CHAPTER

9

Ventilatory Assistance

Lynelle N. B. Pierce, MSN, RN, CCRN

evolve WEBSITE

Many additional resources, including self-assessment exercises, are located on the Evolve companion website at http://evolve.elsevier.com/Sole.

- Review Questions
- Mosby's Nursing Skills Procedures
- Animations
- Video Clips

INTRODUCTION

Essential nursing interventions for all patients include maintaining an adequate airway, and ensuring adequate breathing (ventilation) and oxygenation. These nursing interventions provide the framework for this chapter. Respiratory anatomy and physiology are reviewed to provide a basis for discussing ventilatory assistance. Assessment of the respiratory system includes physical examination, arterial blood gas (ABG) interpretation, and noninvasive methods for assessing gas exchange. Airway management, oxygen therapy, and mechanical ventilation, important therapies in the critical care unit, are also discussed.

REVIEW OF RESPIRATORY ANATOMY AND PHYSIOLOGY

The primary function of the respiratory system is gas exchange. Oxygen and carbon dioxide are exchanged via the respiratory system to provide adequate oxygen to the cells and to remove carbon dioxide, the by-product of metabolism, from the cells. The respiratory system is divided into (1) the upper airway, (2) the lower airway, and (3) the lungs.[49] The upper airway conducts gas to and from the lower airway, and the lower airway provides gas exchange at the alveolar-capillary membrane. The anatomical structure of the respiratory system is shown in Figure 9-1.

Upper Airway

The upper airway consists of the nasal cavity and the pharynx. The nasal cavity conducts air, filters large foreign particles, and warms and humidifies air. When an artificial airway is placed, these natural functions of the airway are bypassed. The nasal cavity also is responsible for voice resonance, smell, and sneeze reflexes. The throat, or pharynx, transports both air and food.

Lower Airway

The lower airway consists of the larynx, trachea, right and left mainstem bronchi, bronchioles, and alveoli. The larynx is the narrowest part of the conducting airways in adults and contains the vocal cords. The larynx is partly covered by the epiglottis, which prevents aspiration of food, liquid, or saliva into the lungs during swallowing. The passage through the vocal cords is the glottis (Figure 9-2).

The trachea warms, humidifies, and filters air. Cilia in the trachea propel mucus and foreign material upward through the airway. At about the level of the fifth thoracic vertebra (sternal angle, or angle of Louis), the trachea branches into the right and left mainstem bronchi, which conduct air to the respective lungs. This bifurcation is referred to as the *carina*. The right mainstem bronchus is shorter, wider, and straighter than the left. The bronchi further branch into the bronchioles and finally the terminal bronchioles, which supply air to the alveoli. Mucosal cells in the bronchi secrete mucus that lubricates the airway and traps foreign materials, which are moved by the cilia upward to be expectorated or swallowed.

The alveoli are the distal airway structures and are responsible for gas exchange at the capillary level. The alveoli consist of a single layer of epithelial cells and fibers that permit expansion and contraction. The type II cells inside the alveolus secrete surfactant, which coats the inner surface and prevents it from collapsing. A network of pulmonary capillaries covers

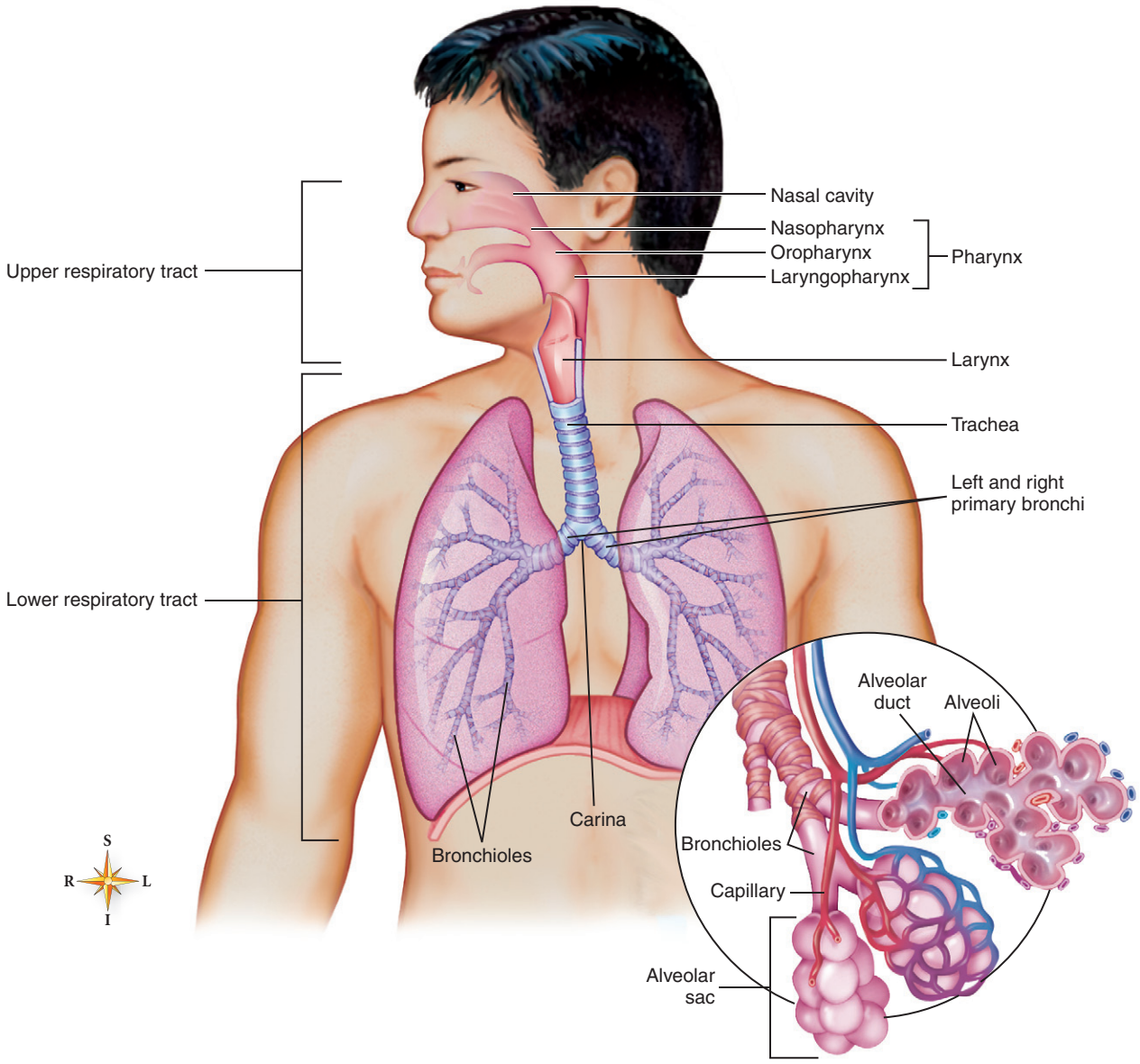

FIGURE 9-1 Anatomy of the upper and lower respiratory tracts. The inset shows the grapelike clusters of alveoli and their rich blood supply, which supports the exchange of oxygen and carbon dioxide. (From Patton KT and Thibodeau GA. *Anatomy and Physiology.* 7th ed. St. Louis: Mosby; 2010.)

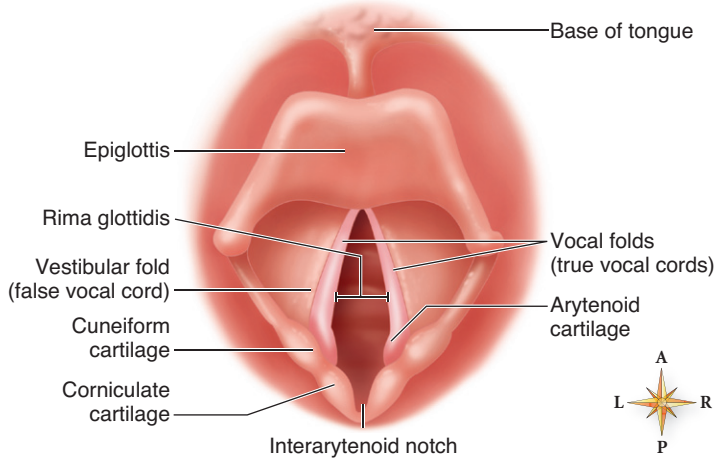

FIGURE 9-2 The vocal cords and glottis. (From Patton KT, Thibodeau GS. *Anatomy and Physiology.* 7th ed. St. Louis: Mosby; 2010.)

the alveoli. Gas exchange occurs between the alveoli and these capillaries.[49,61] The large combined surface area and single cell layer of the alveoli promote very efficient diffusion of gases.

Lungs

The lungs consist of lobes; the left lung has two lobes, and the right lung has three lobes. Each lobe consists of lobules, or segments, that are supplied by one bronchiole. The top of each lung is the apex, and the lower part of the lung is the base.

The lungs are covered by pleura. The visceral pleura cover the lung surfaces, whereas the parietal pleura cover the internal surface of the thoracic cage. Between these two layers the pleural space is formed, which contains pleural fluid. This thin fluid lubricates the pleural layers as they slide across each other during breathing. It also holds the two pleurae together because it creates surface tension, an attractive force between liquid molecules. It is this surface tension between the two pleurae, opposing the tendency of the elastic lung to want to collapse, that leads to a pressure of negative 5 cm H_2O within the pleural space.[27] In disorders of the pleural space, such as pneumothorax, this negative pressure is disrupted, leading to collapse of the lung and the need for a chest tube.

PHYSIOLOGY OF BREATHING

The basic principle behind the movement of gas in and out of the lung is that gas travels from an area of higher to lower pressure. During inspiration, the diaphragm lowers and flattens and the intercostal muscles contract, lifting the chest up and outward to increase the size of the chest cavity. Subsequently, intrapleural pressure becomes even more negative than stated earlier, and intraalveolar pressure (the pressure in the lungs) becomes negative, causing air to flow into the lungs *(inspiration)*.[27] Expiration is a passive process in which the diaphragm and intercostal muscles relax and the lungs recoil. This recoil generates positive intraalveolar pressure relative to atmospheric pressure, and air flows out of the lungs *(expiration)*.[69]

Gas Exchange

The process of gas exchange (Figure 9-3) consists of four steps: (1) ventilation, (2) diffusion at pulmonary capillaries,

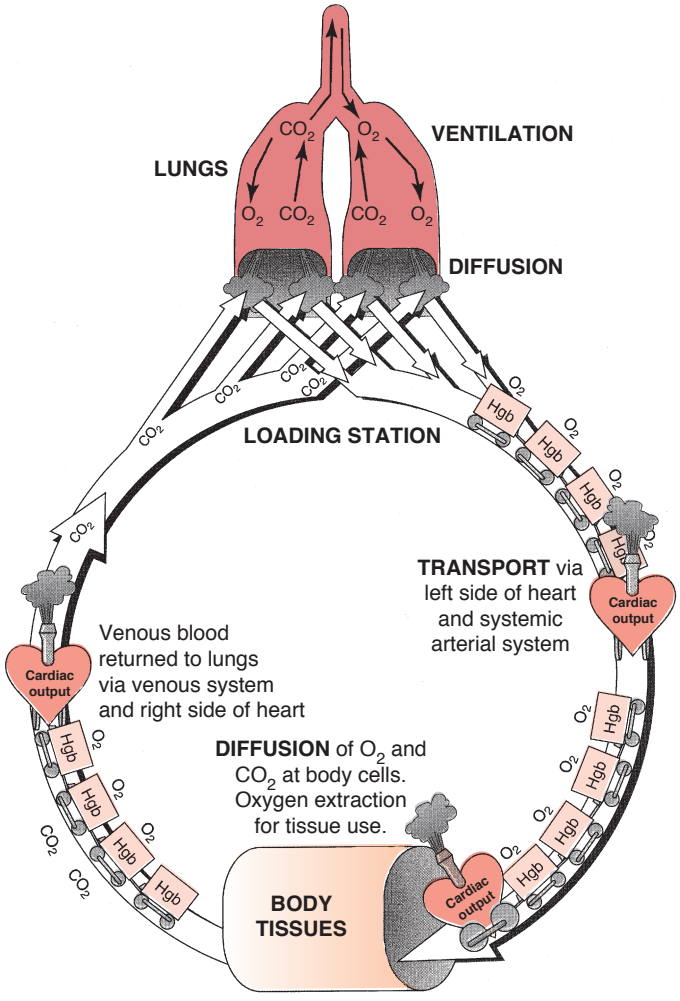

FIGURE 9-3 Schematic view of the process of gas exchange. *Hgb,* Hemoglobin. (Modified from Alspach J. *AACN Instructor's Resource Manual for AACN Core Curriculum for Critical Care Nursing.* 4th ed. Philadelphia: Saunders; 1992.)

(3) perfusion (transportation), and (4) diffusion to the cells.[27,69,70]

1. Ventilation is the movement of gases (oxygen and carbon dioxide) in and out of the alveoli.
2. Diffusion of oxygen and carbon dioxide occurs at the alveolar-capillary membrane (Figure 9-4). The driving force to move the gas from the alveoli to the capillary and vice versa is the pressure of the gases across the alveolar-capillary membrane. Diffusion is the movement of gas molecules from an area of higher to lower pressure. Oxygen pressure is higher in the alveoli than in the capillaries, thus promoting oxygen diffusion from the alveoli into the blood. Carbon dioxide pressure is higher in the capillaries, thus promoting the diffusion of carbon dioxide into the alveoli for elimination during exhalation.
3. The oxygenated blood in the pulmonary capillary is transported via the pulmonary vein to the left side of the heart. The oxygenated blood is perfused and transported to the tissues.
4. Diffusion of oxygen and carbon dioxide occurs at the cellular level based on pressure gradients. Oxygen diffuses from blood into the cells, and carbon dioxide leaves the cells and diffuses into the blood in a process called internal respiration. Carbon dioxide is transported via the vena cava to the right side of the heart and into the pulmonary capillaries where it diffuses into the alveoli and is eliminated through exhalation.

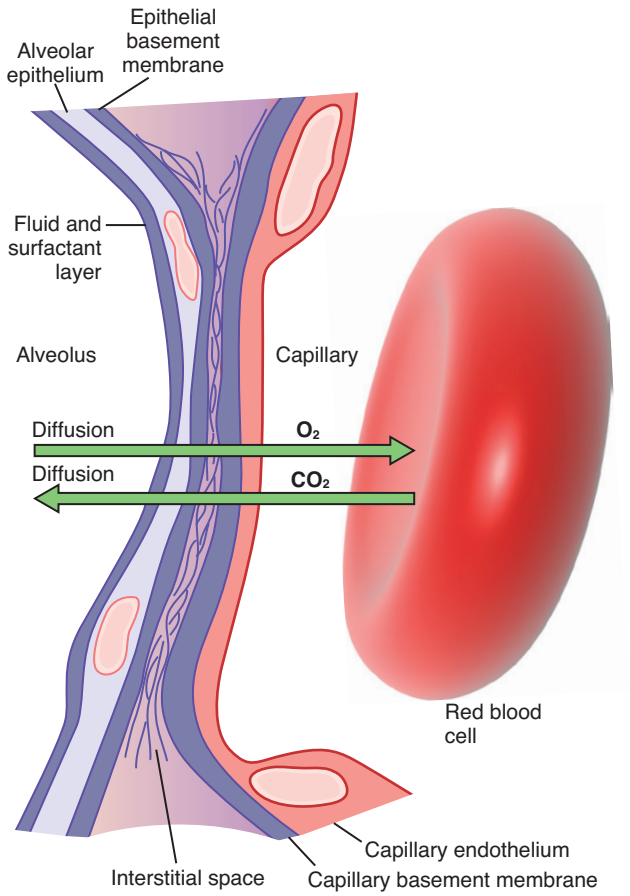

FIGURE 9-4 Diffusion of oxygen and carbon dioxide at the alveolar-capillary membrane. (From Hall JE, Guyton AC. *Guyton and Hall Textbook of Medical Physiology.* 12th ed. Philadelphia: Saunders; 2011.)

Regulation of Breathing

The rate, depth, and rhythm of ventilation are controlled by respiratory centers in the medulla and pons. When the carbon dioxide level is high or the oxygen level is low, chemoreceptors in the respiratory center, carotid arteries, and aorta send messages to the medulla to regulate respiration. In persons with normal lung function, high levels of carbon dioxide stimulate respiration. However, patients with chronic obstructive pulmonary disease (COPD) maintain higher levels of carbon dioxide as a baseline, and their ventilatory drive in response to increased carbon dioxide levels is blunted. In these patients, the stimulus to breathe is hypoxemia, a low level of oxygen in the blood.[27]

Respiratory Mechanics

Work of Breathing

The work of breathing (WOB) is the amount of effort required for the maintenance of a given level of ventilation. When the lungs are not diseased, the respiratory muscles manage the WOB using unlabored respirations. The respiratory pattern changes automatically to manage an increased WOB when lung disease is present, and the patient may use accessory muscles. As the WOB increases, more energy must be expended to obtain adequate ventilation, which requires proportionately more oxygen and glucose. If the WOB becomes too high, respiratory failure ensues and mechanical ventilatory support is warranted.[51,53]

Compliance

Compliance is a measure of the distensibility, or stretchability, of the lung and chest wall. The lungs are primarily made up of elastin and collagen fibers that in disease states become less elastic, leading to so-called stiff lungs. *Distensibility* refers to how easily the lung is stretched when the respiratory muscles work and expand the thoracic cavity. Compliance, a clinical measurement of the lung's distensibility, is defined as the change in lung volume per unit of pressure change.[51,69]

Various pathological conditions such as pulmonary fibrosis, acute respiratory distress syndrome (ARDS), and pulmonary edema lead to low pulmonary compliance. In these situations, the patient must generate more work to breathe to create negative pressure to inflate the stiff lungs. Compliance is also decreased in obesity secondary to the increased mass of the chest wall.

In emphysema, destruction of lung tissue and enlarged air spaces cause the lungs to lose their elasticity, which increases compliance. The lungs are more distensible in this situation, require lower pressures for ventilation, and may collapse during expiration, causing air to become trapped in the distal airways.

Monitoring changes in compliance provides an objective clinical indicator of changes in the patient's lung condition

and ability to ventilate, especially the mechanically ventilated patient with decreased lung compliance. Compliance of the lung tissue is best measured under static conditions (no airflow), and is achieved by instituting a 2-second inspiratory hold maneuver with the mechanical ventilator.[51,53] Static compliance in patients with normal lungs usually ranges from 50 to 170 mL/cm H_2O.[69] This means that for every 1–cm H_2O change of pressure in the lungs, the volume of gas increases by 50 to 170 mL. A single measurement of compliance is not useful in monitoring patient progress; it is important to trend compliance over time.

Dynamic compliance is measured while gases are flowing during breathing; it measures not only lung compliance but also airway resistance to gas flow. The normal value for dynamic compliance is 50 to 80 mL/cm H_2O.[69] Dynamic compliance is easier to measure because it does not require breath holding or an inspiratory hold; however, it is not a pure measurement of lung compliance. A decrease in dynamic compliance may signify a decrease in compliance or an increase in resistance to gas flow.

The respiratory therapist (RT) or nurse measures compliance in the mechanically ventilated patient to identify trends in the patient's condition. Compliance can easily be obtained on most modern ventilators when the operator requests it using the menu options. Poor compliance requires higher ventilatory pressures to achieve adequate lung volume. Higher ventilatory pressures place the patient at increased risk for complications, such as volutrauma.

Resistance

Resistance refers to the opposition to the flow of gases in the airways. Factors that affect airway resistance are airway length, airway diameter, and the flow rate of gases. Airway resistance is increased when the airway is lengthened or narrowed, as with an artificial airway, or when the natural airway is narrowed by spasms (bronchoconstriction), the presence of mucus, or edema. Finally, resistance increases when gas flow is increased, as with increased breathing effort or when a patient requires mechanical ventilation. When resistance increases, more effort is required by the patient to maintain gas flow. If the patient is unable to generate the increased WOB, the amount of gas flow the patient produces decreases. Thus increasing airway resistance may result in reduced lung volume and inadequate ventilation.[27,69]

LUNG VOLUMES AND CAPACITIES

Air volume within the lung is measured with an instrument called a *spirometer*. Lung volumes and capacities (two or more lung volumes added together) are important for determining adequate pulmonary function, and are shown graphically in Figure 9-5. Descriptions of the lung volumes and capacities are provided in Table 9-1. Measurements of lung volumes and capacities allow the practitioner to assess baseline pulmonary function and to monitor the improvement or progression of pulmonary diseases and patient response to therapy. For example, when the patient performs incentive spirometry, the nurse and RT assess the patient's inspiratory capacity and trend its improvement or decline over time and with interventions. Lung capacities decline gradually with aging.

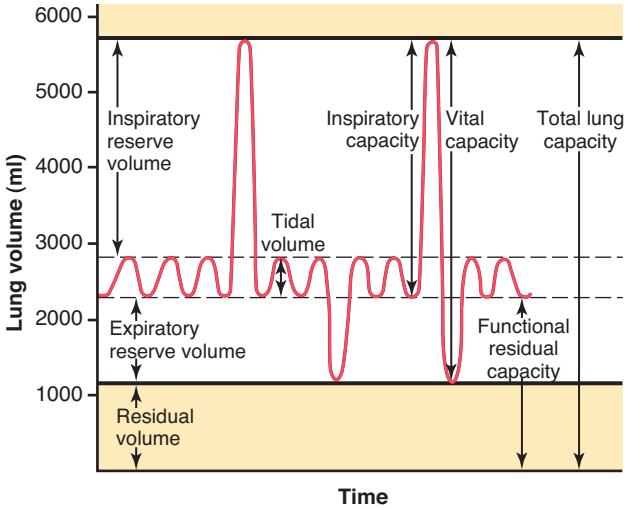

FIGURE 9-5 Lung volumes and capacities. (From Hall JE, Guyton AC. *Guyton and Hall Textbook of Medical Physiology* 12th ed. Philadelphia: Saunders; 2011.)

RESPIRATORY ASSESSMENT

The ability to perform a physical assessment of the respiratory system is an essential skill for the critical care nurse. Assessment findings assist in identifying potential patient problems and in evaluating patient response to interventions. See the box "Geriatric Considerations" for information related to assessment of elderly patients and the box "Bariatric Considerations" for information related to assessment of the obese patient.

Health History

Several questions pertinent to the respiratory system should be asked when the health history is obtained:

1. Tobacco use: type, amount, and number of pack-years (number of packs of cigarettes per day × number of years smoking)
2. Occupational history such as coal mining, asbestos work, farming, and exposure to dust, fumes, smoke, toxic chemicals, paints, and insulation
3. History of symptoms such as shortness of breath, dyspnea, cough, anorexia, weight loss, chest pain, or sputum production; further assessment of sputum, including amount, color, consistency, time of day, and whether its appearance is chronic or acute
4. Use of oral and inhalant respiratory medications, such as bronchodilators and steroids
5. Use of over-the-counter or street inhalant drugs
6. Allergies: medication, food, or environmental
7. Dates of last chest radiograph and tuberculosis screening

TABLE 9-1 LUNG VOLUMES AND CAPACITIES

NAME	DEFINITION	AVERAGE	FORMULA
Volumes*			
Tidal volume (V_T)	Volume of a normal breath	500 mL	
Inspiratory reserve volume (IRV)	Maximum amount of gas that can be inspired at the end of a normal breath (over and above the V_T)	3000 mL	
Expiratory reserve volume (ERV)	Maximum amount of gas that can be forcefully expired at the end of a normal breath	1200 mL	
Residual volume (RV)	Amount of air remaining in the lungs after maximum expiration	1300 mL	
Capacities			
Inspiratory capacity (IC)	Maximum volume of gas that can be inspired at normal resting expiration; the IC distends the lungs to their maximum amount	3500 mL	$IC = V_T + IRV$
Functional residual capacity (FRC)	Volume of gas remaining in the lungs at normal resting expiration	2500 mL	$FRC = ERV + RV$
Vital capacity (VC)	Maximum volume of gas that can be forcefully expired after maximum inspiration	4700 mL	$VC = V_T + IRV + ERV$
Total lung capacity (TLC)	Volume of gas in the lungs at end of maximum inspiration	6000 mL	$TLC = V_T + ERV + RV$

*Volumes are average in a 70-kg young adult. There is a range of normal values that varies by age, height, body size, and gender. Volumes are less in women than men when height and age are equal.

GERIATRIC CONSIDERATIONS

Physiological Changes with Aging

- ↓ Chest wall distensibility (costal cartilage calcifies)
- ↓ Alveolar surface area (enlarged alveoli)
- ↓ Alveolar elasticity
- ↓ Lung volume
- ↓ Diffusing capacity
- ↓ Physiological compensatory mechanisms in response to hypercapnea or hypoxia
- Weaker respiratory muscles
- Decreased cough and gag

Assessment Changes

Normal Findings Because of Aging Process

- Kyphosis
- Barrel chest
- ↓ Chest expansion
- Lower PaO_2 levels on ABG

Increased Risk for

- Secretion retention and pneumonia
- Poor gas exchange
- Mental status changes as an early sign of gas exchange problems
- Aspiration
- Respiratory distress
- Respiratory failure

ABG, Arterial blood gas; *PaO_2,* partial pressure of oxygen in arterial blood.

Data from Miller CA. (2012). Respiratory function. In CA Miller (Ed.) *Nursing for Wellness in Older Adults,* 6th ed. Philadelphia: Lippincott, Williams & Wilkins; West JB. (2011). *Respiratory Physiology: The Essentials* (9th ed.). Baltimore, MD: Lippincott Williams & Wilkins.

