacing pulmonary angiography as the standard because it is noninvasive and has a high level of sensitivity and specificity.[54]

Prevention

The best therapy for VTE and subsequent PE is prevention.[25,31] Evidence-based strategies for prevention of VTE include the following. (1) Assess patients on hospital admission and routinely throughout their hospital stay for risk of VTE. The nurse, physician, and other members of the multiprofessional team need to review daily both risk and use of VTE prophylaxis. All critically ill patients should receive prophylaxis treatment. (2) It is recommended that patients at high risk for bleeding use mechanical prophylaxis with graduated compression stockings, intermittent pneumatic compression devices, or both, until the risk for bleeding has been resolved. When using these devices, it is imperative to ensure that they are applied correctly and removed for only short periods each day. (3) Patients at moderate risk for developing VTE should receive either low-dose unfractionated heparin or low–molecular weight heparin. (4) Low–molecular weight heparin is recommended for patients at high risk for VTE. (5) The nurse should implement a mobilization regimen for the patient, with the goal of maximizing the patient's mobility.[31] Box 14-7 outlines some nursing interventions to prevent VTE.

BOX 14-7 NURSING MEASURES TO PREVENT VENOUS THROMBOEMBOLISM

Assess Patient on Admission to Unit for Risk for VTE and Anticipate Prophylaxis Orders

Review Daily with Healthcare Team
- Current VTE risk factors
- Necessity for central venous catheter
- Current VTE prophylaxis
- Risk for bleeding
- Response to treatment

Implement Prescribed Prophylactic Regimen
- Pharmacological (according to risk level)
  - *Moderate-risk:* low-dose unfractionated heparin, low–molecular weight heparin, or fondaparinux
  - *High-risk:* low–molecular weight heparin, fondaparinux, or oral vitamin K antagonist
- Nonpharmacological (mechanical) (patient at high risk for bleeding or in conjunction with pharmacological prophylaxis)
  - Graduated compression stockings and/or intermitted pneumatic compression device

Document Implementation Tolerance, and Complications, of Prophylaxis

Assess Extremities on a Regular Basis
- Pain/tenderness
- Unilateral edema
- Erythema
- Warmth

Implement a Mobility Program

Monitor for Low-Grade Fever

Encourage Fluids to Prevent Dehydration;
- Administer IV Fluids as Prescribed; Maintain Accurate Intake and Output Records

Avoid Adjusting the Knee Section of the Bed or Using Pillows Under Knees

Provide Patient Education Regarding Prevention

DVT, Deep vein thrombosis; *IV,* intravenous; *VTE,* venous thromboembolism.

Treatment

Thrombolytic therapy is indicated for a patient with a massive PE who is in cardiogenic shock or has hypotension unless absolute contraindications are present. Thrombolytics may also be considered for a patient who is hemodynamically stable but has signs and symptoms of reduced right ventricular function.[36] Thrombolytic regimens with short infusion times are recommended. Of the four fibrinolytic drugs available—streptokinase, urokinase, t-PA (alteplase), and r-PA (reteplase)—alteplase has the shortest infusion times and is the most widely used agent.[65,66]

Parenteral anticoagulation therapy is recommended for the treatment of an acute PE and in conjunction with thrombolytic therapy in massive PE.[31,54,65] Anticoagulants do not dissolve the existing clot, but allow the fibrinolytic system to function decreasing the thromboembolic burden. Oral anticoagulation with warfarin may also be initiated concurrently on day 1, and overlap therapy should continue until the international normalized ratio (INR) is in the therapeutic range for at least 2 days. It is essential that the critical care nurse regularly monitor the laboratory values and to assess the patient for any signs or symptoms of bleeding or heparin-induced thrombocytopenia (HIT). The nurse must be attuned to major bleeding, such as intracranial or retroperitoneal hemorrhage, and minor bleeding. Heparin-induced thrombocytopenia is a well-known complication of low–molecular weight heparin and unfractionated heparin. It is caused by antibodies that activate platelets and leads to thrombocytopenia.[61] Direct thrombin inhibitors such as argatroban or lepirudin are alternatives in patients who have developed or have a history of HIT.