BARIATRIC CONSIDERATIONS

Physiological Changes with Obesity

- Diaphragm is cephaloid-displaced, especially in the supine position, resulting in ↓ lung volumes
- Collapse of small airways and alveoli
- Decreased lung compliance and increased airway resistance
- Fatty infiltration of respiratory muscles
- Increased oxygen consumption to perform the work of breathing due to increased mass of thorax
- Increased fat distribution within the soft tissue of the airway
- Increased fat distribution in the neck, causing redundant skin folds

Assessment Changes Related to Obesity

- Increased respiratory rate
- Decreased breath sounds
- ↓ Chest expansion
- Snoring
- Dyspnea at rest or with talking
- Lower PaO_2 levels on ABG
- Decreased quality of portable CXR

Increased Risk for

- Atelectasis
- Poor gas exchange
- Respiratory distress
- Respiratory failure
- Obesity hypoventilation syndrome (OHS), also known as obstructive sleep apnea
- Difficult intubation
- Difficult ventilation with a bag-valve-mask device
- Complications related to tracheostomy

ABG, Arterial blood gas; *CXR,* chest x-ray; *PaO_2,* partial pressure of oxygen in arterial blood.
From Siela D, (2009). Pulmonary aspects of obesity in critical care. *Critical Care Nursing Clinics of North America,* 21(3), 301-310.

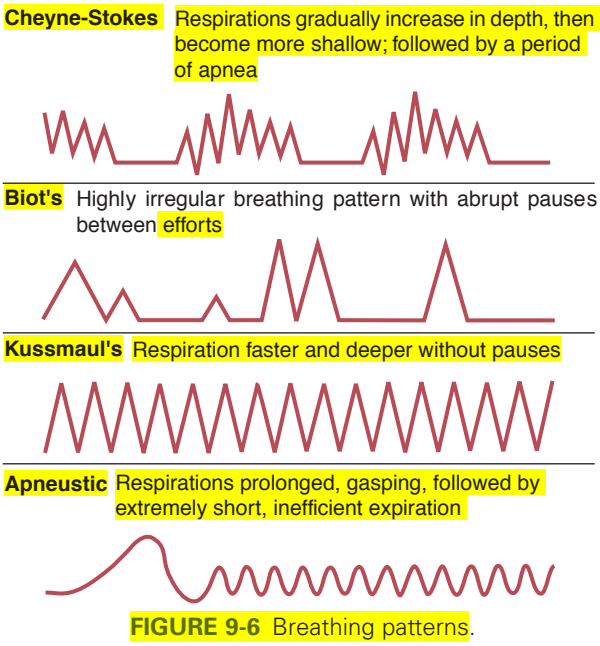

FIGURE 9-6 Breathing patterns.

Physical Examination

Inspection

Inspection provides an initial clue for potential acute and chronic respiratory problems. The head, neck, fingers, and chest are inspected for abnormalities.

The chest is observed for shape, breathing pattern, and chest excursion. During inspiration, chest wall excursion should be symmetrical. Asymmetrical excursion is usually associated with unilateral ventilation problems. The trachea is normally in a midline position; a tracheal shift may occur with a tension pneumothorax. Signs of acute respiratory distress include labored respirations, irregular breathing pattern, use of accessory muscles, asymmetrical chest movements, chest-abdominal asynchrony, open-mouthed breathing, or gasping breaths. Cyanosis is a late sign of hypoxemia and should not be relied on as an early warning of distress. Other indications of respiratory abnormalities include pallor or rubor, pursed-lip breathing, jugular venous distention, prolonged expiratory phase of breaths, poor capillary refill, clubbing of fingers, and a barrel-shaped chest.[68]

The respiratory rate (RR) should be counted for a full minute in critically ill patients. The normal RR is 12 to 20 breaths per minute, and expiration is usually twice as long as inspiration (inspiration-to-expiration ratio is 1:2). The normal breathing pattern is regular and even with an occasional sigh, and is called *eupnea. Tachypnea,* a RR of greater than 20 breaths per minute, may occur with anxiety, fever, pain, anemia, low PaO_2, and elevated $PaCO_2$. *Bradypnea,* a RR of less than 10 breaths per minute may occur in central nervous system disorders including administration or ingestion of central nervous system depressant medications or alcohol, severe metabolic alkalosis, and fatigue. The depth of respirations is as important as the rate and provides information about the adequacy of ventilation. Alterations from normal rate and depth should be documented and reported.

Several abnormal breathing patterns (Figure 9-6) are possible and should be reported.[68] *Cheyne-Stokes respirations* have a cyclical respiratory pattern. Deep, increasingly shallow respirations are followed by a period of apnea that lasts approximately 20 seconds, but the period may vary and progressively lengthen. Therefore, the duration of the apneic period is timed for trending. The cycle repeats after each apneic period. Cheyne-Stokes respirations may occur in central nervous system disorders and congestive heart failure. *Biot's respirations,* or cluster breathing, are cycles of breaths that vary in depth and have varying periods of apnea. Biot's respirations are seen with brainstem injury. *Kussmaul's respirations* are deep, regular, and rapid (usually more than 20 breaths per minute), and are commonly observed in diabetic ketoacidosis

and other disorders that cause metabolic acidosis. *Apneustic respirations* are gasping inspirations followed by short, ineffective expirations. They are often associated with lesions to the pons.

Palpation

Palpation is frequently performed simultaneously with inspection. Palpation is used to evaluate chest wall excursion, tracheal deviation, chest wall tenderness, subcutaneous crepitus, and tactile fremitus. The chest wall should not be tender to palpation; tenderness is usually associated with inflammation or trauma, including rib fractures. *Subcutaneous crepitus* or *subcutaneous emphysema* is the presence of air beneath the skin surface that has escaped from the airways or lungs. It is palpated with the fingertips and may feel like crunching Rice Krispies under the skin. The temptation to further palpate should be resisted, because palpation promotes air dissection in the skin layers. Subcutaneous air may result from chest trauma, such as rib fractures, and from barotrauma. It indicates that the lungs or airways are not intact.

Percussion

The chest may be percussed to identify respiratory disorders such as hemothorax, pneumothorax, and consolidation. In percussion, the middle finger of one hand is tapped twice by the middle finger of the opposite hand placed against the patient's chest. The vibrations produced by tapping create different sounds, depending on the density of the underlying tissue being percussed. Five sounds may be audible on percussion: resonance (normal), dullness (tissue more dense than normal as in consolidation), flatness (absence of air as in lung collapse), hyperresonance (increased amount of air as in emphysema), and tympany (large amount of air as in pneumothorax).[68]

Auscultation

Lung sounds are routinely assessed every 4 hours in critically ill patients using the diaphragm of the stethoscope pressed firmly against the chest wall. The stethoscope should be placed directly on the patient's chest; sounds are difficult to distinguish if they are auscultated through the patient's gown or clothing. The friction of chest hair on the stethoscope may mimic the sound of crackles; wetting the chest hair may reduce this sound. In addition, the stethoscope tubing should not rest against skin or objects such as sheets, bed rails, or ventilator circuitry during auscultation.[51]

A systematic sequence should be used during auscultation, with sounds from one side of the chest wall compared with those from the other (Figure 9-7). Auscultation is best performed with the patient sitting in an upright position and breathing deeply in and out through the mouth. It may not be feasible for a critically ill patient to assume a sitting position for auscultation. In this circumstance, auscultation of the anterior and lateral chest is often performed. However, every opportunity should be taken to turn the patient and auscultate the chest posteriorly. When the patient has an artificial airway, the trachea should be auscultated for the presence of an air leak.

Breath Sounds

The nurse listens carefully for both normal and abnormal, or *adventitious,* breath sounds. Types of normal breath sounds include bronchial (larynx, trachea), bronchovesicular (large central airways), and vesicular (smaller airways). Adventitious sounds the nurse must be familiar with and able to report include crackles, rhonchi, wheezes, pleural friction rub, and stridor (Table 9-2). Breath sounds may be decreased because of the presence of fluid, air, or increased tissue density. Shallow respirations can also mimic decreased breath

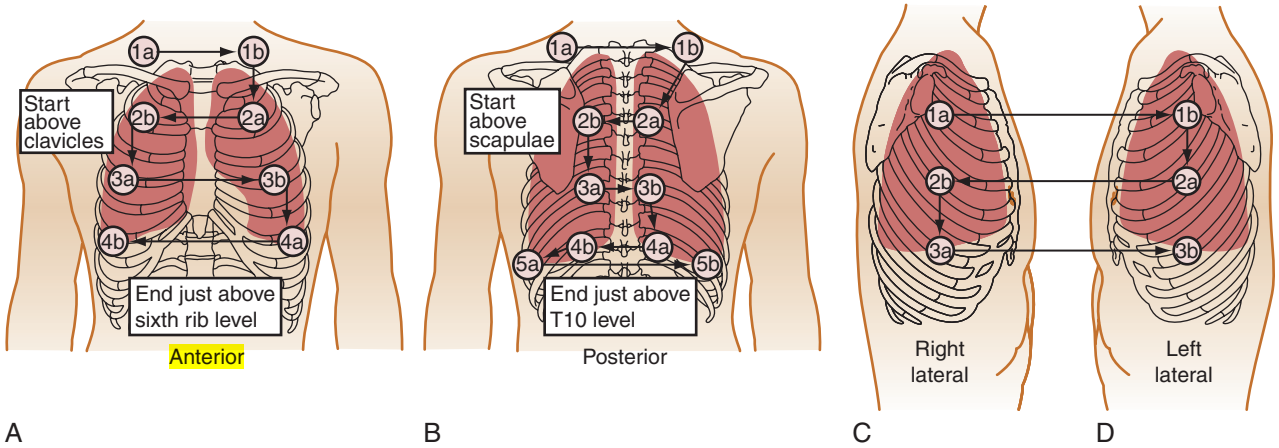

FIGURE 9-7 Systematic method for palpation, percussion, and auscultation of the lungs in anterior **(A),** posterior **(B),** and lateral regions **(C** and **D).** The techniques should be performed systematically to compare right and left lung fields.

TABLE 9-2 ADVENTITIOUS BREATH SOUNDS

SOUND/DESCRIPTION	CAUSE	CLINICAL SIGNIFICANCE	ADDITIONAL DESCRIPTORS/ COMMENTS
Crackles—discontinuous, explosive, bubbling sounds of short duration	Air bubbling through fluid or mucus, or alveoli popping open on inspiration	Atelectasis, fluid retention in small airways (pulmonary edema), retention of mucus (bronchitis, pneumonia), interstitial fibrosis	Fine: soft, short duration Coarse: loud, longer duration Wet or dry May disappear after coughing, suctioning, or deep inspiration if alveoli remain inflated
Rhonchi—coarse, continuous, low-pitched, sonorous, or rattling sound	Air movement through excess mucus, fluid, or inflamed airways	Diseases resulting in airway inflammation and excess mucus (e.g., pneumonia, bronchitis, or excess fluid, as in pulmonary edema)	Inspiratory and/or expiratory; may clear or diminish with coughing if caused by airway secretions
Wheezes—high- or low-pitched whistling, musical sound heard during inspiration and/or expiration	Air movement through narrowed airway, which causes airway wall to oscillate or flutter	Bronchospasm, as in asthma, partial airway obstruction by tumor, foreign body or secretions, inflammation, or stenosis	High or low pitched; inspiratory and/or expiratory
Stridor—high-pitched, continuous sound heard over upper airway; a crowing sound	Air flowing through constricted larynx or trachea	Partial obstruction of upper airway, as in laryngeal edema, obstruction by foreign body, epiglottitis	Potentially life-threatening
Pleural friction rub—coarse, grating, squeaking, or scratching sound, as when two pieces of leather rub together	Inflamed pleura rubbing against each other	Pleural inflammation, as in pleuritis, pneumonia, tuberculosis, chest tube insertion, pulmonary infarction	Occurs during breathing cycle and is eliminated by breath holding Need to discern from pericardial friction rub, which continues despite breath holding

sounds; therefore the patient must take deep breaths during auscultation. The breath sounds should be carefully documented and abnormalities reported.

Arterial Blood Gas Interpretation

The ability to interpret ABG results rapidly is an essential critical care skill. ABG results reflect oxygenation, adequacy of gas exchange, and acid-base status. Blood for ABG analysis is obtained from either a direct arterial puncture (radial, brachial, or femoral artery) or an arterial line. ABGs aid in patient assessment and must be interpreted in conjunction with the patient's physical assessment findings, clinical history, and previous ABG values (Table 9-3). Noninvasive measures of gas exchange have reduced the frequency of ABG measurements.

Oxygenation

The ABG values that reflect oxygenation include the partial pressure of oxygen dissolved in arterial blood (PaO_2) and the arterial oxygen saturation of hemoglobin (SaO_2). Approximately 3% of the available oxygen is dissolved in plasma. The remaining 97% of the oxygen attaches to hemoglobin in red blood cells, forming oxyhemoglobin.[69]

Partial pressure of arterial oxygen. The normal PaO_2 is 80 to 100 mm Hg at sea level. The PaO_2 decreases in the elderly; the value for persons 60 to 80 years of age usually ranges from 60 to 80 mm Hg.

Arterial oxygen saturation of hemoglobin. The SaO_2 is the percentage of hemoglobin saturated with oxygen and is normally 92% to 99%. The SaO_2 is very important because it represents the primary way oxygen is transported to the tissues. The SaO_2 is measured directly from an arterial blood sample or continuously monitored indirectly with the use of a pulse oximeter (SpO_2).

Both the PaO_2 and the SaO_2 are used to assess oxygenation. Decreased oxygenation of arterial blood (PaO_2 <60 mm Hg) is referred to as *hypoxemia*, which may present with numerous symptoms described in Box 9-1. A patient with a PaO_2 of less than 60 mm Hg requires immediate intervention with supplemental oxygen to treat the hypoxemia while further assessment is done to identify the cause. A PaO_2 of less than 40 mm Hg is life-threatening because oxygen is not available for metabolism. Without treatment, cellular death will occur.[27,70]

The relationship between the PaO_2 and the SaO_2 is shown in the S-shaped *oxyhemoglobin dissociation curve* (Figure 9-8). The upper portion of the curve (PaO_2 >60 mm Hg) is flat. In this area of the curve, large changes in the PaO_2 result in only small changes in SaO_2. For example, the normal PaO_2 of 80 to 100 mm Hg is associated with an SaO_2 of 92% to 100%. If the PaO_2 decreases from 80 to 60 mm Hg, the SaO_2 decreases from 92% to 90%. Although this example reflects a drop in PaO_2, the patient is not immediately compromised,

TABLE 9-3 BLOOD GAS INTERPRETATION

STATUS	pH	PCO_2	HCO_3^-	BASE EXCESS
Respiratory Acidosis				
Uncompensated	↓ 7.35	↑ 45	Normal	Normal
Partially compensated	↓ 7.35	↑ 45	↑ 26	↑ +2
Compensated	7.35-7.45	↑ 45	↑ 26	↑ +2
Respiratory Alkalosis				
Uncompensated	↑ 7.45	↓ 35	Normal	Normal
Partially compensated	↑ 7.45	↓ 35	↓ 22	↓ −2
Compensated	7.40-7.45	↓ 35	↓ 22	↓ −2
Metabolic Acidosis				
Uncompensated	↓ 7.35	Normal	↓ 22	↓ −2
Partially compensated	↓ 7.35	↓ 35	↓ 22	↓ −2
Compensated	7.35-7.45	↓ 35	↓ 22	↓ −2
Metabolic Alkalosis				
Uncompensated	↑ 7.45	Normal	↑ 26	↑ +2
Partially compensated*	↑ 7.45	↑ 45	↑ 26	↑ +2
Compensated*	7.40-7.45	↑ 45	↑ 26	↑ +2
Combined Respiratory and Metabolic Acidosis	↓ 7.35	↑ 45	↓ 22	↓ −2
Combined Respiratory and Metabolic Alkalosis	↑ 7.45	↓ 35	↑ 26	↑ +2

*Partially compensated or compensated metabolic alkalosis generally is rarely seen clinically because of the body's mechanism to prevent hypoventilation.

Modified from Kacmarek RM, Dimas S, & Mack CW. (2005). Acid-base balance and blood gas interpretation. In RM Kacmarek, S Dimas, & CW Mack (Eds.), *The Essentials of Respiratory Care*. St. Louis: Mosby.

BOX 9-1 SIGNS AND SYMPTOMS OF HYPOXEMIA

Integumentary System
- Pallor
- Cool, dry
- Cyanosis (late)
- Diaphoresis (late)

Respiratory System
- Dyspnea
- Tachypnea
- Use of accessory muscles

Cardiovascular System
- Tachycardia
- Dysrhythmias
- Chest pain
- Hypertension early, followed by hypotension
- Increased heart rate early, followed by decreased heart rate

Central Nervous System
- Anxiety
- Restlessness
- Confusion
- Fatigue
- Combativeness/agitation
- Coma

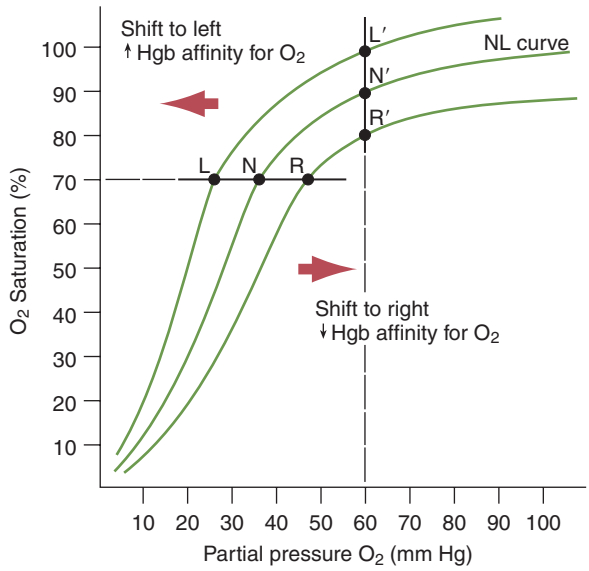

FIGURE 9-8 Oxyhemoglobin dissociation curve. A PaO_2 of 60 mm Hg correlates with an oxygen saturation of 90%. When the PaO_2 falls below 60 mm Hg, small changes in PaO_2 are reflected in large changes in oxygen saturation. Shifts in the oxyhemoglobin curve are shown. *L,* Left shift; *N,* normal; *R,* right shift. (From Alspach J. *AACN Instructor's Resource Manual for AACN Core Curriculum for Critical Care Nursing.* 5th ed. Philadelphia: Saunders; 2001.)

because the hemoglobin responsible for carrying oxygen to all the tissues is still well saturated with oxygen.

The critical zone of the oxyhemoglobin dissociation curve occurs when the PaO_2 decreases to less than 60 mm Hg. At this point, the curve slopes sharply, and small changes in PaO_2 are reflected in large changes in the oxygen saturation. These changes in SaO_2 may cause a significant decrease in oxygen delivered to the tissues.[69,70]

As shown in Figure 9-8, the oxyhemoglobin dissociation curve may shift under certain conditions. When the curve shifts to the right, a decreased hemoglobin affinity for oxygen exists; therefore oxygen is more readily released to the tissues. Conditions that cause a right shift include acidemia, increased temperature, and increased levels of the glucose metabolite 2,3-diphosphoglycerate (2,3-DPG), which occurs in anemia, chronic hypoxemia, and low cardiac output states. When conditions exist where the curve has shifted to the right, the PaO_2 is higher than expected at the normal curve.[69]

When the curve shifts to the left, hemoglobin affinity for oxygen increases and hemoglobin clings to oxygen. Conditions that cause a left shift include alkalemia, decreased temperature, high altitude, carbon monoxide poisoning, and a decreased 2,3-DPG level. Common causes of decreased 2,3-DPG include administration of stored bank blood, septic shock, and hypophosphatemia.[27,70] With a left shift, the PaO_2 is lower than expected at the normal curve. Therefore, if the patient's SpO_2 is 92%, an ABG should be drawn to assess whether hypoxemia is present.

Ventilation and Acid-Base Status

Blood gas values that reflect ventilation and acid-base or metabolic status include the partial pressure of carbon dioxide ($PaCO_2$), pH, and bicarbonate (HCO_3^-).[27,69,70]

pH. The concentration of hydrogen ions (H^+) in the blood is referred to as the *pH.* The normal pH range is 7.35 to 7.45 (exact value, 7.40). If the H^+ level increases, the pH decreases (becomes <7.35) and the patient is said to have *acidemia.* Conversely, a decrease in H^+ level results in an increase in the pH (>7.45), and the patient is said to have *alkalemia.*

Partial pressure of arterial carbon dioxide. $PaCO_2$ is the partial pressure of carbon dioxide (CO_2) dissolved in arterial plasma. The $PaCO_2$ is regulated by the lungs and has a normal range of 35 to 45 mm Hg. A $PaCO_2$ of less than 35 mm Hg indicates respiratory alkalosis; a $PaCO_2$ greater than 45 mm Hg indicates respiratory acidosis. The respiratory system controls the $PaCO_2$ by regulating ventilation (the patient's rate and depth of breathing). If the patient hypoventilates, carbon dioxide is retained, leading to respiratory acidosis ($PaCO_2$ >45 mm Hg). Conversely, if a patient hyperventilates, excess carbon dioxide is excreted by the lungs, resulting in respiratory alkalosis ($PaCO_2$ <35 mm Hg).[69] Conditions that cause respiratory acidosis and alkalosis are noted in Box 9-2.

Sodium bicarbonate. Whereas H^+ ions are an acid in the body, HCO_3^- is a base, a substance that neutralizes or buffers acids. HCO_3^- is regulated by the kidneys. Its normal range is 22 to 26 mEq/L. An HCO_3^- level greater than 26 mEq/L indicates metabolic alkalosis, whereas an HCO_3^- level less than 22 mEq/L indicates metabolic acidosis. Conditions that cause metabolic acidosis and alkalosis are noted in Box 9-2.

Buffer systems. The body regulates acid-base balance through buffer systems, which are substances that minimize the changes in pH when either acids or bases are added. For example, acids are neutralized through combination with a base, and vice versa. The most important buffering system, the bicarbonate buffer system, accounts for more than half of the total buffering and is activated as the H^+ concentration increases. HCO_3^- combines with H^+ to form carbonic acid (H_2CO_3), which breaks down into carbon dioxide (which is

BOX 9-2 CAUSES OF COMMON ACID-BASE ABNORMALITIES

Respiratory Acidosis: Retention of CO_2
- Hypoventilation
- CNS depression (anesthesia, narcotics, sedatives, drug overdose)
- Respiratory neuromuscular disorders
- Trauma: spine, brain, chest wall
- Restrictive lung diseases
- Chronic obstructive pulmonary disease
- Acute airway obstruction (late phases)

Respiratory Alkalosis: Hyperventilation
- Hypoxemia
- Anxiety, fear
- Pain
- Fever
- Stimulants
- CNS irritation (e.g., central hyperventilation)
- Excessive ventilatory support (bag-valve-mask, mechanical ventilation)

Metabolic Acidosis

Increased Acids
- Diabetic ketoacidosis
- Renal failure
- Lactic acidosis
- Drug overdose (salicylates, methanol, ethylene glycol)

Loss of Base
- Diarrhea
- Pancreatic or small bowel fluid loss

Metabolic Alkalosis

Gain of Base
- Excess ingestion of antacids
- Excess administration of sodium bicarbonate
- Citrate in blood transfusions

Loss of Metabolic Acids
- Vomiting
- Nasogastric suctioning
- Low potassium and/or chloride
- Diuretics (loss of chloride and/or potassium)

CNS, Central nervous system; *CO_2,* carbon dioxide.

excreted through the lungs) and water (H_2O). The equation for this mechanism is as follows:

$$H^+ + HCO_3^- \rightarrow H_2CO_3 \rightarrow H_2O + CO_2$$

The bicarbonate buffering system operates by using the lungs to regulate CO_2 and the kidneys to regulate HCO_3^-.[35,69,70]

Base excess or base deficit. The base excess or base deficit is reported on most ABG results. This lab value reflects the sum of all of the buffer bases in the body, the total buffer base. The normal range for base excess/base deficit is −2 to +2 mEq/L. In metabolic acidosis, the body's buffers are used up in an attempt to neutralize the acids, and a base deficit occurs. In metabolic alkalosis, the total buffer base increases and the patient will have a base excess. All metabolic acid-base disturbances are accompanied by a change in the base excess/base deficit, making it a very reliable indicator of metabolic acid-base disorders.[35] In pure respiratory acid-base disturbances, the base excess/base deficit is normal; however, once compensation occurs, the base excess/base deficit changes.

Compensation. Compensation involves mechanisms that normalize the pH when an acid-base imbalance occurs. The kidneys attempt to compensate for respiratory abnormalities, whereas the lungs attempt to compensate for metabolic problems. The lungs quickly respond to compensate for a primary metabolic acid-base abnormality For example, in metabolic acidosis, the depth and rate of ventilation is increased in an effort to blow off more CO_2 (acid). Conversely in metabolic alkalosis, the rate and depth of ventilation may be decreased in an effort to retain acid.[27]

The kidneys compensate for primary respiratory acid-base abnormalities by excreting excess H^+ and retaining HCO_3^-. The renal system activates more slowly taking up to 2 days to regulate acid-base balance. The kidneys excrete HCO_3^- when respiratory alkalosis is present, and retain HCO_3^- when respiratory acidosis is present.[27,35] The renal and respiratory systems exist in harmony to maintain acid-base balance (Figure 9-9).