Catheter embolectomy or local intraembolic thrombolytic therapies are reserved for patients who have contraindications to thrombolytic therapy. Surgical embolectomy is rarely used and involves manual removal of the thrombus from the pulmonary artery. The patient must be placed on a cardiopulmonary support system during the procedure.[65]

Vena cava filters may be placed in the inferior vena cava to prevent recurrence of PE by preventing clots from migrating from the lower extremities. Two types of filters are available: permanent and temporary retrievable. Permanent vena cava filters are rarely used and have a number of associated complications. Temporary retrievable vena cava filters are used to prevent PE in patients who have contraindications for anticoagulation therapy, major bleeding during anticoagulation therapy, or have recurring PE. These devices can be removed by a minimally invasive technique under fluoroscopy.[65]

Other treatments are focused on maintaining the airway, breathing, and circulation. Supplemental O_2 may be administered to maintain SaO_2 at more than 90%. If the location of the PE is known, positioning the patient with the "good" lung in the dependent position is warranted. Analgesics are given to alleviate pain and anxiety. If the patient is hemodynamically unstable, inotropic or vasopressor support may be required.

ACUTE RESPIRATORY FAILURE IN ADULT PATIENTS WITH CYSTIC FIBROSIS

Definition

Cystic fibrosis (CF) is a genetic disorder (see box, "Genetics") resulting from defective chloride ion transport. The mutation in chloride transport causes the formation of mucus with

little water. The thick, sticky mucus obstructs the glands of the lungs, pancreas, liver, salivary glands, and testes, causing organ dysfunction. Although CF is a multisystem disease, it has the greatest effect on the lungs. The thick mucus narrows the airways and reduces airflow. The constant presence of thick mucus provides an excellent breeding ground for bacteria, leading to chronic lower respiratory tract bacterial infection, chronic bronchitis, and dilatation of the bronchioles. The mucus-producing cells in the lungs increase in number and size over time. Respiratory complications of CF include pneumothorax, arterial erosion, hemorrhage, chronic bacterial infection, and respiratory failure.[35]

GENETICS

Cystic Fibrosis: A Heritable Disorder with Pulmonary and Gastrointestinal Complications

Cystic fibrosis (CF) is a lifelong disorder that may lead to critical illness. The basic pathological abnormality in this disease is a defect in a protein that forms part of the ion channel that transports chloride across epithelial cell membranes on mucosal surfaces. The defective protein is a result of a variation in the *CFTR* gene on chromosome 7.[1] There are more than 1500 variations in this gene. All of them cause some degree of alteration in the construction and function of the chloride ion channel in epithelial cells, typically resulting in reduced chloride transport across epithelial cell membranes in mucus secreting organs.[1]

Organs and tissues most profoundly affected by the defective chloride ion channels are in the pancreas, intestines, lungs, sweat glands, and vas deferens. As a result of defects in the chloride ion channel, there is reduced secretion of chloride and increased reabsorption of sodium and water across epithelial cells. This leads to synthesis of thick mucus. Thick mucus in the respiratory and gastrointestinal tracts, the pancreas, the sweat glands, and other tissues is difficult to clear and interferes with normal organ function. For example, mucus production in the lungs interferes with gas exchange, leading to chronic hypoxemia. Mucus production in the gastrointestinal tract blocks intestinal fluids, resulting in gastrointestinal obstruction. Thick mucus is also more likely to colonize microorganisms, contributing to infection and inflammation with subsequent adverse and irreversible changes in these organs.[1]

The degree of impairment in the chloride ion channel varies as a result of differences in the protein coded by the *CTFR* gene. For example, the most common genetic variation among patients diagnosed with CF, prevents the CTFR protein from folding properly so that the chloride pump does not reach the cell surface. A different genetic defect, less common and associated with less severe symptoms, results in a functional pump but one with a "sticky" gate that disturbs the ability of the epithelial cell to secrete chloride.[5]