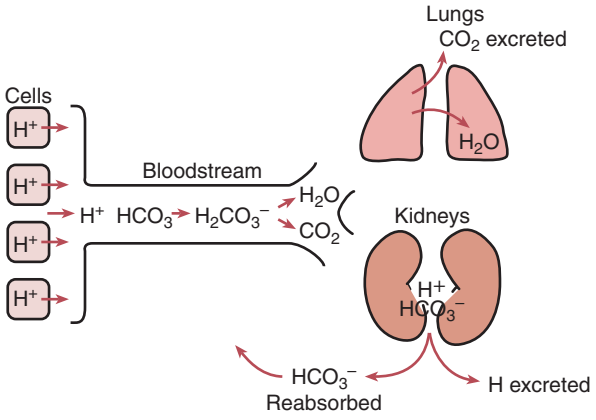

FIGURE 9-9 The kidneys and lungs work together to compensate for acid-base imbalances in the respiratory or metabolic systems. *HCO_3^-*, Bicarbonate; *H_2CO_3*, carbonic acid. (Modified from Harvey MA. *Study Guide to the Core Curriculum for Critical Care Nursing.* 3rd ed. Philadelphia: Saunders; 2000.)

Steps In Arterial Blood Gas Interpretation

Systematic analysis of ABG values involves five steps.[51] Table 9-3 lists lab values associated with acid-base abnormalities. Critical ABG values are noted in the box, "Laboratory Alert."

! LABORATORY ALERT

Arterial Blood Gas Critical Values*

PaO_2 <60 mm Hg
$PaCO_2$ >50 mm Hg
pH <7.25 or >7.60

*These are critical values only if they differ from baseline values (i.e., an acute change). Some patients with pulmonary disease tolerate highly "abnormal" arterial blood gas values.

$PaCO_2$, Partial pressure of carbon dioxide in arterial blood; *PaO_2*, partial pressure of oxygen in arterial blood.

Step 1: Look at each number individually and label it. Decide whether the value is high, low, or normal and label the finding. For example, a pH of 7.50 is high and labeled as alkalemia.

Step 2: Evaluate oxygenation. Oxygenation is analyzed by evaluating the PaO_2 and the SaO_2. Hypoxemia is present and considered a significant problem when the PaO_2 falls to less than 60 mm Hg or the SaO_2 falls to less than 90%. A complete assessment must take into account the level of supplemental oxygen a patient is receiving when the ABG is drawn.

Step 3: Determine acid-base status. Assess the pH to determine the acid-base status. A pH of 7.40 is the absolute normal. If the pH is less than 7.4, the primary disorder is acidosis. If the pH is greater than 7.4, the primary disorder is alkalosis. Therefore, even if the pH is within the normal range, noting whether it is on the acid or alkaline side of 7.40 is important.

Step 4: Determine whether primary acid-base disorder is respiratory or metabolic. Assess the $PaCO_2$, which reflects the respiratory system, and the HCO_3^- level, which reflects the metabolic system, to determine which one is altered in the same manner as the pH. The ABG results may reflect only one disorder (respiratory or metabolic). However, two primary acid-base disorders may occur simultaneously (mixed acid-base imbalance). For example, during cardiac arrest, both respiratory acidosis and metabolic acidosis commonly occur because of hypoventilation and lactic acidosis. Use the base excess to confirm your interpretation of the primary acid-base disturbance, especially if the disorder is mixed.

Step 5: Determine whether any form of compensatory response has taken place. Compensation refers to a return to a normal blood pH by means of respiratory or renal mechanisms. The system opposite the primary disorder attempts the compensation. For example, if a patient has respiratory acidosis, such as occurs in COPD (low pH, high $PaCO_2$), the kidneys respond by retaining more HCO_3^- and excreting H^+. Conversely, if a patient has metabolic acidosis, such as occurs in diabetic ketoacidosis (low pH, low HCO_3^-), the lungs respond by hyperventilation and excretion of carbon dioxide (respiratory alkalosis).[27,69] So if the $PaCO_2$ and the HCO_3^-

BOX 9-3 EXAMPLES OF ARTERIAL BLOOD GASES AND COMPENSATION

Example 1

PaO_2	80 mm Hg (normal)
pH	7.30 (low; acidosis)
$PaCO_2$	50 mm Hg (high; respiratory acidosis)
HCO_3^-	22 mEq/L (normal)
SaO_2	95% (normal)

Interpretation: Normal oxygenation, respiratory acidosis; no compensation.

Example 2

PaO_2	80 mm Hg (normal)
pH	7.32 (low; acidosis)
$PaCO_2$	50 mm Hg (high; respiratory acidosis)
HCO_3^-	28 mEq/L (high; metabolic alkalosis)
SaO_2	95% (normal)

Interpretation: Normal oxygenation, partly compensated respiratory acidosis. The arterial blood gases are only partly compensated because the pH is not yet within normal limits.

Example 3

PaO_2	80 mm Hg (normal)
pH	7.36 (acid side of normal)
$PaCO_2$	50 mm Hg (high; respiratory acidosis)
HCO_3^-	29 mEq/L (high; metabolic alkalosis)
SaO_2	95% (normal)

Interpretation: Normal oxygenation, completely (fully) compensated respiratory acidosis. The pH is now within normal limits; therefore complete compensation has occurred.

HCO_3^-, Bicarbonate; *$PaCO_2$*, partial pressure of carbon dioxide in arterial blood; *PaO_2*, partial pressure of oxygen in arterial blood; *SaO_2*, saturation of hemoglobin with oxygen in arterial blood.

are abnormal in the same direction, then compensation is occurring.

Compensation may be absent, partial, or complete. Compensation is absent if the system opposite the primary disorder is within normal range. If compensation has occurred but the pH is still abnormal, compensation is referred to as partial. Compensation is complete if compensatory mechanisms are present and the pH is within normal range. The body does not overcompensate.[69,70] Examples of ABG compensation are shown in Box 9-3.

Noninvasive Assessment of Gas Exchange

Intermittent ABG results have been the gold standard for the monitoring of gas exchange and acid-base status. Improvements in technology for noninvasive assessment of gas exchange by pulse oximetry and capnography have reduced the number of ABG samples obtained in critically ill patients.

Assessment of Oxygenation

Pulse oximetry. Pulse oximetry measures the saturation of oxygen in pulsatile blood (SpO_2) which reflects the SaO_2. The oxyhemoglobin dissociation curve (see Figure 9-8) shows the relationship between SaO_2 and PaO_2 and provides the basis for pulse oximetry. The sensor that measures SpO_2 is placed on the patient's finger, toe, ear, or forehead where blood flow is not diminished. Light emitted from the sensor is absorbed by hemoglobin with oxygen, or hemoglobin without oxygen providing the necessary information for the device to calculate the percent hemoglobin saturated with oxygen in the pulsatile (arterial) blood. Critically ill patients have continuous pulse oximetry. SpO_2 values are sometimes "spot checked" in patients who are less acutely ill. Pulse oximetry values are used to monitor a patient's response to treatment (e.g., ventilator changes, suctioning, inhalation therapy, body position changes) by following trends in oxygen saturation. However, SpO_2 only measures fluctuation in oxygenation and cannot be used to assess carbon dioxide levels.[51,57]

To ensure accurate SpO_2 readings, the nurse must ensure that the sensor is placed correctly on a warm, well-perfused area and an adequate pulsatile signal is detected. Several factors affect the accuracy of SpO_2 values. Artifact from patient motion or edema at the sensor site may prevent an accurate measurement. The SpO_2 measurements may be lower than the actual SaO_2 if the perfusion to the sensor site is reduced (e.g., limb ischemia, or inflated blood pressure cuff), or in the presence of sunlight, fluorescent light, nail polish or artificial nails, and intravenous dyes. The SpO_2 measurements may be higher than the actual SaO_2 reported by ABG analysis if the patient has an abnormal hemoglobin, such as methemoglobin or carboxyhemoglobin.[28,35]

Assessment of Ventilation

End-tidal carbon dioxide monitoring. End-tidal carbon dioxide monitoring ($ETCO_2$) is the noninvasive measurement of alveolar CO_2 at the end of exhalation when CO_2 concentration is at its peak.[35,65] It reflects alveolar CO_2 level, which in turn reflects the arterial CO_2 ($PaCO_2$) and therefore is used to monitor and assess trends in the patient's ventilatory status. Expired gases are sampled from the patient's airway and are analyzed by a CO_2 sensor that uses infrared light to measure exhaled CO_2 at the end of inspiration. Both a numeric value and a waveform are provided for assessment of ventilation (Figure 9-10).[22,35,65] The sensor may be

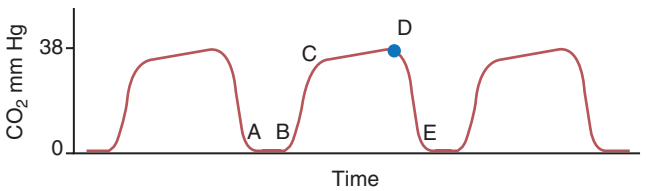

FIGURE 9-10 Capnogram or graphic display of exhaled carbon dioxide (CO_2). Rise in the waveform from *A* to *D* represents CO_2 leaving the lung. Point *D* is where end-tidal CO_2 is measured and represents the highest concentration of exhaled alveolar CO_2.

attached to an adaptor on the endotracheal tube (ETT) or the tracheostomy tube. A nasal cannula with a sidestream capnometer can be used in patients without an artificial airway.[51] The sampling port should be placed as close as possible to the patient's airway.

Normally, $ETCO_2$ values average 2 to 5 mm Hg less than the $PaCO_2$ in individuals with normal lung and cardiac function.[35] To determine the baseline correlation between $ETCO_2$ and $PaCO_2$, the $ETCO_2$ is measured at the same time an ABG is obtained. $ETCO_2$ is subtracted from the $PaCO_2$, providing an index known as the $PaCO_2$-$ETCO_2$ gradient. For example, if a blood gas shows that the $PaCO_2$ is 40 mm Hg and simultaneously the $ETCO_2$ is noted to be 36 mm Hg, the $PaCO_2$-$ETCO_2$ gradient is +4. Knowing the gradient allows for noninvasive assessment of the patient's ventilation by trend monitoring the $ETCO_2$ and inferring the $PaCO_2$ by use of the gradient.[51]

$ETCO_2$ monitoring is used to evaluate ventilation for trending data when precision is not essential. Clinical applications of $ETCO_2$ monitoring include assessment of the patient's response to ventilator changes and respiratory treatments, determining the proper position of the ETT, trending CO_2 in patients with traumatic brain injury or subarachnoid hemorrhage, or detecting disconnection from the ventilator.[22,65] The most common pitfall of $ETCO_2$ monitoring is believing that the value reflects only the patient's ventilatory status. Changes in exhaled CO_2 may occur because of changes not only in ventilation, but also in CO_2 production (metabolism), transport of CO_2 to the lung, and accuracy of the equipment. For example, a decreased $ETCO_2$ value could indicate decreased alveolar ventilation, a reduction in lung perfusion as in hypotension or pulmonary embolus, a reduction in metabolic production of CO_2 as in hypothermia or return to normothermia after fever, or obstruction of the CO_2 sampling tube.[22,65]

Colorimetric carbon dioxide detector. Disposable colorimetric $ETCO_2$ detectors are routinely used after intubation to differentiate tracheal from esophageal intubation (Figure 9-11).[35] When CO_2 is detected, the color of the indicator changes, verifying correct tube placement.

FIGURE 9-11 Disposable colorimetric carbon dioxide (CO_2) detector for confirming endotracheal tube placement. Detection of CO_2 confirms tube placement in the lungs because the only source of CO_2 is the alveoli. (Image used by permission from Nellcor Puritan Bennett LLC, Boulder, Colorado, doing business as Covidien.)

OXYGEN ADMINISTRATION

Oxygen is administered to treat or prevent hypoxemia. Oxygen may be supplied by various sources such as piped into wall devices, oxygen tanks, or oxygen concentrators. The amount of oxygen being administered to the patient is described as the fraction of inspired oxygen (FiO_2). Oxygen concentrations are reported in percentages, whereas the FiO_2 is reported as a decimal. Devices can deliver low (<35%), moderate (35% to 60%), or high (>60%) oxygen concentrations.[30,35]

Oxygen delivery devices are classified into two general categories: low-flow systems (nasal cannula, simple face mask, partial-rebreather mask, and non-rebreather mask), and high-flow systems (air-entrainment or Venturi mask and high-flow nasal cannula).[30,51] Low-flow systems deliver oxygen at flow rates that are less than the patient's inspiratory demand for gas; total patient demand is not met. Low-flow system devices require that the patient entrain, or draw in, room air along with the delivered O_2 enriched gas. FiO_2 cannot be precisely controlled or predicted, because it is determined not only by the amount of oxygen delivered but also the patient's ventilatory pattern and thus the amount of air the patient entrains. For example, if the patient's ventilation increases, the delivered FiO_2 decreases because the patient entrains a larger percentage of room air. Conversely, if the patient's ventilation decreases, the oxygen delivered is less diluted and the FiO_2 rises.[30,51] In high-flow systems, the flow of oxygen enriched gas is sufficient for the patient's total inspiratory demand. The FiO_2 remains fairly constant. In general, for delivery of a consistent FiO_2 to a patient with a variable (deep, irregular, shallow) ventilatory pattern, a high-flow system should be used.

The successful administration of oxygen therapy is important in treating hypoxemia. When administering oxygen, it is important to consider not only the adequacy of the flow delivered by a device, but also fit and function. To ensure proper fit and function, the nurse or RT inspects the patient's face to assess how well the oxygen delivery device is positioned and whether the airway is patent. The oxygen-connecting tubing is traced back to the gas source origin to ensure that it is connected. Finally, it is important to ensure that the gas source is oxygen and that it is turned on and set properly.

Humidification

Humidification of oxygen is recommended when O_2 flow is greater than 4 L/min to prevent the mucous membranes from

drying. At lower flow rates, the patient's natural humidification system provides adequate humidity.[21,49,69] The nurse monitors the quantity and quality (consistency) of the patient's secretions to determine the adequacy of humidification. If the secretions are thick despite adequate humidification of the delivered gases, the patient needs systemic hydration.

Humidification is also an important element of ventilator management. It is essential to maintain the inspired gas reaching the patient's airway at as close to 37° C and 100% relative humidity as possible.[30,69] Two approaches are used to provide humidification. One method functions by actively passing the dry inspired gas through a water-based humidification system before it reaches the patient's airway. The second method is to attach a heat-moisture exchanger (HME) to the ventilator circuit. The HME functions as an artificial "nose" to warm and humidify the patient's inspired breath with his or her own expired moisture and body heat.

During mechanical ventilation, frequent inspection of the humidification unit is needed. If a water-based humidification is used, routine checks include maintaining the water reservoir level and removing condensate from loops in the ventilator circuit. During manipulation of the circuit tubing, it is important to prevent emptying the condensate into the patient's airway. This can lead to contamination of the patient's airway as well as breathing difficulty.[33] If an HME is used, it must be inspected regularly for accumulation of patient secretions in the device, which could result in partial or complete obstruction, increased airway resistance, and increased WOB.[21]

Oxygen Delivery Devices

Nasal Cannula

The nasal cannula is relatively comfortable to wear and easy to secure on the patient. In adult patients, nasal cannulas provide oxygen concentrations between 24% and 44% oxygen at flow rates up to 6 L/min.[30,35] An increase in oxygen flow rate by 1 L/min generally increases oxygen delivery by 4% (e.g., 2 L/min nasal cannula delivers 28% of oxygen, whereas 3 L/min provides 32%). Flow rates higher than 6 L/min are not effective in increasing oxygenation because the capacity of the patient's anatomical reservoir in the nasopharynx is surpassed. An important nursing intervention for patients receiving oxygen via nasal cannula is to assess the skin above the ears for skin breakdown. It may be necessary to pad the tubing over the ear with gauze.

High Flow Nasal Cannula

Oxygen delivered at rates ranging from 15 to 40 L/min is known as high-flow therapy and has historically has been delivered with face masks. However, the delivery of high-flow therapy, which provides high concentrations of oxygen ranging from 60% to 90% and greater is possible through a nasal cannula when it is properly humidified with a special high-flow system. Compliance with therapy is usually better with a nasal cannula because the patient is more comfortable and can eat, drink, and talk. The high-flow system fills the patient's nasopharynx so that it becomes a reservoir of oxygen, thereby improving the oxygen delivered to the alveoli with each breath. It is important to collaborate with the RT to ensure the water in the system remains sufficient to humidify the high flow of gas.

Simple Face Mask

Placing a mask over the patient's face creates an additional oxygen reservoir beyond the patient's natural anatomical reservoir. The mask should fit tight and the flow rate set to at least 5 L/min to prevent rebreathing carbon dioxide. Oxygen is delivered at flow rates of 5 to 12 L/min, which provides concentrations of 30% to 60%.[35] The patient should be instructed about the importance of wearing the mask as applied. The inside of the mask should be cleaned as needed, and the skin should be assessed for areas of pressure.[30]

Face Masks with Reservoirs

Both the partial rebreathing and non-rebreathing masks are similar to the design of a simple face mask, but with the addition of an oxygen reservoir bag. The reservoir increases the amount of oxygen available to the patient during inspiration and allows for the delivery of concentrations of 35% to 60% (partial rebreather) or 60% to 80% (non-rebreather) dependent on the flowmeter setting, the fit of the mask, and the patient's respiratory pattern. The main difference between these two devices is that the non-rebreather mask has one-way valves between the mask and reservoir bag and over one of the exhalation ports. These valves ensure the patient breathes a high concentration of oxygen-enriched gas from the reservoir with each breath (Figure 9-12). The flow rate on the meter should be set to prevent the reservoir bag from deflating no more than one half during inspiration for the partial rebreather, and to prevent the bag from deflating for the non-rebreather.[30] Either mask may be used in the critically ill patient with severe hypoxemia in an effort to prevent the need for endotracheal intubation and mechanical ventilation.

Venturi or Air-Entrainment Mask

The Venturi or air-entrainment mask appears much like a simple face mask; however, it has a jet adapter placed between the mask and the tubing to the oxygen source. The jet adapters come in various sizes and are often color coded to the FiO_2 they deliver. The appropriate oxygen flow rate is often inscribed on the adapter (Figure 9-13). The Venturi mask delivers a fixed FiO_2. Because the level of oxygen can be closely regulated, the Venturi mask is commonly used in the hypoxemic patient with chronic pulmonary disease for whom the delivery of excessive oxygen could depress the respiratory drive.[30,35]

Aerosol and Humidity Delivery Systems

The goal of adding humidity to the inspired gases is to prevent dehydration of the airways and secretions secondary to breathing dry medical gases. The high-humidity face mask or face tent is an option for patients who do not have artificial airways (Figure 9-14). High-flow devices used for administering

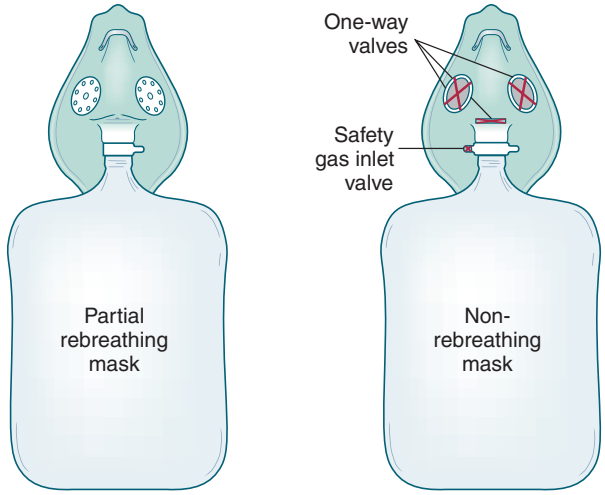

FIGURE 9-12 Partial rebreathing and non-rebreathing oxygen masks. (From Kacmarek RM, Dimas S, Mack CW. *The Essentials of Respiratory Care*. 4th ed. St. Louis: Mosby; 2005.)

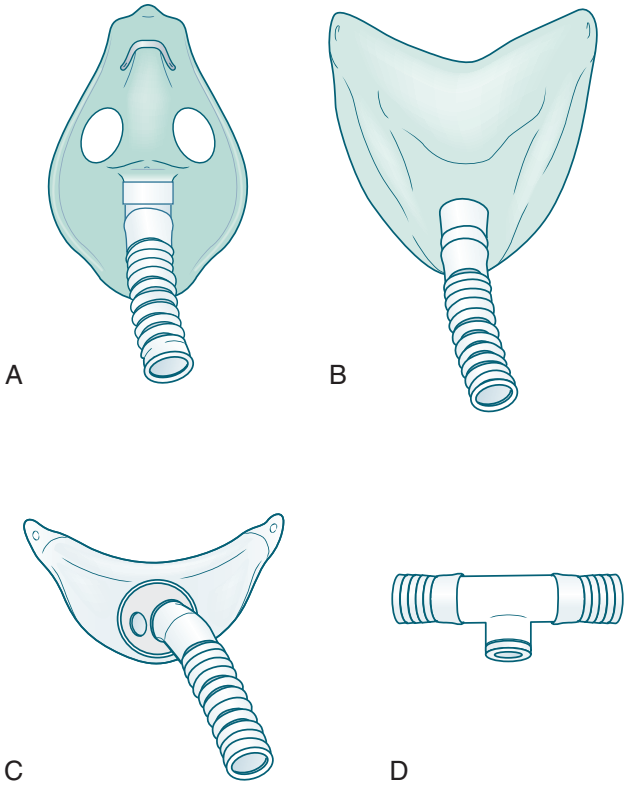

FIGURE 9-14 Devices used to apply high-flow, high-humidity oxygen therapy. **A,** Aerosol mask. **B,** Face tent. **C,** Tracheostomy collar. **D,** Briggs T-piece. (From Kacmarek RM, Dimas S, Mack CW. *The Essentials of Respiratory Care*. 4th ed. St. Louis: Mosby; 2005.)

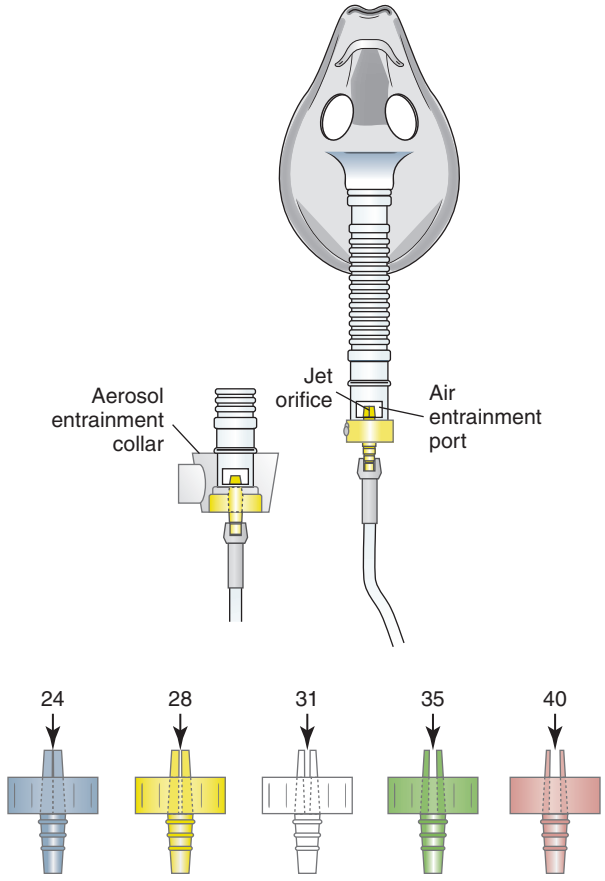

FIGURE 9-13 Air-entrainment (Venturi) mask with various jet orifices. Each orifice provides a specific delivered FiO_2. (Modified from Kacmarek RM, Dimas S, Mack CW. *The Essentials of Respiratory Care*. 4th ed. St. Louis: Mosby; 2005.)

humidified oxygen to patients with an artificial airway are the T-piece and the tracheostomy mask/collar. Humidity is added through a nebulizer that delivers a fixed FiO_2. The initial flow rate is set at 10 L/min and is adjusted so that a constant mist is seen coming from the exhalation port[30,35,51]

Manual Resuscitation Bag (Variable Performance)

A manual resuscitation bag, or bag-valve device, is used to ventilate and oxygenate a patient manually (see Chapter 10). The device is attached to a face mask or connected directly to an ETT or tracheostomy tube to ventilate the patient. When used on an emergency basis, the bag-valve device should have a reservoir attached to increase the FiO_2. The oxygen flowmeter attached to the bag is set at 15 L/min.[51]

AIRWAY MANAGEMENT

Positioning

A patent airway is essential to adequate ventilation and is a priority of nursing care. When the airway is partially or totally obstructed the first method for reinstating a patent airway is proper head position with the head-tilt/chin-lift or jaw thrust. An airway adjunct such as the oral or nasopharyngeal airway may be needed to help maintain the airway.

Oral Airways

The oropharyngeal airway prevents the tongue from falling back and obstructing the pharynx (Figure 9-15). It is indicated when the patient has a depressed level of consciousness. It may also be used to make ventilation with a manual resuscitation bag more effective, or to prevent an unconscious patient from biting and occluding an ETT. It is contraindicated in a patient who is awake because it stimulates the gag reflex, resulting in discomfort, agitation, and possibly emesis. It is important to choose the proper size oral airway: too short an airway forces the patient's tongue back into the pharynx and too long stimulates the gag reflex.[58,60] Nursing care includes assessing the lips and tongue for signs of pressure ulceration and suctioning the oropharynx of accumulated secretions.[51] The technique for inserting an oral airway is described in Box 9-4.

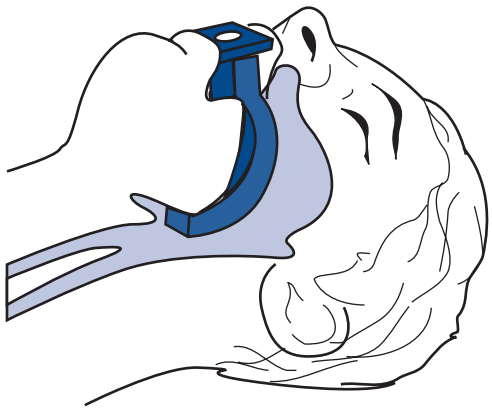

FIGURE 9-15 Maintaining a patent airway with an oral airway. (Modified from Shilling A, Durbin CG. Airway management. In: Cairo JM, ed. *Mosby's Respiratory Care Equipment.* 8th ed. St. Louis: Mosby; 2010.)

BOX 9-4 INSERTION OF ORAL AIRWAY

1. Choose the proper size by measuring the airway on the patient. Airway should extend from the edge of the patient's mouth to the ear lobe.
2. Suction mucus from the mouth using a tonsil (Yankauer) tip catheter.
3. Turn the airway upside down with its tip against the hard palate and slide airway into mouth until the soft palate is reached; then rotate the airway to match the curvature of the tongue into the proper position.
4. An alternative method to step 3 is to use a tongue blade to depress the patient's tongue while inserting the airway, matching its curvature to that of the tongue.
5. Advance tip to back of mouth. Ensure end of airway rests between the teeth but does not compress the lips against the teeth, which would cause injury.
6. Assess airway patency, breath sounds, and chest movement. Noises indicating upper airway obstruction should be absent.
7. Maintain the patient's proper head alignment after airway insertion.

Nasopharyngeal Airways

The nasopharyngeal airway, also known as a nasal airway or nasal trumpet, is a soft rubber or latex tube placed in the nose and extending to the posterior portion of the pharynx (Figure 9-16). It is indicated when an oropharyngeal airway is contraindicated or too difficult to place, such as when the patient's jaw is tight during a seizure, or if oral trauma is present. Nasopharyngeal airways are better tolerated than oral airways in the conscious patient, are more comfortable, and facilitate the passage of a suction catheter during nasotracheal suctioning.

The procedure for inserting a nasotracheal airway is described in Box 9-5. Complications of nasopharyngeal airways include insertion into the esophagus if the airway is too long, nosebleeds, and ulceration of the nares. Extended use of nasopharyngeal airways is not recommended because of an increased risk for sinusitis or otitis.

Endotracheal Intubation

Intubation refers to the insertion of an ETT into the trachea through either the mouth or the nose. Advantages of oral versus nasal endotracheal intubation are listed in Box 9-6. The ETT (Figure 9-17, *A*) is typically made of a polyvinyl

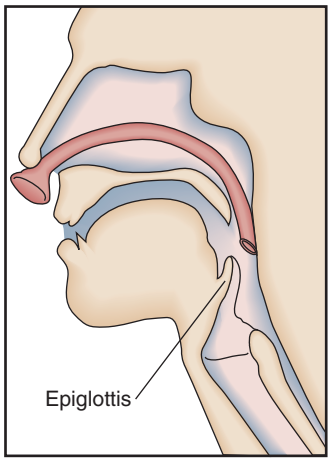

FIGURE 9-16 The nasopharyngeal airway is used to relieve upper airway obstruction and to facilitate passage of a suction catheter.

BOX 9-5 INSERTION OF NASAL AIRWAY

1. Choose the proper size by positioning the airway along the side of the head. The proper length airway extends from the nostril to the earlobe, or just past the angle of the jaw.
2. Generously lubricate the tip and sides of the nasal airway with a water-soluble lubricant.
3. If time allows, lubricate the nasal passage with a topical anesthetic.
4. Insert the airway medially and downward, not upward because the nasopharynx lies directly behind the nares. It may be necessary to rotate the airway slightly.
5. After insertion, assess airway patency, breath sounds, and chest movement.

BOX 9-6 ORAL VERSUS NASOTRACHEAL INTUBATION

Oral Intubation

Advantages

- Quickly performed, emergency airway
- Larger tube facilitates secretion removal and bronchoscopy; creates less airway resistance
- Less kinking of tube
- Preferred method; less sinusitis and otitis media

Disadvantages

- Discomfort
- Mouth care more difficult to perform
- Impairs ability to swallow
- May increase oral secretion production
- May cause irritation and ulceration of the mouth
- Greater risk of self-extubation
- More difficult to communicate by mouthing words
- Patient may bite on airway, reducing gas flow

Nasotracheal Intubation

Advantages

- Greater patient comfort and tolerance
- Better mouth care possible
- Fewer oral complications
- Less risk of accidental extubation
- Facilitates swallowing of oral secretions
- Communication by mouthing words enhanced

Disadvantages

- More difficult to place
- Possible epistaxis during insertion
- Increases risk for sinusitis and otitis media
- May be more difficult to perform
- Secretion removal more difficult because of smaller tube diameter
- Increases work of breathing associated with smaller diameter tube

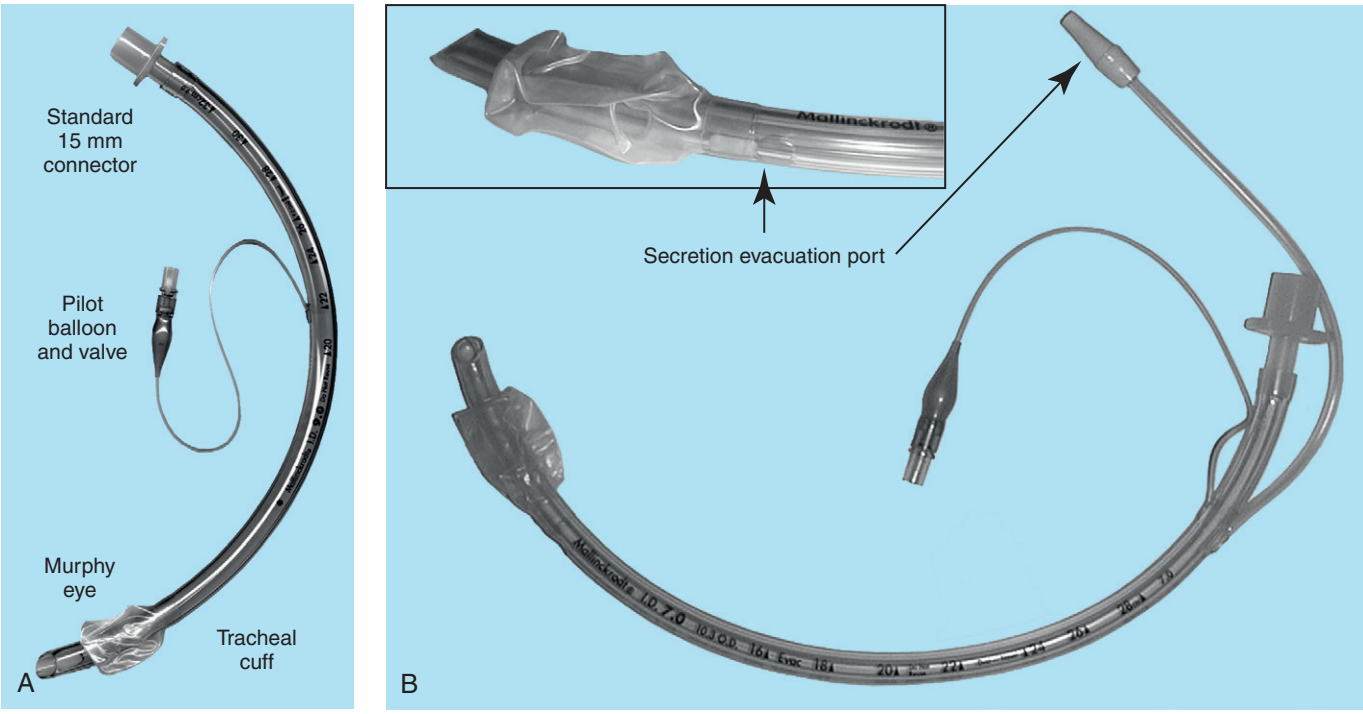

FIGURE 9-17 A, Endotracheal tube. **B,** Hi-Lo Evac endotracheal tube. Note suction port above the cuff for removal of pooled secretions. (From Shilling A, Durbin CG. Airway management. In: Cairo JM, ed. *Mosby's Respiratory Care Equipment.* 8th ed. St. Louis: Mosby; 2010.)

EVIDENCE-BASED PRACTICE

Outcomes of Endotracheal Tubes with Subglottic Secretion Drainage

Problem
Specialized endotracheal tubes (ETT) have been developed with the target of reducing ventilator-associated pneumonia (VAP). One such tube has an additional port for subglottic secretion drainage (SSD-ETT). Outcomes of such devices need to be evaluated.

Clinical Question
What is the impact of the SSD-ETT on preventing VAP?

Evidence
A meta-analysis of 13 randomized clinical trials was conducted. VAP was reduced in 12 of the trials. The overall risk for VAP was reduced by 50%. Use of the SSD-ETT also resulted in a reduction in the critical care length of stay and duration of mechanical ventilation. In subjects who developed VAP, the onset was delayed in those with the SSD-ETT.

Implications for Nursing
Despite strong evidence as to the outcomes associated with the SSD-ETT, the tubes have not been widely adopted. Primary reasons for nonuse are the higher costs associated with the devices, and ensuring that patients who may benefit from the tube get intubated with the specialized devices. Nurses can assist in developing protocols for implementing the SSD-ETT in clinical practice, such as availability of the tube on crash carts and in the emergency department. When a patient has an SSD-ETT, the nurse must collaborate with the respiratory therapist to ensure that the suction port is connected to the suction regulator at the correct pressure and that it is draining appropriately. Periodic flushing of the suction port with air is needed.

Level of Evidence
A—Meta-analysis

Reference
Muscedere J, Rewa O, Mckechnie K, Jiang X, Laporta D, & Heyland DK. Subglottic secretion drainage for the prevention of ventilator-associated pneumonia: a systematic review and meta-analysis. *Critical Care Medicine* 2011;39, 1985-1991.

chloride or silicone material with a distal cuff that is inflated via a one-way valve pilot balloon. The purpose of the cuff is to facilitate ventilation of the patient by sealing the trachea and allowing air to pass through, not around the ETT. Standard ETT cuffs are the high-volume, low-pressure type, and most cuffs are inflated with air (some tubes have a foam-filled cuff). The pilot balloon is used to monitor and adjust cuff pressure as indicated.[58]

ETTs capable of continuous suctioning of subglottic secretions are used in some facilities. These tubes have an extra suction port just above the cuff for removal of secretions that accumulate above the cuff (Figure 9-17, *B*). Evidence shows a decrease in ventilator-associated pneumonia by nearly 50% when these tubes are used (see box, "Evidence-Based Practice").[14,15] Additional interventions are required when these tubes are in place. Continuous low-pressure suction not exceeding −20 mm Hg is applied to the suction lumen. The suction lumen must remain patent. Administration of a bolus of air through the suction port is often needed to relieve obstruction and maintain continuous suction.

Intubation is performed to establish an airway, assist in secretion removal, protect the airway from aspiration in patients with a depressed cough and gag, and provide mechanical ventilation. Personnel who are trained and skilled in intubation perform the procedure: anesthesiologists, nurse anesthetists, acute care nurse practitioners, emergency department physicians, intensivists, RTs, and some paramedics.[51] Intubation may be performed emergently on a patient in cardiac or respiratory arrest, or electively in a patient with impending respiratory failure.

The nurse must be familiar with and be able to gather intubation equipment quickly. The nurse also needs to know how to connect the laryngoscope blade to the handle, check to see that it illuminates properly, and change the bulb as needed. Intubation equipment is frequently kept together in an emergency cart or special procedures box to facilitate emergency intubation (Figure 9-18). The nurse notifies the RT to obtain a ventilator, explains the procedure to the patient, removes dentures if present, gathers all equipment, and ensures that suction equipment is in working order. The nurse assists in positioning the patient, verifies that the patient has a patent intravenous line for the administration of fluids and medications, and provides the necessary equipment while anticipating the needs of the individual performing the intubation.

Procedure for Oral Endotracheal Intubation

The proper size ETT is chosen; it is important that the ETT not be too small, because a smaller-diameter ETT substantially increases airway resistance and the patient's WOB. The average-sized ETT ranges from 7.5 to 8.0 mm for women and from 8.0 to 9.0 mm for men.[58] After the proper size ETT is selected, the cuff is inflated to check for symmetry and any leaks. A plastic-coated malleable stylet may be used to stiffen the ETT to facilitate insertion, but it should be carefully placed inside the ETT to avoid its protrusion beyond the end of the ETT. The ETT is lubricated with a water-soluble lubricant to facilitate passage through the structures of the oropharynx.

The laryngoscope is attached to the appropriate size and type of blade (straight or curved) based on the patient's anatomy and the preference of the clinician performing the intubation. Blade sizes range from 0 to 4. The average-sized adult is intubated with a size-3 blade.[60] Optional equipment

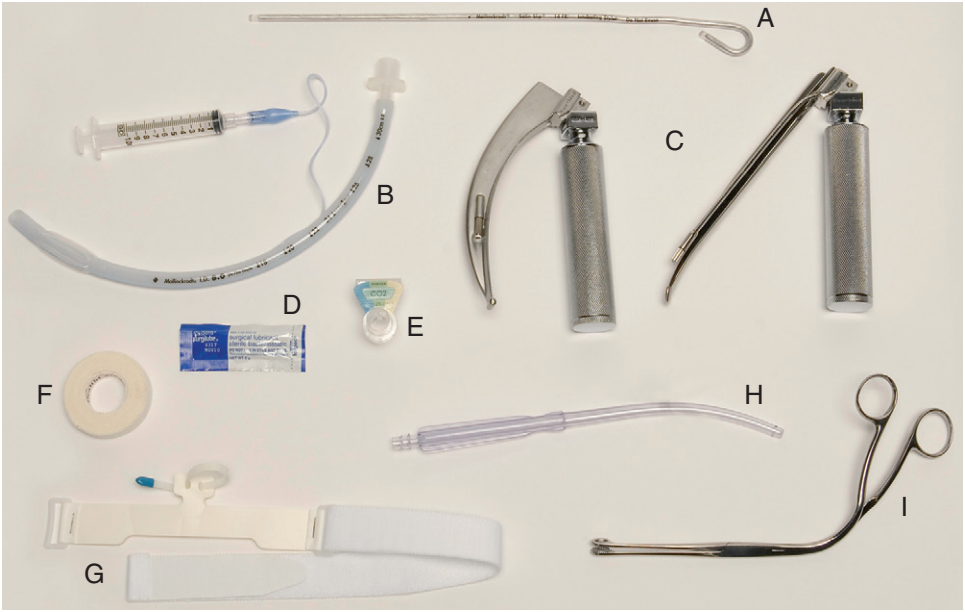

FIGURE 9-18 Equipment used for endotracheal intubation: **A,** stylet (disposable); **B,** endotracheal tube with 10-mL syringe for cuff inflation; **C,** laryngoscope handle with attached curved blade *(left)* and straight blade *(right)*; **D,** water-soluble lubricant; **E,** colorimetric CO_2 detector to check tube placement; **F,** tape or **G,** commercial device to secure tube; **H,** Yankauer disposable pharyngeal suction device; **I,** Magill forceps (optional). Additional equipment, not shown, includes suction source and stethoscope.

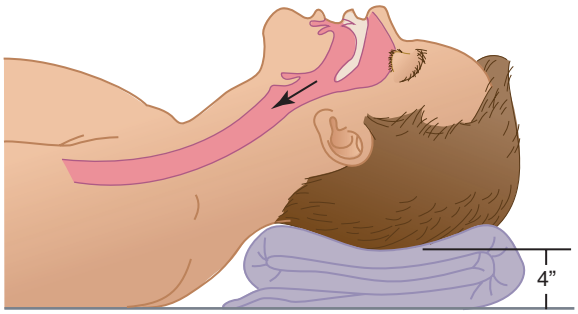

FIGURE 9-19 Elevating the head with a blanket or folded towels places the patient in the "sniffing position" to facilitate endotracheal intubation.

includes a fiberoptic laryngoscope or equipment for video-assisted intubation.

The patient is placed in a "sniffing" position to facilitate visualization of the glottis, or vocal cords. Placing a folded towel or bath blanket under the head may help to achieve this position (Figure 9-19). Time permitting, the patient is premedicated with a sedative and possibly a paralytic agent to allow for easier manipulation of the mandible and visualization of the glottis. The patient is then hyperoxygenated with 100% oxygen by using a bag-valve device connected to a face mask. The intubation procedure should be performed within 30 seconds. If the intubation is difficult and additional attempts are required to secure the airway, the patient must be manually ventilated between each intubation attempt.

The person doing the intubation, while taking care not to damage the patient's teeth or other structures, inserts the laryngoscope blade into the patient's mouth to visualize the vocal cords. If secretions and vomitus are present, the oral cavity is suctioned. A rigid tonsil tip suction (e.g., Yankauer) is very efficient in removing thick secretions and is often used. When the tube is properly inserted about 5 to 6 cm beyond the vocal cords into the trachea, the laryngoscope and stylet are removed and the ETT cuff is inflated.[58,60]

Procedure for Nasotracheal Intubation

Two approaches to nasal intubation are possible: blind and direct visualization.[60] The equipment for nasotracheal intubation is the same as for oral intubation with the addition of Magill forceps. The naris selected for the ETT passage is prepared with a topical vasoconstricting agent to reduce bleeding, and an anesthetic agent. One option is to lubricate the ETT with a water-soluble gel containing 2% lidocaine. The patient is positioned as indicated by the preference of the person performing the intubation: semi-Fowler, high Fowler, or supine.

After the patient's naris and the ETT have been prepared, the ETT is inserted "blindly"; that is, no laryngoscope is used to visualize the cords. The ETT is advanced toward the glottis as the intubator listens to the intensity of the patient's breathing. Blind intubation can be performed only in the patient

who is capable of spontaneous respirations. The closer the intubator comes to the glottis, the more intense the sound of air movement becomes until the ETT passes through the vocal cords and moves into the trachea. The passage of the ETT beyond the vocal cords usually elicits a cough from the patient and vocal silence.

Because some patients have atypical upper airway anatomy, nasal intubation can also be performed through direct visualization. In this method, the practitioner uses a laryngoscope and Magill forceps, or fiberoptic bronchoscopy, for the procedure. When the tube reaches the oropharynx, the laryngoscope is inserted to visualize the cords, and the Magill forceps are used to grasp the tube just above the ETT cuff and direct it between the vocal cords. With nasal intubation, the correct placement level of the ETT at the naris is usually 28 cm for males and 26 cm for females.[58]

Verification of Endotracheal Tube Placement

Correct placement of the ETT in the trachea (versus incorrect placement in the esophagus) is verified by clinical assessment and confirmation devices. Clinical assessment includes auscultation of the epigastrium and lung fields, and observing for bilateral chest expansion.[66] Failure to hear breath sounds while hearing air over the epigastrium represents esophageal rather than tracheal intubation. Breath sounds are equal bilaterally when the tube is placed correctly. Intubation of the right mainstem bronchus is common because the right mainstem is straighter than the left, and the ETT is occasionally placed deeper in the trachea than necessary. Right mainstem bronchus intubation is suspected when unilateral expansion of the right chest is observed during ventilation and the breath sounds are louder on the right than left.

Another method of assessment is done with a confirmation device. Monitoring devices to confirm ETT placement include either a disposable $ETCO_2$ detector, or a bulb aspiration device (esophageal detector device). The disposable $ETCO_2$ detector is attached to the end of the ETT. This device changes color when carbon dioxide is detected and is a highly reliable method of confirming tracheal (versus esophageal) intubation.[58,60] Another option is to attach an aspiration device that is similar to a bulb syringe. The device is compressed and deflated and is attached to the ETT. If the tube is in the trachea, the bulb inflates rapidly. If the tube is in the esophagus, filling is delayed. Pulse oximetry also assists in assessment of tube placement. SpO_2 will fall if the esophagus has been inadvertently intubated, and it may be decreased in right mainstem intubation. Finally, a portable chest radiograph is ordered to confirm tube placement.[51]

The tip of the ETT should be approximately 3 to 4 cm above the carina.[60] Once the placement is confirmed, the centimeter depth marking at the lip or naris should be noted in the medical record. An indelible marker can be used to mark the ETT at the lip or naris. These nursing measures assist with ongoing monitoring of proper tube position. The nurse and RT collaborate to ensure the ETT is properly secured with tape or a commercial device to prevent dislodging. Figure 9-20 shows two methods for securing the ETT.

Tracheostomy

A tracheostomy tube provides an airway directly into the anterior portion of the neck. Tracheostomy tubes are indicated for long-term mechanical ventilation, long-term secretion management, protecting the airway from aspiration when the cough and gag reflexes are impaired, bypassing an upper airway obstruction that prevents placement of an ETT, and reducing the WOB associated with an ETT. The tracheostomy tube reduces the WOB because it is shorter than an ETT and airflow resistance is less.[18,66]

A tracheostomy is the preferred airway for the patient requiring a long-term airway who is able to transfer to a progressive care unit, because it is associated with several advantages.[66] It is better tolerated than an ETT; therefore patients may require less sedation or restraint use. A patient may be permitted oral intake if swallowing studies demonstrate absence of aspiration. Oral hygiene is more easily performed, and some tube designs allow for talking and therefore facilitate patient communication.

There is no clearly defined time for when a tracheostomy should be performed. If mechanical ventilation and an artificial airway are projected to be needed for a prolonged period, the decision to perform a tracheostomy should be made early.[17]

The tracheostomy has traditionally been a surgical technique performed in the operating room. However, a percutaneous dilatational tracheostomy (PDT) procedure may be performed safely at the bedside by a trained physician.[17] The PDT is performed by making a small incision into the anterior neck down to the trachea. Once this location has been reached, the physician inserts a needle and sheath into the trachea. The needle is removed, and a guidewire is passed through the sheath. Progressively larger dilators are introduced over the guidewire until the patient's stoma is large enough to accommodate a tracheostomy tube.[17,18]

Collaboratively, the nurse and RT assist in the PDT procedure. Before the procedure, the nurse ensures that intravenous access lines are accessible for administration of sedatives and analgesic medications. The patient is properly positioned, and the height of the bed is adjusted relative to the individual performing the procedure. Sterile supplies are gathered, and sterility is maintained throughout the procedure. Physiological parameters are monitored continuously and documented at least every 15 minutes throughout the PDT, and for a least an hour after the procedure.[51]

The most significant postprocedure complication of PDT is accidental decannulation. When a patient undergoes a surgical tracheostomy, the trachea is surgically attached to the skin. This promotes prompt identification of the tract and reinsertion of the tracheal tube should it become dislodged. With a PDT, the trachea is not secured in this way, and a mature tract takes approximately 2 weeks to form. Accidental decannulation and attempted reinsertion of the airway during this time may result in difficulty securing the airway, bleeding, tracheal injury, and death. Oral intubation may be required if the airway becomes dislodged or needs to be replaced.[51]

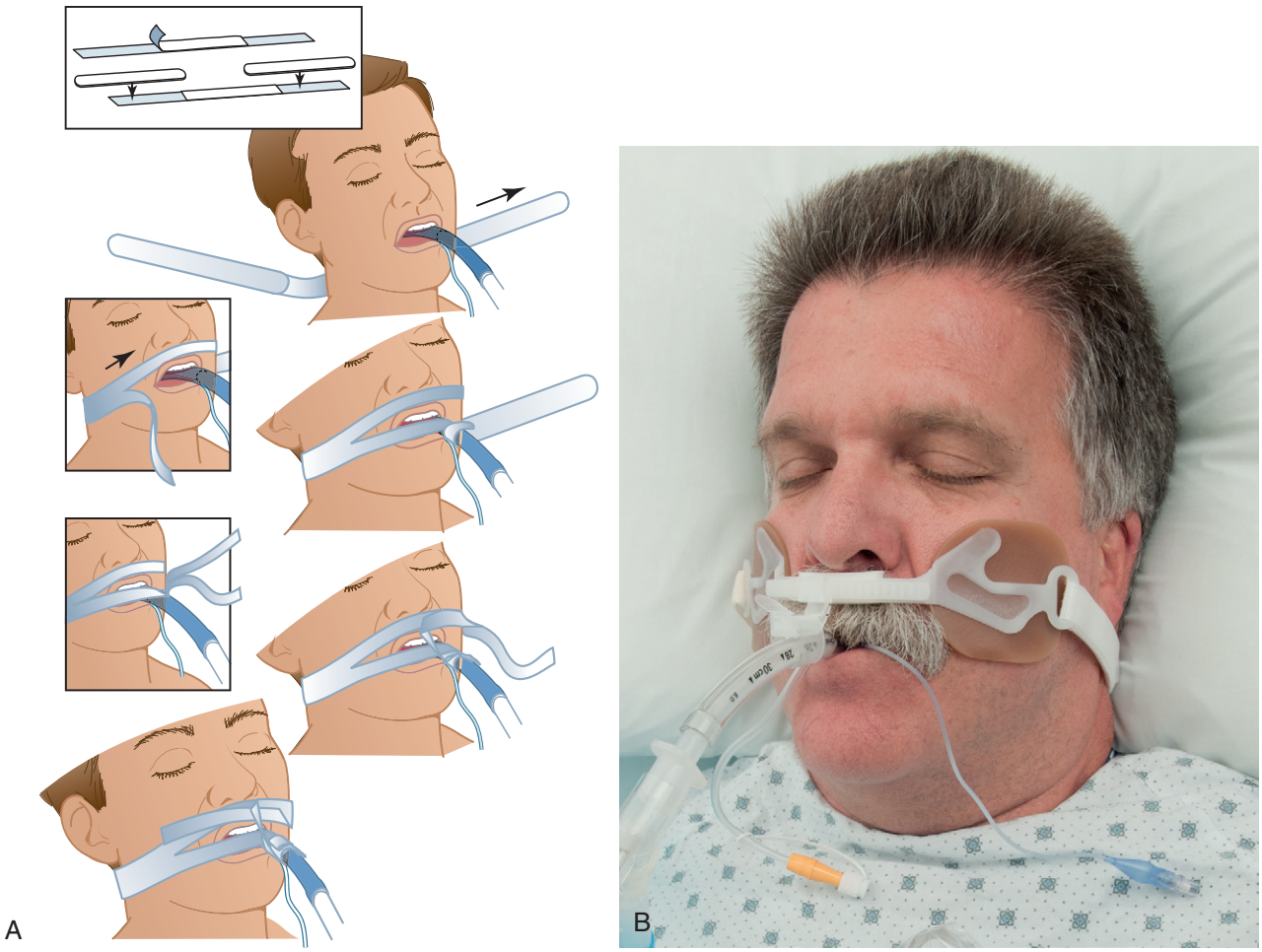

FIGURE 9-20 Two methods for securing the endotracheal tube: tape **(A)** and harness device **(B).** Harness device shown is the SecureEasy Endotracheal Tube Holder. Nonelastic headgear reduces the risk of self-extubation. A soft bite block prevents tube occlusion. (**B** Reprinted with permission, Cleveland Clinic Center for Medical Art & Photography © 2011-2012. All rights reserved.)