Abnormalities are typically not seen unless CFTR function is less than 10%.[3] Those patients with the most severe symptoms have less than 1% CFTR activity and manifest the full spectrum of disease involvement including pancreatic insufficiency; recurrent, severe pulmonary infections; gastrointestinal obstruction; and congenital absence of the vas deferens.[3]

CF is the most common lethal disease inherited by the white population. One in 22 people of European heritage carry one gene for CF.[2] CF is an autosomal recessive disease, which means that both parents must be a carrier of variant *CFTR* genes or have the disease in order for their children to inherit the gene variation that causes CF. Because so many individuals in the United States are symptomless carriers of CF (estimated at more than 10 million people), genetic testing for all couples who are at high risk for being a carrier because of their ethnicity or family history is recommended.[3,4] In general, testing is performed on just one future parent initially; if that person is a carrier, then the other future parent is tested to calculate the risk that their children will have CF. It is not possible to test for all 1300 variations of the *CFTR* gene in a single genetic test. Testing typically looks for 32 to 70 common mutations.[3] Therefore a negative screen does not guarantee that a child will not have CF. A child with CF usually has the same mutation as the carrier parent. If a family has a known uncommon variant, then specific testing for that polymorphism can be performed.

Symptoms of CF are most often manifested in infancy and early childhood by a persistent cough with mucus production that is frequently colonized with bacteria; by loose, bulky stools; and by failure to thrive. Those with milder disease may not have CF diagnosed until adolescence or early adulthood. The presence of aspermia or male infertility is an indication to the clinician to include CF as a diagnostic possibility. In addition to genotyping, tests of pancreatic function and nasal potential-difference measurements are used to diagnose CF. Diagnosis by sweat testing is also used because the defect in chloride ion channels leads to salty secretions. About 1000 new cases of CF are diagnosed in the United States annually.[2]

If CF genetic testing shows both parents are carriers, genetic counseling is strongly recommended by healthcare providers. When both parents have a *CFTR* variant, as with any autosomal disorder, there is a 1-in-4 chance with each pregnancy that the child will have CF.[1,3] Remember, many variants of CF are not included in the typical genetic test, manifestations of CF can be mild, and not all parents will perceive the diagnosis of CF as a serious disorder. In addition, genetic testing and counseling may not be covered by insurance companies. Thus the healthcare provider needs to individualize the approach to advising persons seeking CF genetic testing. The Cystic Fibrosis Foundation has

GENETICS

Cystic Fibrosis: A Heritable Disorder with Pulmonary and Gastrointestinal Complications—cont'd

links to information as well as support groups when CF is a potential or actual condition in a family.[2] Because of improved treatments over the past four decades, the average life span of a child with diagnosed CF has increased from 10 to 36 years.[1] Genomic science is also offering new strategies for treatment.

Therapies for CF have traditionally focused on alleviating symptoms. However, genomic science has resulted in a line of research to identify small molecules that correct the different defects in *CFTR*. Currently, a new drug is in phase 1 testing (in humans). This drug, VX-770, is designed to "prop open" chloride channels characterized by a specific genetic variation (i.e., the G551D mutation).[5] This milestone provides new hope for people who have CF in terms of providing treatment to prevent permanent, irreversible pathology. Active research programs in several academic centers continue to pursue a cure.

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Etiology

CF affects primarily whites but is occasionally seen in other races. For many years, CF was considered a disease of children. Because of significant improvements in care, most people with CF are now living into the third decade of life or longer, and 40% of CF patients are older than 18 years.[16] The diagnosis of CF is typically made early in life (70% by age 1 year), but a few patients receive a diagnosis of CF as adults. A sweat test is the typical diagnostics tool for CF in children. Patients who do not receive a diagnosis until adulthood generally present with respiratory problems and have fewer other systems involved. Many of these patients have a normal or borderline sweat test result. They generally have a better prognosis.