Tracheostomy Tube Designs

Tracheostomy tubes come in a variety of sizes and styles, and are primarily made of plastic. Design features are shown in Figure 9-21. The flange lies against the patient's neck and has an opening on both ends for the placement of tracheostomy ties for securing the airway. Similar to the ETT, some tracheostomy tubes have a distal cuff and pilot balloon. An important part of the tracheostomy system is the obturator, which is inserted into the trachea tube during insertion. The rounded end of the obturator extends just beyond the end of the tracheostomy tube and creates a smooth tip, allowing for easy entry into the stoma. The obturator is removed after tube insertion to allow for air passage through the trachea. It must be kept in a visible location in the patient's room should emergency reinsertion of a misplaced tube be necessary. In this situation the obturator is inserted into the tracheostomy to create a rounded, smooth end promoting reentry into the stoma without tissue injury.[51]

Cuffed versus uncuffed tracheostomy tubes. Critically ill patients who need mechanical ventilation require cuffed tubes to ensure delivery of ventilation and prevent aspiration. The cuff may be a conventional low-pressure, high-volume type, or it may be constructed of foam. The foam-cuff tube may prevent trauma to the airway because of the low pressure exerted to the airway, and it is sometimes used for patients who have difficulty maintaining a good seal with conventional cuffed tracheostomy tubes. Many other types of tracheostomy tubes are available.[58,60,66] An uncuffed tracheostomy tube is used for long-term airway management in a patient who does not require mechanical ventilation and is at low risk of aspiration. For example, a patient with a neurological injury may require a tracheostomy for airway management and secretion removal. Metal tracheostomy tubes are uncuffed.[63]

Single- versus double-cannula tracheostomy tubes. Tracheostomy tubes may have one or two cannulas. A single-cannula

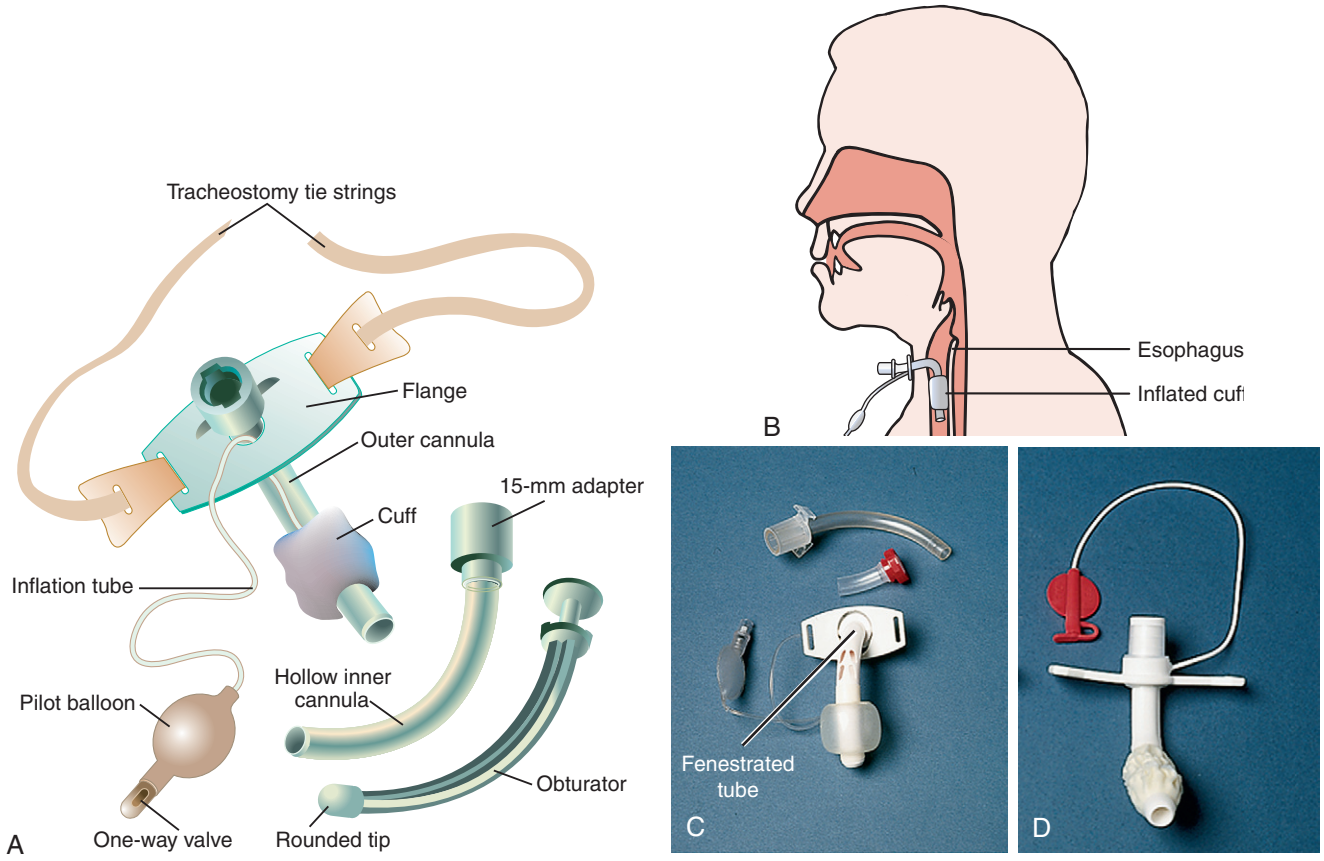

FIGURE 9-21 A, General design features of the tracheostomy tube. **B,** Trach tube in place. **C,** Fenestrated tracheostomy tube (see text for description). **D,** Fome cuff tracheostomy tube. (From Lewis SL, Dirkson SR, Heitkemper MM, et al. *Medical-Surgical Nursing.* 8th ed. St. Louis: Mosby; 2011.)

tube does not have an inner cannula, whereas a double-cannula tube has both an inner and outer cannula. The inner cannula is removable to facilitate cleaning of the inner lumen and to prevent tube occlusion from accumulated secretions. Inner cannulas can be reusable or disposable. Cuffed tracheostomy tubes with disposable inner cannulas are commonplace in the critical care unit.

Fenestrated tracheostomy tube. The fenestrated tracheostomy tube has a hole in the outer cannula that allows air to flow above the larynx. The tube functions as a standard tracheostomy tube when the inner cannula is in place. When the inner cannula is removed, the fenestrated tracheostomy tube assists in weaning a patient from the tracheostomy by gradually allowing the patient to breathe through the natural upper airway. The fenestrated tube also allows the patient to emit vocal sounds, thereby facilitating communication.[60,66] To use a cuffed fenestrated tracheostomy tube for speaking or to promote breathing through the natural airway, the inner cannula is carefully removed and the cuff is deflated. The inner cannula must be reinserted and the cuff reinflated for eating, suctioning, mechanical ventilation, or use of a bag-valve device.[51]

Speaking tracheostomy valves. One-way speaking valves are available to allow patients with a tracheostomy an opportunity to speak. Although these valves can be used in both ventilated and nonventilated patients, they can be used only in patients capable of initiating and maintaining spontaneous ventilation.[60] Examples of these adjunctive devices include the Passy-Muir Valve (Passy-Muir, Inc., Irvine, CA) and the Shiley Phonate Speaking Valve (Covidien, Boulder, CO). For the speaking valve to work correctly, the valve is connected to the tracheostomy tube, the cuff on the tracheostomy tube is deflated, and the patient is allowed to breathe and exhale through the natural airway. The valve itself is a one-way device allowing gas to enter through it into the tracheostomy tube and to the patient. Because this is a one-way valve, exhaled gas exits the trachea via the natural airway, past the deflated cuff of the tracheostomy tube and through the vocal cords.[58]

If a speaking valve is used in conjunction with mechanical ventilation, it must be used with a tracheostomy tube, not an ETT. The delivered tidal volume (V_T) must be increased to ensure an adequate volume to ventilate the patient because a portion of the delivered V_T is lost via the deflated

tracheostomy cuff.[51] While the valve is in place, the patient is carefully assessed for respiratory stability and tolerance. Monitoring includes measurements of the patient's SpO_2, heart rate, RR, and blood pressure; observations about the patient's anxiety level and perception of the experience; and assessment of the patient's WOB. Management of secretions is another important nursing intervention.[5]

Endotracheal Suctioning

Patients with an artificial airway need to be suctioned to ensure airway patency because the normal protective ability to cough and expel secretions is impaired. Suctioning is performed according to a standard procedure to prevent complications such as hypoxemia, airway trauma, infection, and increased intracranial pressure in patients with head injury. Suctioning also stimulates the cough reflex and promotes the mobilization and removal of secretions.

Because suctioning is associated with complications, it is performed only as indicated by physical assessment and not according to a predetermined schedule. Indications for endotracheal suctioning include visible secretions in the tube, frequent coughing, presence of rhonchi, oxygen desaturation, a change in vital signs (e.g., increased or decreased heart rate or RR), dyspnea, restlessness, increased peak inspiratory pressure (PIP), or high-pressure ventilator alarms.[12,51] The number of suction passes is usually one to three; however, suctioning should be continued until secretions are removed. Suction duration is limited to 10 to 15 seconds and rest periods are provided between suction passes.

Key points related to endotracheal suctioning are discussed in Box 9-7. Hyperoxygenation with 100% oxygen should be performed for 30 seconds before suctioning, during the procedure, and immediately after suctioning.[12] Most ventilators have a built-in suction mode that delivers 100% oxygen for a short period (e.g., 2 minutes). Hyperoxygenation can also be administered with a bag-valve device. If the patient does not tolerate suctioning with hyperoxygenation alone, hyperinflation may be used. Hyperinflation involves the delivery of breaths 1.0 to 1.5 times the V_T and is performed by giving the patient three to five breaths before and between suctioning attempts using either the ventilator or bag-valve device.[12]

The closed tracheal, or in-line, suction catheter is an alternative to the single use suction catheter. The closed tracheal suction system consists of a suction catheter enclosed in a plastic sheath that is attached to the patient's ventilator circuit and airway (Figure 9-22). The device assists in maintaining oxygenation during suctioning, reduces symptoms associated with hypoxemia, maintains positive end-expiratory pressure (PEEP), and protects staff from the patient's secretions; the data are inconsistent regarding its cost-effectiveness.[19,34] Depending on the institution, closed suctioning may be used on all ventilated patients; or it may be used for specific indications, such as for clinically unstable patients receiving high levels of PEEP, and for those requiring frequent suctioning.[45,63]

Saline instillation into the trachea during suctioning should not be routinely performed.[52] Although use of saline

BOX 9-7 KEY POINTS FOR ENDOTRACHEAL SUCTIONING

- Suction only as indicated by patient assessment.
- Choose the proper-size device. The diameter of the suction catheter should be no more than half the diameter of the artificial airway.
- Assemble equipment: suction kit with two gloves or closed suction system (CSS), sterile water or saline for rinsing the catheter. The CSS is attached to the ventilator circuit, usually by a respiratory therapist.
- Set the suction regulator at 80 to 120 mm Hg.
- Use sterile technique for suctioning.
- Hyperoxygenate the patient via the ventilator circuit before, between, and after suctioning.
- Gently insert suction catheter until resistance is met, then pull back 1 cm.
- Suction the patient no longer than 10 to 15 seconds while applying intermittent or constant suction.
- Repeat endotracheal suctioning until the airway is clear.
- Rinse the catheter with sterile saline after endotracheal suctioning is performed.
- Suction the mouth and oropharynx with the single-use suction catheter, suction swabs, or a tonsil suction device.
- Auscultate the lungs to assess effectiveness of suctioning, and document findings.
- Document the amount, color, and consistency of secretions.
- *Steps specific to closed suctioning* (in addition to those just noted):
  - Using the dominant hand, insert the suction catheter into the airway until resistance is met. Simultaneously, use the nondominant hand to stabilize the artificial airway.
  - Withdraw the suction catheter while depressing the suction valve; be careful to not angle the wrist of the hand while withdrawing the catheter, because kinking of the catheter and loss of suction may occur.
  - Ensure that CSS catheter is completely withdrawn from the airway. A marking is visible on the suction catheter when it is properly withdrawn.
  - Rinse the catheter after the procedure. Connect a small vial or syringe of normal saline for tracheal instillation (without preservatives) to the irrigation port, and simultaneously instill the saline into the port while depressing the suction control.
  - Keep the CSS suction catheter out of the patient's reach to avoid accidental self-extubation.

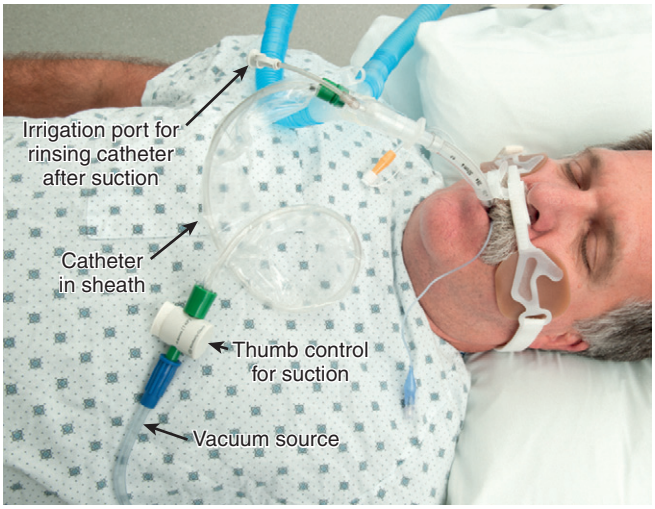

FIGURE 9-22 Closed tracheal suction device. (Reprinted with permission, Cleveland Clinic Center for Medical Art & Photography © 2011-2012. All rights reserved.)

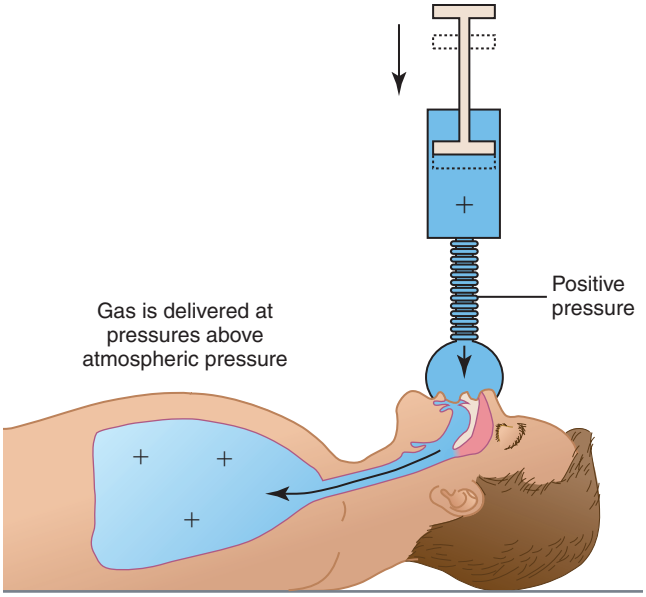

FIGURE 9-23 Concept of positive-pressure ventilation.

was a common practice for many years, saline instillation is associated with problems such as oxygen desaturation, washing organisms in the ETT into the lower airway, and patient discomfort.[1,10,46,55] Purported benefits of liquefying secretions and increasing volume of secretions removed are not proven. Adequate patient hydration and airway humidification, rather than saline instillation, facilitate secretion removal.

MECHANICAL VENTILATION

The purpose of mechanical ventilation is to support the respiratory system until the underlying cause of respiratory failure can be corrected. Most ventilatory support requires an artificial airway; however, it may be applied without an artificial airway and is called noninvasive ventilation.

Indications

Mechanical ventilation is warranted for patients with acute respiratory failure who are unable to maintain adequate gas exchange as reflected in the ABGs. A clinical definition of respiratory failure is as follows:

- PaO_2 ≤60 mm Hg on a FiO_2 greater than 0.5 (oxygenation)
- $PaCO_2$ ≥50 mm Hg, with a pH of 7.25 or less (ventilation)[35,53]

The patient may also demonstrate progressive physiological deterioration such as rapid, shallow breathing and an increase in the WOB as evidenced by increased use of the accessory muscles of ventilation, abnormal breathing patterns, and complaints of dyspnea. As lifesaving therapy, the purpose of mechanical ventilation is to support the respiratory system while a treatment plan is instituted to correct the underlying abnormality.[35,51,52]

Positive-Pressure Ventilation

In the critical care setting, most patients are treated with positive-pressure ventilation. This method uses positive pressure to force air into the lungs via an artificial airway, as illustrated in Figure 9-23. Movement of gases into the lungs through the use of *positive pressure* is the opposite of spontaneous breathing. Spontaneous ventilation begins when energy is expended to contract the muscles of respiration. This enlarges the thoracic cavity, increases *negative pressure* within the chest and lungs, and results in the flow of air, at atmospheric pressure, into the lungs. If mechanical ventilators could mimic the intrathoracic pressures present during spontaneous ventilation, it would be ideal. Negative-pressure ventilators, which originated with the iron lung, perform in this manner; however, these ventilators are for management of chronic conditions. Many of the complications of mechanical ventilation are related to air being forced into the lungs under positive pressure.

Ventilator Settings

In most institutions in the United States and Canada, ventilators are set up and managed by RTs. However, the nurse must be familiar with selected values on the control panel or graphic interface unit to assess ventilator settings, patient response to ventilation, and alarms. Representative control panels and screens of ventilators are shown in Figure 9-24. Although the control panel of a microprocessor type of ventilator can appear overwhelming, it is important for the nurse to learn to identify the common screen views that provide the settings and patient data that are integral to patient assessment. The nurse must know the basic ventilator settings of mode of ventilation, FiO_2, V_T, set RR rate, PEEP, and pressure support. Additional settings of inspiratory-to-expiratory (I:E) ratio, sensitivity, and sigh are also discussed to provide a basis for the nurse to knowledgeably communicate with the RT and provider.[51]

FIGURE 9-24 Examples of mechanical ventilators and their control panels and graphic interface unit (GIU). **A,** Dräger Evita XL. **B,** Puritan Bennett 840 Ventilator GIU. (**A** Copyright Drägerwerk AG & Co. KGa, Lubeck. All rights reserved. **B** Image used by permission from Nellcor Puritan Bennett LLC, Boulder, Colorado, doing business as Covidien.)

Fraction of Inspired Oxygen

The FiO_2 is set from 0.21 (21% or room air) to 1.00 (100% oxygen). The initial FiO_2 setting is based on the patient's immediate physiological needs and should be set to whatever is necessary to maintain a PaO_2 between 60 and 100 mm Hg and/or a SpO_2 of *at least* 90%. After the patient is stabilized, the setting is adjusted based on ABG or pulse oximetry values.

Tidal Volume

The amount of air delivered with each preset breath is the V_T. The V_T is dictated by body weight and by the patient's lung characteristics (compliance and resistance), and it is set to ensure that excessive stretch and pressure on the lung tissue is avoided. A starting point for the V_T setting is 6 to 8 mL/kg of ideal body weight with the lowest value recommended in patients with obstructive airway disease or ARDS.[7,31,35] The parameters monitored to avoid excessive pressure are the PIP and plateau airway pressure (Pplat). These pressures should remain below 40 cm H_2O and 30 cm H_2O, respectively.[7] The V_T setting can be reduced if the resulting airway pressures are nearing the maximum. Conversely, if the airway pressures are acceptable and a larger V_T is needed to remove CO_2, it can be increased. When choosing and adjusting the V_T setting, the goal is to achieve the lowest Pplat while maintaining gas exchange and patient comfort.

Respiratory Rate

The RR is the frequency of breaths (f) set to be delivered by the ventilator. The RR is set as near to physiological rates (14 to 20 breaths per minute) as possible. Frequent changes in the RR are often required based on observation of the patient's WOB and comfort, and assessment of the $PaCO_2$ and pH. During initiation of mechanical ventilation, many patients require full ventilatory support. The RR at this time is selected on the basis of the V_T, to achieve a minute ventilation (VE) that maintains an acceptable acid-base status ($VE = RR \times V_T$). As the patient becomes capable of participating in the ventilatory work, the ventilator RR is decreased, or the mode of ventilation is changed, to encourage more spontaneous breathing.

Inspiratory-to-Expiratory Ratio

The I:E ratio is the duration of inspiration in comparison with expiration. In spontaneous ventilation, inspiration is shorter than expiration. When a patient undergoes mechanical ventilation, the I:E ratio is usually set at 1:2 to mimic this pattern of spontaneous ventilation; that is, 33% of the respiratory cycle is spent in inspiration and 66% in the expiratory phase. Longer expiratory times, I:E ratio of 1:3 or 1:4, may be needed in patients with COPD to promote more complete exhalation and reduce air trapping.[35,51,53]

Inverse Inspiratory-to-Expiratory Ratio

Inspiratory-to-expiratory (I:E) ratios such as 1:1, 2:1, and 3:1 are called inverse I:E ratios. An inverse I:E ratio is used to improve oxygenation in patients with noncompliant lungs, such as in ARDS. During the traditional I:E ratio of 1:2, alveoli in noncompliant lungs may not have sufficient time to reopen during the shorter inspiratory phase, and may collapse during the longer expiratory phase. An inverse I:E ratio allows unstable alveoli time to fill and prevents them from collapsing, because the next inspiration begins before the alveoli reach a volume where they can collapse.[35,51]

Positive End-Expiratory Pressure

PEEP is the addition of positive pressure into the airways during expiration. PEEP is measured in cm H_2O. Typical settings for PEEP are 5 to 20 cm H_2O, although higher levels may be used to treat refractory hypoxemia. Because positive pressure is applied at end expiration, the airways and alveoli are held open, and oxygenation improves. PEEP increases oxygenation by preventing collapse of small airways and maximizing the number of alveoli available for gas exchange (Figure 9-25). By recruiting more alveoli for gas exchange and by holding them open during expiration, the functional residual capacity improves, resulting in better oxygenation.

Many mechanically ventilated patients routinely receive 3 to 5 cm H_2O of PEEP, a value often referred to as *physiological PEEP.* This small amount of PEEP is thought to mimic the normal "back pressure" created in the lungs by the epiglottis in the spontaneously breathing patient that is removed by the displacement of the epiglottis by the artificial airway.

PEEP is often added to decrease a high FiO_2 that may be required to achieve adequate oxygenation. For example, a patient may require a FiO_2 of 0.80 to maintain a PaO_2 of 85 mm Hg. By adding PEEP, it may be possible to decrease the FiO_2 to a level where oxygen toxicity in the lung is not a concern (<0.5) while maintaining an adequate PaO_2.[53] The nurse monitors the PEEP level by observing the pressure level displayed on the ventilator's analog and graphic displays. When no PEEP is set, the pressure reading on the graphic display should be zero at end expiration. When PEEP is applied, the pressure reading does not return to zero at the end of the breath, and the display shows the amount of PEEP.

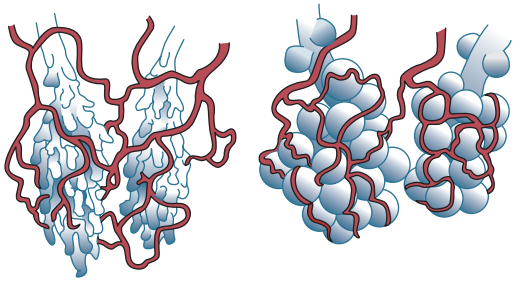

FIGURE 9-25 Effect of application of positive end-expiratory pressure (PEEP) on the alveoli. (Modified from Pierce LNB. *Management of the Mechanically Ventilated Patient.* Philadelphia: Saunders; 2007.)

Although PEEP is often essential for treatment, it is associated with adverse effects. Problems related to PEEP occur as a result of the increase in intrathoracic pressure. These problems include a decrease in cardiac output secondary to decreased venous return, volutrauma or barotrauma, and increased intracranial pressure resulting from impedance of venous return from the head, Whenever the level of PEEP is increased, the nurse should evaluate the patient's hemodynamic response through physical assessment and by available hemodynamic parameters. Management of decreased cardiac output secondary to PEEP includes ensuring the patient has adequate intravascular volume (preload) and administering fluids as needed. If the cardiac output remains inadequate, an inotropic agent such as dobutamine should be considered. Optimal PEEP is defined as the amount of PEEP that affords the best oxygenation without resulting in adverse hemodynamic effects or pulmonary injury.[35,51]

Auto-PEEP. Auto-PEEP is the spontaneous development of PEEP caused by gas trapping in the lung resulting from insufficient expiratory time and incomplete exhalation. These trapped gases create positive pressure in the lung. Both set PEEP and auto-PEEP have the same physiological effects; therefore it is important to know when auto-PEEP is present so it can be managed properly.[35,51,53]

Causes of auto-PEEP formation include rapid RR, high VE demand, airflow obstruction, and inverse I:E ratio ventilation. Auto-PEEP cannot be detected by the ventilator pressure manometer until a special maneuver is performed. This maneuver involves instituting a 2-second end-expiratory pause, which allows the ventilator to read the pressure deep in the lung. The airway pressure manometer reading therefore reflects total PEEP, which is the set PEEP and auto-PEEP added together. To determine auto-PEEP, the following calculation is performed:

$$\text{Auto-PEEP} = \text{Total PEEP} - \text{Set PEEP}$$

Sensitivity

Sensitivity determines the amount of patient effort needed to initiate gas flow through the circuitry on a patient-initiated breath. The sensitivity is set so that the ventilator is "sensitive" to the patient's effort to inspire. If the sensitivity is set too low, the patient must generate more work to trigger gas flow. If it is set too high, auto-cycling of the ventilator may occur, resulting in patient-ventilator dyssynchrony, because the ventilator cycles into the inspiratory phase when the patient is not ready for a breath.[51,53]

Patient Data

The nurse and RT ensure that the ventilator settings are consistent with the physician's orders. The ventilator control panel or graphic interface unit also provides valuable information regarding the patient's response to mechanical ventilation. These patient data include exhaled tidal volume (EV_T), PIP, and total RR.

Exhaled Tidal Volume

The EV_T is the amount of gas that comes out of the patient's lungs on exhalation. The EV_T is not a ventilator setting. It is considered patient data that indicates the patient's response to mechanical ventilation. This is the most accurate measure of the volume received by the patient and therefore is monitored at least every 4 hours and more often as indicated. Although the prescribed V_T is set on the ventilator control panel, it is not guaranteed to be delivered to the patient. Volume may be lost because of leaks in the ventilator circuit, around the cuff of the airway, or via a chest tube if there is a pleural air leak.[50] The volume actually received by the patient, regardless of mode of ventilation, must be confirmed by monitoring the EV_T on the display panel of the ventilator. If the EV_T deviates from the set V_T by 50 mL or more, the nurse and RT must troubleshoot the system to identify the source of gas loss.[50]

Peak Inspiratory Pressure

The PIP is the maximum pressure that occurs during inspiration. The amount of pressure necessary to ventilate the patient increases with increased airway resistance (e.g., secretions in the airway, bronchospasm, biting the ETT) and decreased lung compliance (e.g., pulmonary edema, worsening infiltrate or ARDS, pleural space disease). The PIP should never be allowed to rise above 40 cm H_2O, because higher pressures can result in ventilator-induced lung injury.[7,62]

The nurse should monitor and record the PIP at least every 4 hours and with any change in patient condition that could increase airway resistance or decrease compliance.[50] Increasing PIP or values greater than 40 cm H_2O should be immediately reported so that interventions can be ordered to improve lung function, ventilator settings can be adjusted to reduce the inspiratory pressure, or both.

Total Respiratory Rate

The total RR equals the number of breaths delivered by the ventilator (set rate) plus the number of breaths initiated by the patient. Assessing the total RR provides data regarding the patient's contribution to the WOB, or whether the ventilator is performing all of the work. It also provides an assessment of the ability of the set RR and V_T to meet VE demands. The total RR is a very sensitive indicator of overall respiratory stability.[50] For example, if the patient is on assist/control ventilation at a set RR of 10 breaths per minute, and the total RR for 1 minute is 16, the patient is initiating 6 breaths above the set rate of 10. If the patient is on synchronized intermittent mandatory ventilation at a set RR of 8 breaths per minute, and the total RR is 12 breaths per minute with good spontaneous V_T for body weight, the patient is tolerating the mode of ventilation. If the patient's total RR increases to 26 breaths per minute, this finding indicates that something has changed and the patient needs to be reassessed for causes of the increased rate, such as fatigue, pain, or anxiety. Treatment is based on the identified cause.

Modes of Mechanical Ventilation

Modes of mechanical ventilation describe how breaths are delivered to the patient. Modes of ventilation are classified as volume, pressure, or dual modes. This classification is based on the variable that the ventilator maintains at a preset value during inspiration.[50] In a volume mode of ventilation, the set V_T is maintained during inspiration. In a pressure mode of ventilation, pressure is set and does not vary throughout inspiration. An understanding of the basic volume and pressure modes of ventilation provides a solid foundation for the nurse to learn the dual modes.

Volume Ventilation

In volume ventilation, V_T is constant for every breath delivered by the ventilator. The ventilator is set to allow airflow into the lungs until a preset volume has been reached. A major advantage of this mode is that the V_T is delivered, regardless of changes in lung compliance or resistance. However, the PIP varies in this mode, depending on compliance and resistance. Assist/control (A/C; Figure 9-26, *A*) and synchronized intermittent mandatory ventilation (SIMV; Figure 9-26, *B*) are modes of volume ventilation.

Assist/control ventilation. The volume A/C (V–A/C) mode of ventilation delivers a preset number of breaths of a preset V_T. The patient may trigger additional spontaneous breaths between the ventilator-initiated breaths. When the patient initiates a breath by exerting a negative inspiratory effort, the ventilator delivers an assisted breath of the preset V_T. The V_T of the assisted breaths is constant for both ventilator-initiated and patient-triggered breaths. The V–A/C mode ensures that the patient receives adequate ventilation, regardless of spontaneous efforts. The V–A/C mode is indicated when it is desirable for the ventilator to perform the bulk of the WOB. The only work the patient must perform is the negative inspiratory effort required to trigger the ventilator on the patient-initiated breaths. The A/C mode is useful in a patient with a normal respiratory drive but whose respiratory muscles are too weak or unable to perform the WOB (e.g., patient emerging from general anesthesia or with pulmonary disease such as pneumonia).[53] A disadvantage of V–A/C ventilation is that respiratory alkalosis may develop if the patient hyperventilates because of anxiety, pain, or neurological factors. Respiratory alkalosis is treated or prevented by providing sedation or analgesia as needed, or changing to SIMV.[35] Another disadvantage is that the patient may rely on the ventilator and not attempt to initiate spontaneous breathing if ventilatory demands are met.

During V–A/C ventilation, the nurse monitors several parameters. These include the total RR, to determine whether the patient is initiating spontaneous breaths; the EV_T, to ensure that the set V_T is delivered; the PIP, to determine whether it is increasing (indicating a change in compliance or resistance, which needs to be further evaluated); the patient's sense of comfort and synchronization with the ventilator; and the acid-base status.[50]

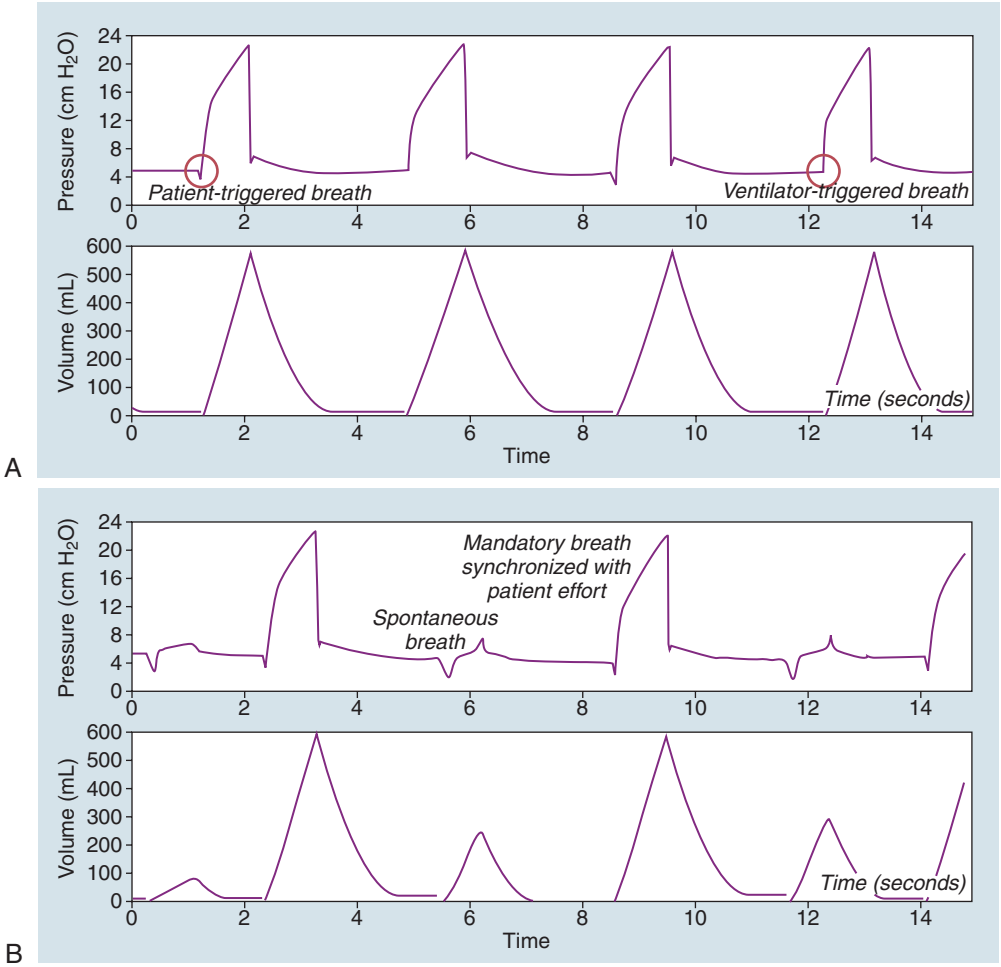

FIGURE 9-26 Waveforms of volume-controlled ventilator modes. **A,** Volume assist/control (V–A/C) ventilation. The patient may trigger additional breaths above the set rate. The ventilator delivers the same volume for ventilator-triggered and patient-triggered (assisted) breaths. **B,** Synchronized intermittent mandatory ventilation (SIMV). Both spontaneous and mandatory breaths are graphed. Mandatory breaths receive the set tidal volume (V_T). V_T of spontaneous breaths depends on work patient is capable of generating, lung compliance, and airway resistance.

Synchronized intermittent mandatory ventilation. The volume SIMV mode of ventilation delivers a set number of breaths of a set V_T, and between these mandatory breaths the patient may initiate spontaneous breaths. If the patient initiates a breath near the time a mandatory breath is due, the delivery of the mandatory breath is synchronized with the patient's spontaneous effort to prevent patient-ventilator dyssynchrony. The volume of the spontaneous breaths depends on the patient's respiratory effort. The main difference between the SIMV and V–A/C modes is the volume of the patient-initiated breaths. Patient-initiated breaths in A/C ventilation result in the patient receiving a set V_T. In SIMV, the V_T of spontaneous breaths is variable because it depends on patient effort and lung characteristics.[50,51]

The SIMV mode helps to prevent respiratory muscle weakness associated with mechanical ventilation because the patient contributes to the WOB. SIMV is indicated when it is desirable to allow patients to breathe at their own RR and assist in maintaining a normal $PaCO_2$, or when hyperventilation has occurred in the V–A/C mode. SIMV is also indicated for weaning patients from mechanical ventilation. As the SIMV rate is lowered, the patient initiates more spontaneous breaths, assuming a greater portion of the ventilatory work. As the patient demonstrates the ability to take on even more WOB, the mandatory breath rate is decreased accordingly. However, compared with other weaning modalities, SIMV is associated with the longest weaning and lowest success rate.[5,20]

During SIMV, the nurse monitors the total RR to determine whether the patient is initiating spontaneous breaths, and the patient's ability to manage the WOB. If the total RR increases, the V_T of the spontaneous breaths is assessed for adequacy. An adequate spontaneous V_T is 5 to 7 mL/kg of ideal body weight. A rising total RR may indicate that the patient is beginning to fatigue, resulting in a more shallow and rapid respiratory pattern. This pattern may lead to

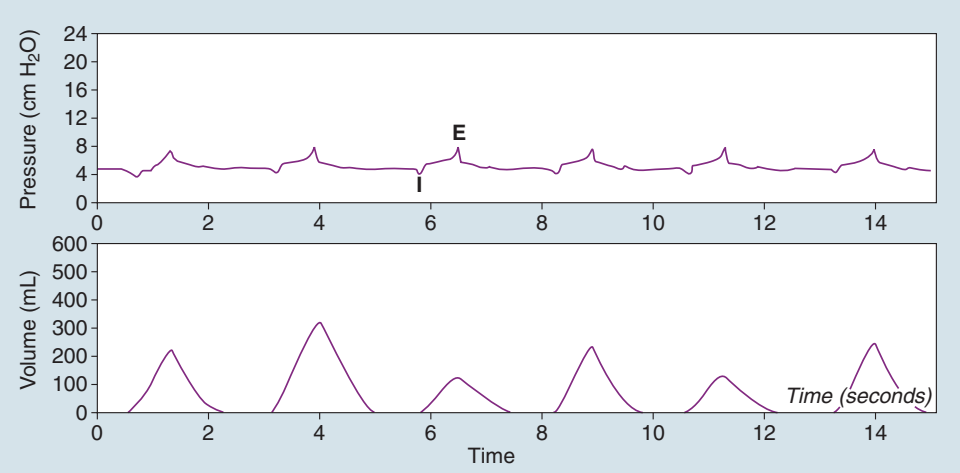

FIGURE 9-27 Continuous positive airway pressure (CPAP) is a spontaneous breathing mode. Positive pressure at end expiration splints alveoli and supports oxygenation. Note that the pressure does not fall to zero, indicating the level of CPAP. *E,* Expiration; *I,* inspiration.

atelectasis, a further increase in the WOB, and the need for greater ventilatory support.[50,51] The nurse monitors the EV_T of both the mandatory and spontaneous breaths to ensure that the set V_T is being delivered with the mandatory breaths, and that the spontaneous V_T is adequate. As in A/C ventilation, the nurse assesses the PIP, the patient's sense of comfort and synchronization with the ventilator, and the acid-base status.

Pressure Ventilation

In pressure ventilation the ventilator is set to allow air to flow into the lungs until a preset inspiratory pressure has been reached. The V_T the patient receives is variable and depends on lung compliance and airway and circuit resistance. Patients with normal lung compliance and low resistance will have better delivery of V_T for the amount of inspiratory pressure set.[35,50,51,53] An advantage of pressure-controlled modes is that the PIP can be reliably controlled for each breath the ventilator delivers. A disadvantage is that hypoventilation and respiratory acidosis may occur since delivered V_T varies; therefore, the nurse must closely monitor EV_T.[51] Pressure modes include continuous positive airway pressure, pressure support, pressure control, pressure-controlled inverse-ratio ventilation, and airway pressure–release ventilation.

Continuous positive airway pressure. Continuous positive airway pressure (CPAP) is positive pressure applied throughout the respiratory cycle to the spontaneously breathing patient (Figure 9-27). The patient must have a reliable respiratory drive and adequate V_T because no mandatory breaths or other ventilatory assistance is given. The patient performs all the WOB. CPAP provides pressure at end expiration, which prevents alveolar collapse and improves the functional residual capacity and oxygenation.[35,50] CPAP is identical to PEEP in its physiological effects. CPAP is the correct term when the end-expiratory pressure is applied in the spontaneously breathing patient. PEEP is the term used for the same setting applied to the patient is receiving an additional form of respiratory support (e.g., A/C, SIMV, pressure support). CPAP is indicated as a mode of weaning, when the patient has adequate ventilation but requires end-expiratory pressure to stabilize the alveoli and maintain oxygenation. Because the ventilator is used to deliver CPAP during weaning, the nurse can monitor the adequacy of the patient's EV_T, alarms can be set to detect low EV_T and apnea, and mechanical breaths can be delivered in the event of apnea.[51]

CPAP can also be administered via a nasal or face mask. Typically, a nasal CPAP system is used to keep the airway open in patients with obstructive sleep apnea.

Pressure support. Pressure support (PS) is a mode of ventilation in which the patient's spontaneous respiratory activity is augmented by the delivery of a preset amount of inspiratory positive pressure. PS may be used as a stand-alone mode (Figure 9-28) or in combination with other modes, such as SIMV, to augment the V_T of the spontaneous breaths (Figure 9-29). The positive pressure is applied throughout inspiration, thereby promoting the flow of gas into the lungs, augmenting the patient's spontaneous V_T, and decreasing the WOB associated with breathing through an artificial airway and the ventilatory circuit.[35,50,53] Typical levels of PS ordered for the patient are 6 to 12 cm H_2O. The V_T is variable, determined by patient effort, the amount of PS applied, and the compliance and resistance of the patient and ventilator system. EV_T must be closely monitored during PS; if it is inadequate, the level of PS is increased. PS may increase patient comfort because the patient has greater control over the initiation and duration of each breath. PS promotes conditioning of the respiratory muscles since the patient works throughout the breath; this may facilitate weaning from the ventilator.

Pressure assist/control. Pressure assist/control (P–A/C) is a mode of ventilation in which there is a set RR, and every breath is augmented by a set amount of inspiratory pressure.

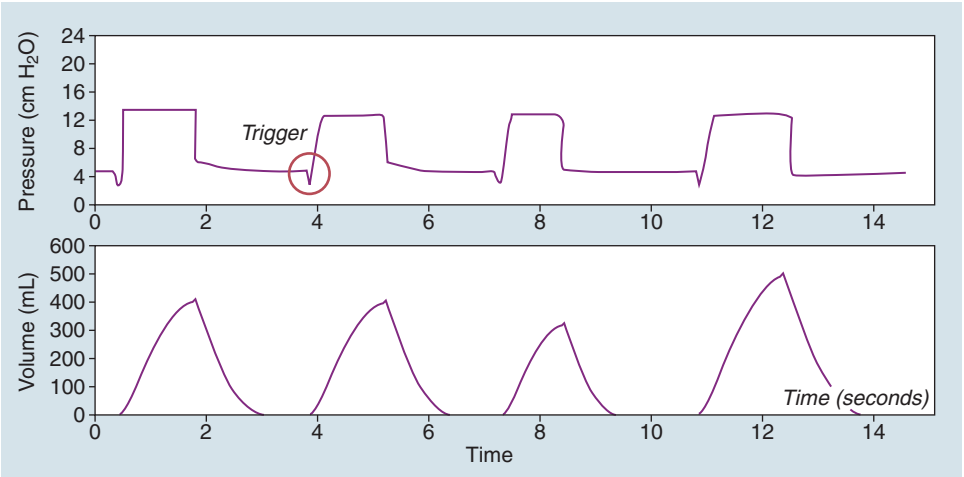

FIGURE 9-28 Pressure support ventilation requires the patient to trigger each breath, which is then supported by pressure on inspiration. Patient may vary amount of time in inspiration, respiratory rate, and tidal volume (V_T).

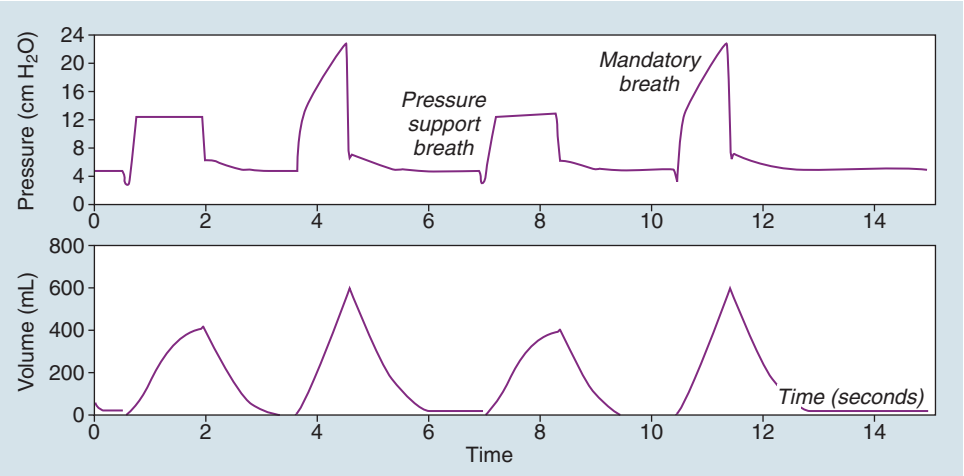

FIGURE 9-29 Synchronized intermittent mandatory ventilation (SIMV) with pressure support (PS). SIMV breaths receive set tidal volume (V_T). Pressure support is applied to the spontaneous, patient-triggered breaths.

If the patient triggers additional breaths beyond the mandatory breaths, those breaths are augmented by the set amount of inspiratory pressure (Figure 9-30). Just as with PS, there is no set V_T. The V_T the patient receives is variable and determined by the set inspiratory pressure, the patient's lung compliance, and circuit and airway resistance. The typical pressure in P–A/C ranges from 15 to 25 cm H_2O, which is higher than a PS level because P–A/C is indicated for patients with ARDS, or those with a high PIP during traditional volume ventilation. Because the lungs are noncompliant in these conditions, higher inspiratory pressure levels are needed to achieve an adequate V_T. P–A/C reduces the risk of barotrauma while maintaining adequate oxygenation and ventilation. During P–A/C, the nurse must be familiar with all the ventilator settings: the level of pressure, the set RR, the FiO_2, and the level of PEEP. The nurse monitors the total RR to evaluate whether the patient is initiating spontaneous breaths, and EV_T for adequacy of volume.[50]

Pressure-controlled inverse-ratio ventilation. With pressure-controlled inverse-ratio ventilation (PC-IRV), the patient receives P–A/C ventilation as described, and the ventilator is set to provide longer inspiratory times. The I:E ratio is inversed to increase the mean airway pressure, open and stabilize the alveoli, and improve oxygenation. PC-IRV is indicated for patients with noncompliant lungs such as in ARDS, when adequate oxygenation is not achieved despite high FiO_2, PEEP, or positioning. Because the reverse I:E ratio ventilation is uncomfortable, the patient must be sedated and possibly paralyzed to prevent ventilator dyssynchrony and oxygen desaturation.[50,53]

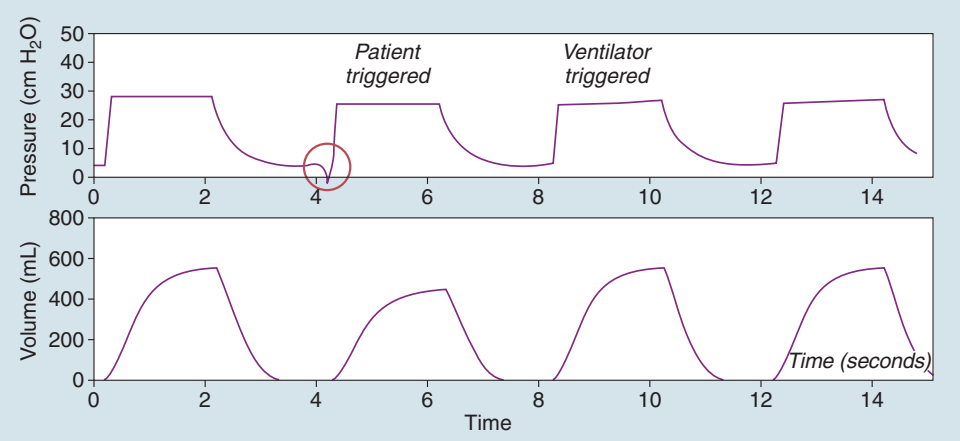

FIGURE 9-30 Pressure assist/control ventilation. Patient can trigger additional breaths above the set rate. Patient- and ventilator-triggered breaths receive the same inspiratory pressure.