Interventions

Respiratory failure is the cause of death for more than 80% of patients with CF.[39] As the disease process progresses, patients develop increased ventilator requirements, air trapping, and respiratory muscle weakness. All of these conditions are complicated by chronic bacterial infections that can quickly become overwhelming. In the past, mechanical ventilation was not considered a treatment option because patient outcomes were poor. During the last 20 years, the standard of care for ARF in CF has been revisited because of improved ventilator modalities, more aggressive pharmacological therapy, and the option of lung transplantation. Lung transplantation provides the opportunity for a tremendous improvement in the quality of life, but acute exacerbations of respiratory failure must be overcome during the wait for a transplant.

The three cornerstones of care for a patient with CF are antibiotic therapy, airway clearance, and nutritional support. Any patient with CF who is admitted to a critical care unit in ARF must have these three issues addressed immediately.

Antibiotic Therapy

A frequent cause of respiratory failure is pneumonia. Antibiotic selection is based on the patient's most recent sputum bacterial isolates. *P. aeruginosa* is the most common pathogen found in adult patients with CF. Patients with CF are at high risk of having MDR bacterial isolates. They require higher doses of antibiotics and shorter dosing intervals than other patients because of differences in the volume of drug distribution and the rate of elimination.

Airway Clearance

Mucolytic agents are routinely administered to facilitate clearance of mucus. Recombinant human DNase (Pulmozyme) is the drug of choice. It decreases the viscosity of sputum by catalyzing extracellular DNA into smaller fragments. Chest physiotherapy is used to increase airway clearance. Bronchodilators are routinely prescribed and administered before chest physiotherapy to increase airway clearance.

Nutritional Support

Enteral nutrition with pancreatic enzyme supplements, if needed, is started early in the course of treatment.[16,35]

Ventilatory Support

If ventilator support is necessary, noninvasive mechanical ventilation is the first line of therapy. Endotracheal intubation with mechanical ventilation is the next step. The goal of mechanical ventilation is the same as with any patient with ARF. Adult patients with CF are at high risk for pneumothorax and massive hemoptysis. The critical care nurse must be aware of these life-threatening complications, constantly monitoring for them, and respond quickly.

CASE STUDY

Mrs. P. is a 57-year-old woman admitted to the critical care unit after a motor vehicle crash. She sustained multiple long bone fractures and a chest contusion, and experienced an episode of hypotension in the emergency department. She received 3 units of red blood cells and 2 L of intravenous fluid in the emergency department. Within 12 hours she became short of breath with an increase in respiratory rate requiring high levels of supplemental oxygen. She was electively intubated and placed on volume-control mechanical ventilation with a positive end-expiratory pressure (PEEP) of 5 cm H_2O. A continuous intravenous sedation infusion was started. The decision was made to titrate the infusion to keep her calm and comfortable. During the next 8 hours, her oxygen saturation by pulse oximetry (SpO_2) steadily deteriorated, and the high-pressure alarms on the ventilator activated frequently. The nurse noted steadily rising peak airway pressures. The fraction of inspired oxygen (FiO_2) had to be increased to 0.80 and the PEEP increased to 14 cm H_2O to maintain her partial pressure of oxygen in arterial blood (PaO_2) at 60 mm Hg. Her chest x-ray study showed bilateral infiltrates with normal heart size. A pulmonary artery catheter was inserted with an initial pulmonary artery occlusion pressure of 14 mm Hg. The sedation infusion required frequent upward titrations to maintain the desired goal of light sedation. The diagnosis of acute respiratory distress syndrome (ARDS) was made.