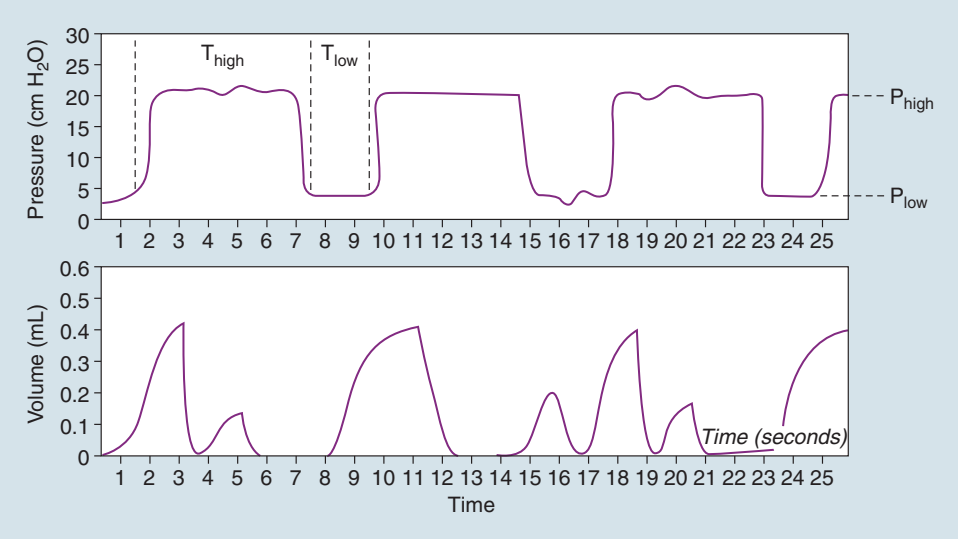

FIGURE 9-31 Airway pressure–release ventilation.

Airway pressure–release ventilation. Airway pressure–release ventilation (APRV) is a mode of ventilation that provides two levels of CPAP, one during inspiration and the other during expiration, while allowing unrestricted spontaneous breathing at any point during the respiratory cycle (Figure 9-31). APRV starts at an elevated pressure, the CPAP level or pressure high (P_{HIGH}), followed by a release pressure, pressure low (P_{LOW}). After the airway pressure release, the P_{HIGH} level is restored.[23] The time spent at P_{HIGH} is known as time high (T_{HIGH}) and is generally prolonged, 4 to 6 seconds. The shorter release period (P_{LOW}) is known as time low (T_{LOW}) and is generally 0.5 to 1.1 seconds. When observing the pressure waveform, APRV is similar to PC-IRV; however, unlike PC-IRV, the patient has unrestricted spontaneous breathing. The patient is more comfortable on APRV, and neither deep sedation nor paralysis is needed. APRV assists in providing adequate oxygenation while lowering PIP. It is indicated as an alternative to V–A/C or P–A/C for patients with significantly decreased lung compliance, such as those with ARDS.[26,35,50]

Noninvasive Positive-Pressure Ventilation

Noninvasive positive-pressure ventilation (NPPV) is the delivery of mechanical ventilation without an ETT or tracheostomy tube. NPPV provides ventilation via (1) a face mask that covers the nose, mouth, or both; (2) a nasal mask or pillow; or (3) a full face mask (Figure 9-32). Complications associated with an artificial airway are reduced, such as vocal cord injury and ventilator-associated pneumonia, and sedation needs are less. During NPPV, the patient can eat and

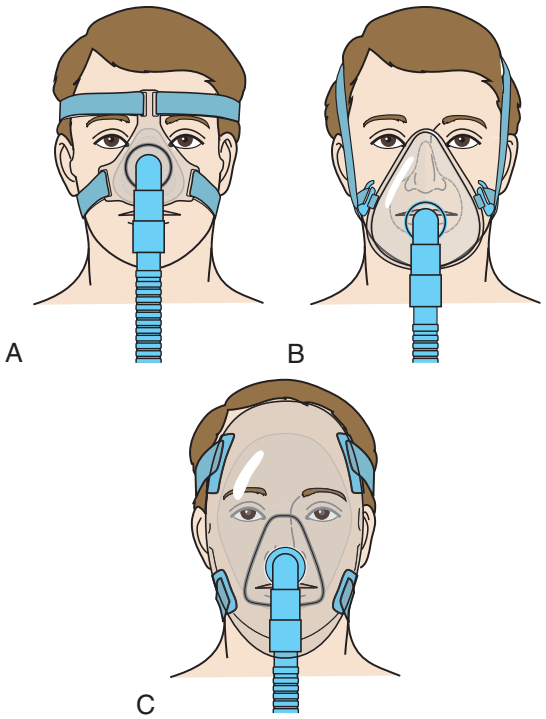

FIGURE 9-32 Masks used for noninvasive positive-pressure ventilation. **A,** Nasal. **B,** Oronasal. **C,** Total face mask. (Redrawn from Mims BC, Toto KH, Luecke LE, et al. *Critical Care Skills*. 2nd ed. Philadelphia: Saunders; 2003.)

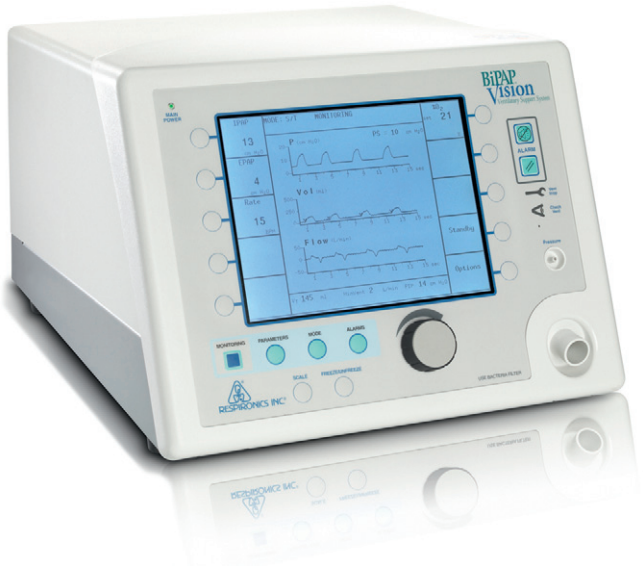

FIGURE 9-33 Noninvasive positive-pressure ventilation (NPPV) may be administered through a mask with the BiPAP Vision ventilator. This ventilator is capable of operating in four modes: pressure support (PS); spontaneous/timed (S/T) mode, which is pressure support with backup pressure control; timed (T), which is pressure control; and continuous positive airway pressure (CPAP). (Courtesy Phillips Healthcare, Andover, Massachusetts.)

speak, and is free from the discomfort of an artificial airway. Treatment with NPPV may prevent the need for intubation in many patients.

NPPV is indicated for the treatment of acute exacerbations of COPD, cardiogenic pulmonary edema (along with other treatments), early hypoxemic respiratory failure in immunocompromised patients, and obstructive sleep apnea. It may also be used to prevent reintubation in a patient who has been extubated but is having respiratory distress, and to provide ventilatory support while an acute problem is treated in patients for whom intubation is undesirable, such as those with "do not intubate" orders.[50] Contraindications to NPPV include apnea, cardiovascular instability (hypotension, uncontrolled dysrhythmias, and myocardial ischemia), claustrophobia, somnolence, high aspiration risk, viscous or copious secretions, inability to clear secretions, recent facial or gastroesophageal surgery, craniofacial trauma, and burns.[35,50]

NPPV can be delivered with critical care ventilators or a ventilator specifically designed to provide NPPV (Figure 9-33). Modes delivered can be pressure or volume; however, pressure modes are better tolerated. The most common modes of ventilation delivered via NPPV are pressure support or pressure control with PEEP and CPAP.

During NPPV, it is important for the nurse to work with the RT to ensure the right size and type of mask is chosen, and that it fits snugly enough to prevent air leaks. The nurse monitors the mask and the skin under the mask edges for signs of breakdown. If signs of excess pressure are noted, interventions include repositioning the mask, placing a layer of wound care dressing on the skin as a protective shield, or trying another type of mask. If mouth breathing is a problem with the nasal mask, a chin strap can be applied, or the mask should be changed to an oronasal or full face mask. Leakage of gases around the mask edges may lead to drying of the eyes and the need for eye drops. The mouth and airway passages should be monitored for excessive drying, and a humidification system applied as indicated. The nurse also monitors the total RR, the EV_T to ensure it is adequate, and the PIP.[50,51]

High Frequency Oscillatory Ventilation

High frequency oscillatory ventilation (HFOV) delivers subphysiological tidal volumes at extremely fast rates (300 to 420 breaths per minute). It is indicated in patients with noncompliant lungs and hypoxemia where conventional ventilation results in high airway pressures. This strategy stabilizes the alveoli and improves gas mixing, thereby improving oxygenation. The small tidal volumes limit peak pressure, preventing overdistention and protecting the lung from further injury. At the same time, collapse of the alveoli at end-expiration is limited through the use of higher end-expiratory pressure. HFOV is delivered with a specialized ventilator that uses a diaphragm, much like a stereo speaker, driven by a piston creating a constant flow of gases in and out of the lung (Figure 9-34). Ventilator settings control the amount, timing, and speed of piston movement. The nurse must learn new monitoring parameters when caring for a patient on HFOV.

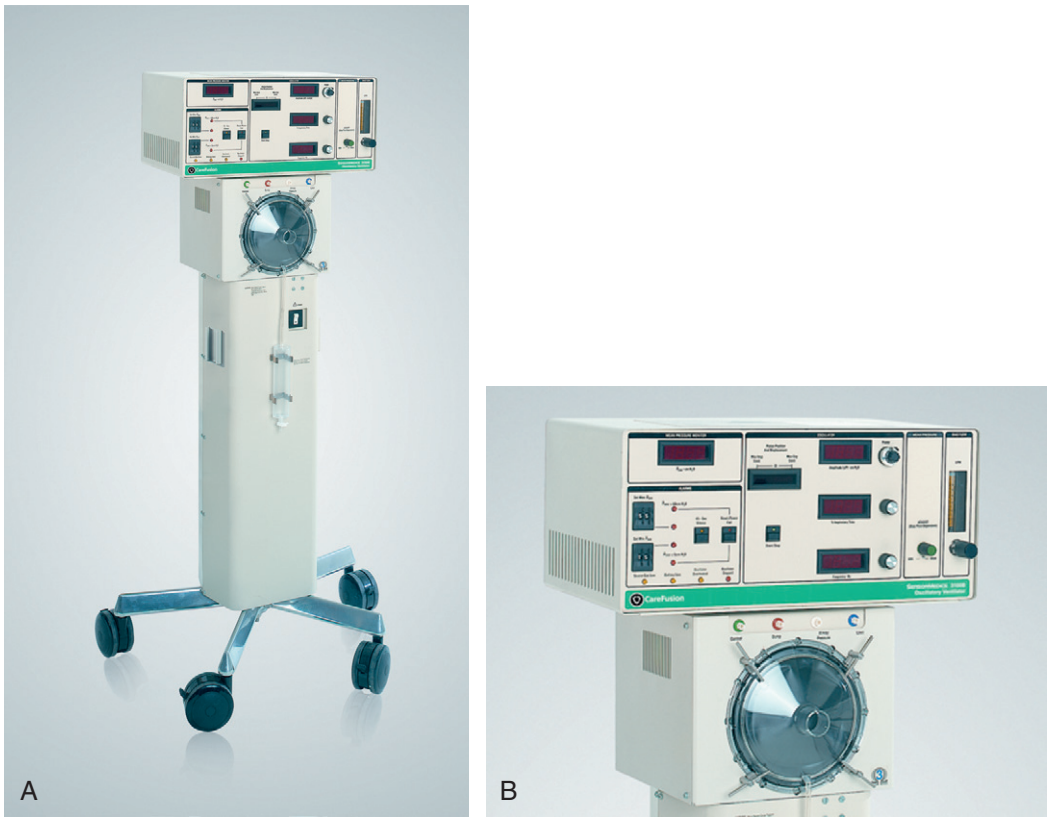

FIGURE 9-34 **A,** SensorMedics 3100B High Frequency Oscillatory Ventilator. **B,** Enlarged view. (Courtesy CareFusion, San Diego, California.)

Advanced Methods and Modes of Mechanical Ventilation

Microprocessor ventilators offer a wide range of options for mechanical ventilation. However, other forms of ventilatory support are available. These advanced techniques are usually ordered to treat patients with respiratory failure that is refractory to conventional treatment. These techniques include, but are not limited to, high-frequency jet ventilation, extracorporeal membrane oxygenation, and inhaled nitric oxide. Specialized equipment and training are essential for these advanced treatments.

Respiratory Monitoring During Mechanical Ventilation

Nurses and RTs routinely monitor many parameters while a patient receives mechanical ventilation. Monitoring done to assess the patient's response to treatment and to anticipate and plan for the ventilator weaning process includes physical assessment of the patient and assessment of the ventilator system: airway, circuitry, accuracy of ventilator settings, and patient data. Physical assessment includes vital signs and hemodynamic parameters, patient comfort and WOB, synchrony of patient's respiratory efforts with the ventilator, breath sounds, amount and quality of respiratory secretions, and assessment of the chest drain system if present. ABG results, pulse oximetry, and $ETCO_2$ values are evaluated to assess oxygenation and ventilation.[20,51,57,65] Patient data evaluated from the ventilator include EV_T (mandatory, spontaneous, and assisted breaths), total RR, and PIP. Further assessment of the PIP may require direct measurements of airway resistance and static lung compliance. The ventilator system should be checked by the nurse at least every 4 hours. The RT performs a more detailed assessment of the ventilator's functioning, including alarms and the appropriateness of alarm settings.

Alarm Systems

Alarms are an integral part of mechanical ventilation because this equipment provides vital life support functions. Alarms warn of technical or patient events that require attention or action; therefore knowledge about alarms and how to troubleshoot them is essential. Two important rules must be followed to ensure patient safety:

1. Never shut off alarms. It is acceptable to silence alarms for a preset delay while working with a patient, such as during suctioning. However, alarms are never shut off.
2. Manually ventilate the patient with a bag-valve device if unable to troubleshoot alarms quickly or if equipment failure is suspected. A bag-valve device must be readily available at the bedside of every patient who is mechanically ventilated.

TABLE 9-4 MANAGEMENT OF COMMON VENTILATOR ALARMS

ALARM	DESCRIPTION	INTERVENTION
High peak pressure	Set 10 cm H_2O above average PIP Triggered when pressure increases anywhere in circuit Ventilator responds by terminating inspiratory phase to avoid pressure injury (barotrauma)	Assess for kinks in endotracheal tube or ventilator circuit and correct Assess for anxiety and level of sedation patient biting or gagging on tube; administer medications if warranted, use airway securing device with bite block Observe for coughing, auscultate lung sounds for need for suctioning or bronchodilator Use communication assistive devices for patient who is attempting to talk Empty water from water traps if indicated Assess for worsening pulmonary pathology resulting in reduction in lung compliance (i.e., pulmonary edema) Notify RT and/or physician if alarm persists
Low pressure Low PEEP/CPAP	Set 10 cm H_2O below average PIP Set 3-5 cm H_2O below set PEEP/CPAP Triggered when pressure decreases in circuit	Assess for leaks in ventilator circuit or disconnection of ventilator circuit from airway; reconnect If malfunction is noted, manually ventilate patient with bag-mask device Notify RT to troubleshoot alarm
Low exhaled tidal volume Low minute ventilation (VE)	Set 10% below the set VT and the patients average VE Ensures adequate alveolar ventilation	Assess for disconnection of ventilator circuit from airway; reconnect Assess for disconnection in any part of the ventilator circuit; reconnect Assess for leak in cuff of artificial airway by listening for audible sounds around the airway and using device to measure cuff pressure; inflate as needed Assess for new or increasing air leak in a chest drain system; connect if system-related, notify provider if patient-related Assess for changes in lung compliance, increase in airway resistance, or patient fatigue in patient on a pressure mode of ventilation
High exhaled tidal volume High minute ventilation (VE)	Set 10% above the set VT and the patients average VE	Assess cause for increased RR or VT such as anxiety, pain, hypoxemia, metabolic acidosis; treat Assess for excess water in tubing; drain appropriately
Apnea alarm	Set for <20 seconds Warns when no exhalation detected Ventilator will default to a backup controlled mode if one is set	Assess for cause of lack of spontaneous respiratory effort (sedation, respiratory arrest, neurological condition); physically stimulate patient, encourage patient to take a deep breath, reverse sedatives or narcotics Manually ventilate patient while RT and/or provider are notified to modify ventilator settings to provide more support

CPAP, Continuous positive airway pressure; *PEEP,* positive end-expiratory pressure; *PIP,* peak inspiratory pressure; *RT,* respiratory therapist; *VE,* minute ventilation; *VT,* tidal volume.

When an alarm sounds, the first thing to do is to look at the patient. If the patient is disconnected from the ventilator circuit, quickly reconnect the patient to the machine. If the circuit is connected to the airway, quickly assess whether the patient is in distress, and whether he or she is adequately ventilated and oxygenated. The nurse quickly assesses the patient's level of consciousness, airway, RR, oxygen saturation level, heart rate, color, WOB, chest wall movement, and lung sounds. The ventilator display is observed to identify the status message related to the alarm, and the alarm is silenced while the cause of the alarm is determined. Immediate action is required if the patient is in acute distress with labored respirations, an abnormal breathing pattern, pallor and diaphoresis, deterioration in breath sounds, or decreasing SpO_2.[53] The nurse quickly disconnects the patient from the ventilator and manually ventilates with a bag-valve device while a second caregiver, often the RT, further assesses the problem. If the patient is not in respiratory distress, the nurse uses the assessment data gathered to proceed with problem solving. Table 9-4 provides an overview of management of common ventilator alarms.

Complications of Mechanical Ventilation

Numerous complications are associated with intubation and mechanical ventilation. Many complications can be prevented or treated rapidly through vigilant nursing care.[51] Best

! CLINICAL ALERT

Implementation of the Ventilator Bundle

The ventilator bundle of care should be implemented in all patients who receive mechanical ventilation.[32] This bundle is a group of evidence-based recommendations that has been demonstrated to improve outcomes. It is expected that all interventions in the bundle be implemented unless contraindicated:

- Maintain head of bed elevation at 30 to 45 degrees
- Interrupt sedation each day to assess readiness to wean from ventilator
- Provide prophylaxis for deep vein thrombosis
- Administer medications for peptic ulcer disease prophylaxis
- Daily oral care with chlorhexidine[4,32] (NOTE: The Institute of Healthcare Improvement [IHI] added this element to the ventilator bundle. Recommended chlorhexidine solution strength is 0.12%. Other bundles recommend oral care with antiseptics and do not specify a particular solution.)

practice includes implementation of the "ventilator bundle" for all mechanically ventilated patients to prevent complications and improve outcomes (see box, "Clinical Alert").

Airway Problems

Endotracheal tube out of position. The ETT can become dislodged if it is not secured properly during procedures such as oral care or changing the ETT securing device, during transport, or if the patient is anxious or agitated and attempts to pull out the tube. The ETT may be displaced upward, resulting in the cuff being positioned between or above the vocal cords. Conversely, the tube may advance too far into the airway and press on the carina or move into the right mainstem bronchus. Symptoms include absent or diminished breath sounds in the left lung and unequal chest excursion. The nurse should notify the provider of these findings so the cuff can be let down, the tube gently retracted as needed, and the cuff properly reinflated.

Whenever the ETT is manipulated, the nurse must assess for bilateral chest excursion, auscultate the chest for bilateral breath sounds after the procedure, and reassess tube position at the lip. A quick check of the centimeter markings can determine whether the tube has advanced or pulled out of proper position. When a serious airway problem cannot be quickly resolved, the nurse attempts to manually ventilate the patient to assess airway patency. If the patient cannot be ventilated and the tube is not obviously displaced or the patient is not biting the airway, the nurse should attempt to pass a suction catheter through the airway to determine whether it is obstructed. If the catheter cannot be passed and the patient has spontaneous respirations, the cuff is deflated to allow air to pass around the tube. If the patient still cannot be adequately ventilated, the airway must be removed and the patient is ventilated with a bag-valve device while preparing for emergent reintubation.[35,53]

Unplanned extubation. The patient may intentionally or inadvertently remove the airway. The two most frequent methods by which self-extubation occurs are (1) by using the tongue, and (2) by leaning forward or scooting downward so the patient uses his or her hands to remove the tube.[4,54] Unplanned extubation can also occur as a result of patient care. For example, the tube can be dislodged if the ventilator circuit or closed suction catheter pulls on the ETT during procedures such as turning. Despite vigilant nursing care, unplanned extubation may result. Strategies for preventing an unplanned extubation are described in Box 9-8.

Laryngeal and tracheal injury. Damage to the larynx and trachea can occur because of tube movement and excess pressure exerted by the distal cuff. The nurse should prevent the patient from excessive head movement, especially flexion and extension, which result in the tube moving up and down in the airway, causing abrasive injury. An intervention for preventing tracheal damage from the cuff is routine cuff pressure monitoring (Figure 9-35). Pressures should not exceed 25 to 30 cm H_2O (18 to 22 mm Hg).[58,61,64,66] Various commercial devices are available to measure cuff pressures quickly and easily. The nurse works with the RT to ensure an appropriate cuff volume and pressure.

Damage to the oral or nasal mucosa. Tape or commercial devices that secure the ETT can cause breakdown of the lip and oral mucosa. Nasal intubation may result in skin breakdown on the nares and also a higher risk of sinusitis. Ongoing assessment and skin care assist in preventing damage to the mouth and nose. The ETT should be repositioned daily to prevent pressure necrosis.

BOX 9-8 STRATEGIES FOR UNPLANNED OR SELF-EXTUBATION

- Provide patient education regarding the purpose of the artificial airway and reassurance that it will be removed as soon as the patient can breathe independently.
- Provide adequate analgesia and sedation.
- Monitor all intubated patients vigilantly; assess for risks for self-extubation (distress, disorientation).
- Apply protective devices (e.g., soft wrist restraints, arm immobilizers, mitts) according to hospital standards of practice.
- Adequately secure the endotracheal tube around the patient's head, not just to the face.
- Cut the end of the endotracheal tube to 2 inches beyond the fixation point.
- Provide support for the ventilator tubing and closed suction systems; keep these items out of the patient's reach.
- Use two staff members when repositioning an endotracheal tube.
- Educate the family to assist in monitoring the patient.
- Extubate the patient in a timely manner when the patient meets established criteria.

FIGURE 9-35 Monitoring endotracheal tube cuff pressures. (Reprinted with permission, Cleveland Clinic Center for Medical Art & Photography © 2011-2012. All rights reserved.)

PULMONARY SYSTEM

Trauma. *Barotrauma,* which means pressure trauma, is the injury to the lungs associated with mechanical ventilation. In barotrauma, alveolar injury or rupture occurs as a result of excessive pressure, excessive peak inflating volume (volutrauma), or both.[7,35] Barotrauma may occur when the alveoli are overdistended, such as with positive-pressure ventilation, PEEP, and high V_T. The alveoli rupture or tear so that air escapes into various parts of the thoracic cavity, causing subcutaneous emphysema (air in the tissue space), pneumothorax or tension pneumothorax, pneumomediastinum, pneumopericardium, or pneumoperitoneum. Signs and symptoms of barotrauma include high PIP and mean airway pressures, decreased breath sounds, tracheal shift, subcutaneous crepitus, new air leak or increase in air leak in a chest drainage system, and symptoms associated with hypoxemia.

A life-threatening complication is a tension pneumothorax. When tension pneumothorax occurs, pressurized air enters the pleural space. Air is unable to exit the pleural space and continues to accumulate. Air in the pleural space causes an increase in intrathoracic pressure, increasing amounts of lung collapse, shifting of the heart and great vessels to the opposite thorax (mediastinal shift), tachycardia, and hypotension. Treatment consists of immediate insertion of a chest tube or a needle thoracostomy. Whenever a pneumothorax is suspected in a patient receiving mechanical ventilation, the patient should be removed from the ventilator and ventilated with a bag-valve device until a needle thoracostomy is performed or a chest tube is inserted.

Lung tissue injury induced by local or regional overdistending volume is called *volutrauma*. The damage that occurs to the lung is similar to the pathological findings of early ARDS and is probably the result of local stress and strain on the alveolar-capillary membrane. Volutrauma results in increased permeability of the alveolar-capillary membrane, pulmonary edema, accumulation of white blood cells and protein in the alveolar spaces, and reduced surfactant production. Because it is difficult to determine the exact distribution of volume in a patient's lung, pressure is used as a surrogate for volume. The PIP is kept below 40 cm H_2O and/or the Pplat is kept at less than 30 cm H_2O as lung protective strategies to prevent both volutrauma and barotrauma.[35]

Oxygen toxicity. The exposure of the pulmonary tissues to high levels of oxygen can lead to pathological changes. The degree of injury is related to the duration of exposure and to the FiO_2, not to the PaO_2. The first sign of oxygen toxicity, tracheobronchitis, is caused by irritant effects of oxygen. Prolonged exposure to high FiO_2 may lead to changes in the lung that mimic ARDS. As a general rule, an FiO_2 up to 1.0 may be tolerated for up to 24 hours. However, the goal is to lower the FiO_2 to less than 0.60.[30] Absorption atelectasis is another problem associated with high FiO_2. Nitrogen is needed to prevent collapse of the alveoli. When the FiO_2 is 1.0, alveolar collapse and atelectasis result from a lack of nitrogen in the distal air spaces.

Respiratory acidosis or alkalosis. Acid-base disturbances may occur secondary to V_T and RR settings on the ventilator. For example, if a patient is receiving A/C ventilation set at 10 breaths per minute but the patient's RR is 28 breaths per minute because of pain or anxiety, respiratory alkalosis may occur. If the ventilator is set at a low RR (e.g., 2 to 6 breaths per minute) and the patient does not have an adequate drive to initiate additional breaths, respiratory acidosis may occur. Ideally the V_T and RR are set to achieve a VE that ensures a normal $PaCO_2$ level.