During the next 6 hours, Mrs. P. steadily became more hypoxemic. She was changed to pressure-controlled ventilation with a PEEP of 20 cm H_2O. The FiO_2 had to be increased to 1.0 (100%) to maintain a PaO_2 of greater than 60 mm Hg. She was extremely restless, with tachycardia, diaphoresis, and a labile SaO_2. The decision was made to start a neuromuscular blocking agent with sedation. During the next few hours her general condition continued to deteriorate. Her SaO_2 ranged from 85% to 87%. Her chest x-ray findings were worse and revealed a complete *whiteout*. The nurses and physicians decided to turn her to the prone position in an effort to improve oxygenation. An hour after turning her to the prone position, her SpO_2 began to slowly rise. After 2 hours in the prone position, her SpO_2 stabilized at 93%. Slowly, the FiO_2 was decreased to 0.60, with a stable SpO_2 of 92%. After 18 hours she was returned to the supine position. Her SpO_2 decreased to 90% and it remained stable. She was weaned off the neuromuscular blocking agent, and the sedation level was reduced to reach a goal of calm and comfortable.

Mrs. P. slowly improved over the next week. Her ventilator settings were changed from pressure-control to assist-control then to pressure support (PS). The PEEP level was decreased to a physiological level. The sedation was interrupted on a daily basis for weaning parameters and spontaneous breathing trial. On the seventh day, she was extubated and the following day transferred to the general orthopedic nursing unit on 4 liters of oxygen per nasal cannula.

Questions

1. Identify the risk factors Mrs. P. had for developing ARDS.
2. The American-European Consensus Conference recommended three criteria for diagnosing ARDS in the presence of a risk factor. List the criteria.
3. Explain the use of the high PEEP and the nursing monitoring responsibilities.
4. Explain the rationale for the use of sedation and neuromuscular blocking agents and what nursing interventions should occur when using these agents.
5. Explain the rationale for placing the patient in the prone position and what nursing interventions should occur before and after turning a patient to the prone position.

SUMMARY

ARF is a disorder that can affect all segments of the population, from young trauma patients to elderly persons with long-standing pulmonary disease. Patients in the critical care areas are at high risk of ARF. The critical care nurse must be constantly alert to signs of impending respiratory failure. Changes in respiratory rate and character, breath sounds, and blood gas values must be closely evaluated. Frequent position changes, good pulmonary hygiene, and careful attention to nutritional status all contribute to maintaining a patient's respiratory system and preventing ARF.

CRITICAL THINKING EXERCISES

1. Mr. R. is a 66-year-old man who has smoked 1.5 packs of cigarettes a day for 40 years (60 pack-years). He is admitted with an acute exacerbation of COPD. His baseline ABGs drawn in the clinic 2 weeks ago showed: pH, 7.36; $PaCO_2$, 55 mm Hg; PaO_2, 69 mm Hg; bicarbonate, 30 mEq/L; SaO_2, 92%. In the critical care unit, Mr. R. has coarse crackles in his left lower lung base and a mild expiratory wheeze bilaterally. His cough is productive of thick yellow sputum. His skin turgor is poor; he is febrile, tachycardic, and tachypneic. Currently, Mr. R.'s ABGs while receiving O_2 at 2 L/min via a nasal cannula are: pH, 7.32; $PaCO_2$, 64 mm Hg; PaO_2, 50 mm Hg; bicarbonate, 30 mEq/L; SaO_2, 86%.
   a. What is your interpretation of Mr. R.'s baseline ABGs from the clinic?
   b. What is the probable cause of Mr. R.'s COPD exacerbation, and what treatment is indicated at this time?
   c. What ABG changes would indicate that Mr. R.'s respiratory status is deteriorating?
2. Ms. T. is a 41-year-old woman admitted to the critical care unit and mechanically ventilated for acute asthma. She was extubated yesterday and will be transferred out of the critical care unit tomorrow. What are the important points you must cover in your teaching with Ms. T.?
3. Mr. B. has just been intubated for ARF. Currently, he is agitated and very restless. What risks are associated with Mr. B.'s agitation? What nursing actions are indicated in this situation?
4. Mr. C., age 27 years, was hospitalized 3 days ago after fracturing his femur in a snow-skiing accident. He has just been admitted to the critical care unit with a PE and is orally intubated and receiving mechanical ventilation. What actions would you take to decrease Mr. C.'s risk of developing VAP?

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