Infection. Patients with artificial airways who are receiving mechanical ventilation are at an increased risk of ventilator-associated pneumonia (VAP) because normal upper airway defense mechanisms are bypassed. About 10% to 20% of ventilated patients develop VAP.[56] The incidence is highest in the first 5 days of mechanical ventilation.[2] The principal mechanism for the development of VAP is aspiration of colonized gastric and oropharyngeal secretions. Factors that contribute to VAP include poor oral hygiene, aspiration, contaminated respiratory therapy equipment, poor hand washing by caregivers, breach of aseptic technique when suctioning, inadequate humidification or systemic hydration, and decreased ability to produce an effective cough because of the

artificial airway.[44] Specific strategies to reduce VAP include the following:

- Elevate head of the bed 30 to 45 degrees if not medically contraindicated to prevent reflux and aspiration of gastric contents. Elevation of the head of the bed is associated with a 26% risk reduction in pneumonia.[16]
- Prevent drainage of ventilator circuit condensate into the patient's airway. Always discard condensate and never drain it back into the humidifier.[11]
- Practice proper hand hygiene and wear gloves when handling respiratory secretions.
- Use an ETT with a lumen for aspirating subglottic secretions that pool above the airway cuff.[11]
- Ensure secretions are aspirated from above the cuff before cuff deflation or tube removal.[11]
- Provide comprehensive oral hygiene that includes a mechanism for dental plaque removal and reduction of bacterial burden in the oral cavity.[11]
- Use noninvasive mechanical ventilation when possible.

Determination of VAP has low sensitivity and specificity. The Centers for Disease Control along with a team of experts has recommended surveillance for ventilator-associated conditions, including infectious and non-infectious causes. See box, "Clinical Alert, Ventilator-Associated Conditions".

! CLINICAL ALERT

Ventilator-Associated Conditions

The Centers for Disease Control has proposed surveillance for ventilator-associated conditions (VAC), including infectious and non-infectious types. Following a baseline period of stability or improvement for 2 or more days on mechanical ventilation, VAC will be determined by indicators of worsening oxygenation: 1) Need to increase FiO_2 by .20 or higher for two or more days; or 2) Need to increase PEEP by 3 cm H_2O for two or more days.

From http://www.cdc.gov/nhsn/PDFs/vae/CDC_VAE_CommunicationsSummary-for-compliance_20120313.pdf. Accessed May 20, 2012.

Dysphagia and aspiration. Artificial airways increase the risk of upper airway injury which in turn affects upper airway mechanics and protective reflexes and can have negative effects on swallowing physiology.[3] Patients intubated 48 hours or longer are at risk for disordered swallowing which can lead to aspiration and pneumonia. Reports of dysphagia following intubation vary widely, ranging from 3% to 83%, and are mostly due to a wide assortment of assessment methods and instruments.[62] Although many factors contribute to dysphagia, the artificial airway interferes with the ability to execute an efficient and safe swallow. After extubation or tracheostomy, the transition to oral feedings may be appropriate; however, before oral feedings are initiated a speech therapy evaluation for swallowing is recommended, because many patients have difficulty with swallowing and are prone to aspiration after prolonged intubation.[3] Simple bedside tests for dysphagia before and after extubation are emerging as methods to identify this potentially serious complication.[13]

CARDIOVASCULAR SYSTEM

Hypotension and decreased cardiac output may occur with mechanical ventilation and PEEP, secondary to increased intrathoracic pressure, which can result in decreased venous return. The hemodynamic effects of mechanical ventilation are more pronounced in patients with hypovolemia or poor cardiac reserve. Patients with a high PIP who receive PEEP of greater than 10 cm H_2O may need a hemodynamic monitor to assess volume status and cardiac output. Management of hypotension and decreased cardiac output involves the administration of volume to ensure an adequate preload, followed by administration of inotropic agents as necessary.

Gastrointestinal System

Stress ulcers and gastrointestinal bleeding may occur in patients who undergo mechanical ventilation. All patients undergoing mechanical ventilation should receive medications for stress ulcer prophylaxis.[32] Enteral feeding is initiated as soon as possible, and the patient is monitored for gross and occult blood in the gastric aspirate and stools. Other interventions include identification and reduction of stressors, communication and reassurance, and administration of anxiolytic or sedative agents, as necessary based on standardized assessment tools (see Chapter 5).

Nutritional support is required for all patients who require mechanical ventilation (see Chapter 6). Inadequate nutrition may occur if the patient is not started on early nutritional support or receives inadequate supplemental nutrition.[47] The type of formula may need to be modified for ventilated patients. Excess CO_2 production may occur with high-carbohydrate feedings and place a burden on the respiratory system to excrete the CO_2, increasing the WOB.[47] Formulas developed for the patient with pulmonary disorders may be indicated.[42]

An essential nursing intervention for the intubated patient who receives enteral nutrition is to reduce the risk of aspiration. The nurse must keep the head of the bed elevated at least 30 degrees during enteral feeding.[16]

Psychosocial Complications

Several psychosocial hazards may occur because of mechanical ventilation. Patients may experience stress and anxiety because they require a machine for breathing. If the ventilator is not set properly or if the patient resists breaths, patient-ventilator dyssynchrony may occur. The noise of the ventilator and the need for frequent procedures, such as suctioning, may alter sleep and wake patterns. In addition, the patient can become psychologically dependent on the ventilator.[23]

NURSING CARE

Nursing care of the patient who requires mechanical ventilation is complex. The nurse must provide care to the patient by using a holistic approach, including competent delivery of a highly sophisticated technology. A detailed plan of care is described in the box, "Nursing Care Plan for the Mechanically Ventilated Patient."

Text continued on p. 212

NURSING CARE PLAN

for the Mechanically Ventilated Patient

NURSING DIAGNOSIS

Impaired Spontaneous Ventilation related to respiratory muscle fatigue, acute respiratory failure, metabolic factors

PATIENT OUTCOMES

- Spontaneous ventilation with normal ABGs; free of dyspnea or restlessness
- No complications associated with mechanical ventilation

NURSING INTERVENTIONS	RATIONALES
• Have bag-valve device and suctioning equipment readily available	• Be prepared in the event of airway incompetency; maintain airway patency
• Maintain artificial airway; secure ETT or tracheostomy with tape or commercial devices; prevent unplanned extubation (see Box 9-8)	• Ensure maintenance of an adequate airway to facilitate mechanical ventilation; prevent unintended removal of artificial airway

• Assess position of artificial airway:
  • Auscultate for bilateral breath sounds
  • Evaluate placement on chest x-ray
  • Once proper position is confirmed, mark the position (cm marking) of the ETT with an indelible pen and note position of the tube at the lip line as part of routine assessment
	• Maintain an adequate airway by ensuring that artificial airway in the proper position

• Monitor oxygenation and ventilation at all times, and respond to changes:
  • Vital signs
  • Total respiratory rate
  • Exhaled tidal volume of ventilator-assisted and patient-initiated breaths
  • Oxygen saturation
  • End-tidal CO_2
  • Mental status and level of consciousness
  • Signs and symptoms of hypoxemia (see Box 9-1)
  • ABGs
	• Ensure adequate oxygenation, ventilation and acid-base balance; identify when ventilator setting changes are indicated

• Assess respiratory status at least every 4 hours and respond to changes:
  • Breath sounds anteriorly and posteriorly
  • Respiratory pattern
  • Chest excursion
  • Patient's ability to initiate a spontaneous breath
	• Ensure patient is breathing comfortably and is not expending excessive energy on the work of breathing; identify reportable changes

• Reposition ETT from side to side every 24 hours; assess and document skin condition[66]
  • Note placement of tube at lip line
  • Use two staff members for procedure
  • Suction secretions above the ETT cuff before repositioning tube
  • After the procedure, assess position of tube at lip and auscultate for bilateral breath sounds
	• Prevent skin breakdown from the tube, tape, or airway securing device; prevent aspiration of oral secretions and ventilator-associated pneumonia; and ensure that tube remains in proper position after manipulation

• Monitor cuff pressure of ETT or tracheostomy and maintain within therapeutic range[61]	• Prevent complications associated with overinflation or underinflation of ETT cuff
• Maintain integrity of mechanical ventilator circuit; monitor ventilator settings; respond to ventilator alarms; keep tubing free of moisture by draining away from the patient and using devices such as water traps to facilitate drainage of moisture	• Ensure safe administration of mechanical ventilation; maximize ventilation and prevent aspiration of contaminated condensate
• Monitor serial chest radiographs	• Assess for correct position of ETT and improvement or worsening of pulmonary conditions

• Implement a multiprofessional plan of care to address underlying pulmonary condition
  • Coordinate with RT, MD, and multiprofessional team
  • Evaluate response to lung expansion bronchial hygiene and pulmonary medication therapies
  • Mobilize patient as much as possible (i.e., turning, progressive upright mobility, lateral rotation therapy)
  • Consider pronation therapy in refractive hypoxemia
  • Ensure adequate hydration, nutrition, and electrolyte balance
	• Mechanical ventilation only supports the respiratory system until the underlying condition is treated or resolved; well-coordinated team effort is essential to avoid fragmentation of care

NURSING CARE PLAN—cont'd

for the Mechanically Ventilated Patient

NURSING INTERVENTIONS	RATIONALES
• Implement a multiprofessional plan of care to maintain patient comfort, mobility, nutrition, and skin integrity; support patient and family	• Prevent complications associated with mechanical ventilation and bed rest; foster patient and family well-being

NURSING DIAGNOSIS

Ineffective Airway Clearance related to ETT, inability to cough, thick secretions, fatigue

PATIENT OUTCOMES

- Airway free of secretions
- Clear lung sounds

NURSING INTERVENTIONS	RATIONALES
• Assess need for suctioning (rising PIP, high pressure or low exhaled V_T alarm on ventilator, audible/visible secretions, rhonchi on auscultation)	• Indicate possibility of airway obstruction with secretions and need for suctioning
• Suction as needed according to standard of practice (see Box 9-7)	• Remove secretions; maintain patent airway; improve gas exchange
• Assess breath sounds, PIP and exhaled V_T after suctioning	• Assess effectiveness of suctioning; breath sounds should improve, PIP should decrease in volume mode, EVT should increase
• If tracheal secretions are thick, assess hydration of patient and humidification of ventilator; avoid instillation of normal saline	• Assist in thinning secretions for easier removal; saline has not shown to be effective and is associated with hypoxemia and increased risk of infection
• Reposition the patient frequently	• Mobilize secretions, improve gas exchange

NURSING DIAGNOSIS

Risk for Infection related to endotracheal intubation and Risk for Aspiration of oropharyngeal secretions

PATIENT OUTCOMES

- Absence of ventilator-associated pneumonia

NURSING INTERVENTIONS	RATIONALES
• Maintain head of bed at 30 degrees or greater	• Decrease risk for aspiration of oropharyngeal and gastric secretions
• Monitor temperature every 4 hours; assess amount, color, consistency, and odor of secretions; notify physician if secretions change	• Identify signs of infection
• Use good hand-washing techniques; wear gloves for procedures, including closed suctioning; use aseptic technique for suctioning	• Prevent transmission of bacteria to the patient
• Implement a comprehensive oral care protocol that includes oral suction at least every 4 hours and brushing teeth at least every 12 hours	• Remove dental plaque and bacteria from the oropharynx, and prevent aspiration of contaminated oral secretions
• Maintain integrity of ETT cuff; keep cuff pressure between 20 and 30 cm H_2O	• Prevent aspiration of oropharyngeal secretions

NURSING DIAGNOSIS

Risk for Ineffective Protection related to ventilator dependence, PEEP, decreased pulmonary compliance, and issues related to mechanical ventilator (settings, alarms, disconnection)

PATIENT OUTCOME

- Free of ventilator-induced lung injury

NURSING INTERVENTIONS	RATIONALES
• Assess prescribed ventilator settings every 2 hours (mode, set rate, VT, FiO_2, PEEP); ensure that alarms are on	• Ensure that patient is receiving therapy as ordered; promote patient safety
• Assess PIP at least every 4 hours	• Identify elevations in PIP, which may indicate worsening lung function, need to adjust pulmonary therapies or ventilator settings to ensure PIP does not exceed 40 cm H_2O

Continued

NURSING CARE PLAN—cont'd

for the Mechanically Ventilated Patient

NURSING INTERVENTIONS	RATIONALES

• Assess tolerance to ventilatory assistance and monitor for patient-ventilator asynchrony; notify RT and licensed provider of potential need to adjust ventilator settings
  • Patient's respiratory cycle out of phase with ventilator
  • High pressure, low EVT alarms
  • Subjective report of breathlessness
  • Labored respirations, especially increased effort on inspiration
  • Tachypnea
  • Anxiety, agitation
	• Provide cues of condition improving or worsening; may indicate need for suctioning or need to adjust ventilator settings that are insufficient to meet patients ventilatory needs

• Assess for signs of pneumothorax every 4 hours or with changes; if symptomatic, determine need for manual ventilation and prepare for chest tube insertion:
  • Subcutaneous crepitus
  • Unequal chest excursion
  • Unilateral decrease in breath sounds
  • Restlessness
  • Increasing PIP
  • Tracheal shift
  • Decreasing SpO_2
	• Early detection of impaired ventilation so emergency chest tube insertion can be efficiently performed

• Respond to all ventilator alarms	• Provide immediate intervention in response to specific alarm; promote patient safety

NURSING DIAGNOSIS

Anxiety and related to need for mechanical ventilation, impaired verbal communication, change in environment, unmet needs, fear of death

PATIENT OUTCOMES

- Calm and cooperative

NURSING INTERVENTIONS	RATIONALES
• Assess patient every 4 hours for signs of anxiety; administer sedation as indicated and assess response using standardized sedation assessment tools	• Identify presence or absence (or relief) of anxiety; standardized tools facilitate communication among team members and assessment of trends
• Collaborate with physician to develop a sedation plan if anxiety or agitation that is not due to pain or delirium impairs ventilation	• Promote effectiveness of mechanical ventilation and patient ventilator synchrony; pain is managed with analgesics, delirium will be worsened by sedation
• Assess respiratory pattern for synchrony with ventilator	• Respiratory efforts that are asynchronous with the ventilator result in discomfort, anxiety, dyspnea, and abnormal ABGs

• Talk to patient frequently; establish method for communication that is appropriate for the patient's native language and abilities; speak slowly and do not shout; expect frustration
  • Yes/no questions
  • Clipboard with paper and pencil
  • Picture communication boards
  • Computerized systems
  • Lip reading
  • Devices that allow the patient to speak
	• Promote communication with the patient and help to identify needs, assess responses to treatment, and reduce anxiety; strategies must be culturally appropriate to facilitate patient understanding

• Implement interventions to reduce anxiety:
  • Calm and reassuring presence
  • Simple explanations before and during procedures
  • Call light within reach
  • Family visitation
  • Diversionary activities, such as music or television at appropriate intervals
  • Promote regular sleep/wake cycle and daytime activity
	• Assist in preventing and/or relieving fear and anxiety
	• Promote rest, healing and recovery

NURSING CARE PLAN—cont'd

for the Mechanically Ventilated Patient

NURSING INTERVENTIONS	RATIONALES
• Collaborate with the healthcare team to develop strategies to reduce anxiety and maximize effectiveness of mechanical ventilation: changes in settings, sedation, analgesia, complementary and alternative therapies	• Medications are frequently needed as an adjunct during mechanical ventilation; complementary therapies such as touch, massage, reflexology, music therapy, and meditation may also be effective

NURSING DIAGNOSIS

Risk for Decreased Cardiac Output related to effects of positive pressure ventilation, PEEP, volume depletion or overload

PATIENT OUTCOME

- Adequate cardiac output

NURSING INTERVENTIONS	RATIONALES
• Assess for hypotension, tachycardia, dysrhythmias, decreased level of consciousness, cool skin, mottling	• Indicate decreased cardiac output
• Measure hemodynamic profile at least every 4 hours if hemodynamic monitoring device is in place; reassess after any ventilator setting changes that affect V_T, PEEP, or PIP	• Assess filling pressure and cardiac output, and identify trends
• Alert the physician to changes in cardiac output and hemodynamic profile	• Ventilator settings, especially PEEP, may need to be adjusted; therapies to improve oxygenation may need to be added (lung expansion, bronchial hygiene, rotation, placing prone, etc.) so PEEP can be reduced and oxygenation maintained
• Maintain optimum fluid balance	• Additional volume may be needed, especially if patient is receiving PEEP; fluid retention may also occur
• Administer other medications as ordered (e.g., inotropic agents or diuretics)	• Medications may be needed to optimize cardiac output and/or relieve fluid retention

NURSING DIAGNOSIS

Dysfunctional Ventilatory Weaning Response related to ineffective airway clearance, sleep-pattern disturbances, inadequate nutrition, pain, anemia, abdominal distention, debilitated condition, and psychological factors

PATIENT OUTCOME

- Liberation from mechanical ventilation
- Adequate ABG values
- Respiratory pattern and rate WNL
- Effective secretion clearance

NURSING INTERVENTIONS	RATIONALE
• Assess patient's readiness to wean (see Box 9-10)	• Identify readiness to begin the weaning process using validated parameters
• Provide weaning method based on protocols and research evidence (see Box 9-9)	• Protocol-driven weaning is an effective strategy for systematic ventilator liberation that reduces ventilator days and ICU and hospital length of stay
• Collaborate with the healthcare team to provide mechanical ventilation modes, patient coaching, and progressive mobility that supports respiratory muscle training	• Promote respiratory conditioning that facilitates patient's ability to resume the work of breathing
• Promote rest and comfort throughout the weaning process, especially between weaning trials; identify strategies that result in relaxation and comfort; ensure that environment is safe and comfortable	• Facilitate weaning from mechanical ventilation
• Support patients in setting goals for weaning	• Promote rehabilitation and give patients some control in the process
• Collaborate with the healthcare team to determine the most effective strategies for weaning those with severe dysfunctional breathing patterns	• Various strategies may be needed to wean the patient; ongoing assessment is essential to determine the most effective strategy

Continued

NURSING CARE PLAN—cont'd

for the Mechanically Ventilated Patient

NURSING INTERVENTIONS	RATIONALES

• Implement strategies that maximize tolerance of weaning:
  • Titrate sedation and analgesia to a level at which patient is calm and cooperative with absence of respiratory depression
  • Schedule when patient is rested
  • Avoid other procedures during weaning
  • Position patient upright to allow for full expansion with abdominal compression on diaphragm
  • Promote normal sleep-wake cycle
  • Limit visitors to supportive persons
  • Coach through periods of anxiety
	• Strategies assist in ensuring that patient is rested, with an adequate level of consciousness and decreased anxiety and in an optimal position for lung expansion; weaning efforts will be maximized

• Terminate weaning if patient is unable to tolerate the process (see Box 9-11)	• Maintain adequate ventilation and gas exchange; prevent fatigue of respiratory muscles
• Consider referring patients with prolonged ventilator dependence to an alternative setting	• Alternative settings specialize in weaning patients who are "difficult to wean"

ABG, Arterial blood gas; *CO,* cardiac output; *CO_2,* carbon dioxide; *ETT,* endotracheal tube; *FiO_2,* fraction of inspired oxygen; *ICU,* intensive care unit; *PEEP,* positive end-expiratory pressure; *PIP,* peak inspiratory pressure; *PS,* pressure support; *RR,* respiratory rate; *SpO_2,* oxygen saturation as measured by pulse oximetry; *V_T,* tidal volume; *WNL,* within normal limits.
Based on data from Gulanick M, & Myers JL. (2011). *Nursing Care Plans: Diagnoses, Interventions, and Outcomes* (7th ed.). St. Louis: Mosby.

Communication

Communication difficulties are common because of the artificial airway. The lack of vocal expression has been identified by patients as a major stressor that elicits feelings of panic, isolation, anger, helplessness, and sleeplessness.[24] Patients express a need to know and to make themselves understood. They need constant reorientation, reassuring words emphasizing a caregiver's presence, and point-of-care information that painful procedures done to them are indeed necessary and helpful. In addition, touch, eye contact, and positive facial expressions are beneficial in relieving anxiety.[44] Caregivers who attempt to individualize communication with intubated patients by using a variety of methods provide patients a greater sense of control, encourage participation in their own care, and minimize cognitive disturbances.[24]

Head nods, mouthing words, gestures, and writing are identified as the most frequently used method of nonverbal communication among intubated ICU patients, but they are often inhibited by wrist restraints.[29] Communication with gestures and lip reading can convey some basic needs; however, augmentative devices may facilitate even better communication. Although writing is sometimes used, critically ill patients are often too weak or poorly positioned to write, or they lack the concentration to spell. A picture board with icons representing basic needs and the alphabet that can be easily cleaned between patients should be available in every ICU. A board with pictures improved communication for patients after cardiothoracic surgery and was preferred by a small group of critical care survivors who were interviewed about augmentative communication methods.[48] Family members can serve as a communication link between the patient and the care providers. It is important to reassure the patient that the loss of their voice is temporary and that speech will be possible after the tube is removed.

Maintaining Comfort and Reducing Distress

Intubation, mechanical ventilation, advanced methods for ventilation (e.g., inverse-ratio ventilation), and suctioning contribute to patient discomfort and distress. Patients often need both pharmacological and nonpharmacological methods to manage discomfort and to treat anxiety.[67] Strategies to promote patient comfort are discussed in-depth in Chapter 5.

Medications

Commonly used medications include analgesics, sedatives, and neuromuscular blocking agents; many patients need a combination of these drugs.[36,38] Medications are chosen based on the hemodynamic stability of the patient, the diagnosis, and the desired patient goals and outcomes. It is very important that the nurse, RT, and physician all use the same objective sedation and analgesia scoring systems to promote unambiguous assessment and communication. In some institutions, nurses use decision trees or algorithms to guide initiation and titration of medications to targeted sedation and analgesia goals.[6] Medications are tapered or discontinued when the patient is ready to be weaned from mechanical ventilation.

Analgesics, such as morphine and fentanyl, are administered to provide pain relief. Sedatives, such as dexmedetomidine, benzodiazepines, and propofol, are given to sedate the patient, reduce anxiety, and promote synchronous breathing with the ventilator. Benzodiazepines promote amnesia but are also associated with an increase in delirium.[33] Patients who have acute lung injury or increased intracranial pressure,

or who require nontraditional modes of mechanical ventilation may require deep sedation or therapeutic paralysis with neuromuscular blocking agents. Chemical paralysis must be discontinued before attempting to wean the patient from mechanical ventilation.[33]

When sedation of the mechanically ventilated patient is indicated it must be titrated to a specific goal agreed upon by the multiprofessional team. Insufficient sedation may precipitate ventilator dyssynchrony and physiological alterations in thoracic pressures and gas exchange. Inadequate sedation is also associated with unplanned extubation. Oversedation and prolonged sedation are associated with a longer duration of mechanical ventilation and lengths of stay in the critical care unit and hospital.[36] Prolonged duration of mechanical ventilation predisposes the patient to an increased risk of VAP, lung injury, and other complications. Depth of sedation also contributes to delayed weaning from mechanical ventilation. Since sedation, duration of mechanical ventilation, and ventilator weaning are tightly interrelated, the nurse must ensure that the patient is maintained on the lowest dose and lightest level of sedation as possible. "Daily interruption," "sedation vacation," or a "spontaneous awakening trial" to evaluate the patient's cognitive status, to reduce the overall dose of sedation, and to determine what dose, if any, sedation is needed to achieve a calm, cooperative patient is an important nursing intervention.[33,37] Optimal sedation of the mechanically ventilated patient is present when the patient resides at a state in which patient-ventilator harmony exists and the patient remains capable of taking spontaneous breaths in readiness for weaning, when appropriate.[37,38] Some patients may achieve this state without sedative agents. Administering sedatives intermittently and as needed rather than continuously is another strategy the nurse should consider.

Nonpharmacological Interventions

Nonpharmacological, complementary, and alternative medicine strategies can be used to reduce distress, promote patient-ventilator synchrony, and maintain a normal cognitive state.[67] The nurse creates a healing environment by involving the patient and family in the plan of care, reducing excess noise and light stimulation, providing a reassuring presence, and minimizing unnecessary patient stimulation to promoting a normal sleep-wake cycle. Adequate rest and frequent reorientation are also important for the prevention of delirium. A progressive mobility plan reduces deconditioning and promotes endurance of the respiratory muscles to facilitate ventilator liberation. Daytime exercise may also promote a more restful nighttime sleep.

Complementary and alternative strategies may also be helpful in reducing distress and promoting rest. Examples of these strategies include meditation, guided imagery and relaxation, prayer, music therapy, massage, accupressure, therapeutic touch, herbal products and dietary supplements, and presence.[67] Nurses should ask the family and patient if they are already using complementary strategies and, if so, incorporate them as possible. The goal of learning to incorporate these therapies into practice is to reduce patient distress, promote sleep, and create a healing environment conducive to reducing ventilator days.

WEANING PATIENTS FROM MECHANICAL VENTILATION

Mechanical ventilation is a therapy designed to support the respiratory system until the underlying disease or indication for mechanical ventilation is resolved. The team caring for a ventilated patient should always be planning for how the patient will be weaned or "liberated" from the ventilator. Another term for liberation is discontinuation of ventilator support. In general, patients who require short-term ventilatory support, defined as 3 days or less of mechanical ventilation, are weaned quickly.[9,40] Conversely, weaning patients who require long-term ventilatory support, is usually a slower process and may be characterized by periods of success as well as setbacks. Reduction of ventilator support can be done as the patient demonstrates the ability to resume part of or all of the WOB.

Approach to Weaning Using Best Evidence

A systematic approach to weaning is indicated for patients. Based on a comprehensive review of the research, evidenced-based guidelines for ventilator weaning were developed.[39-41] Box 9-9 summarizes these guidelines. Weaning protocols managed by nurses and RTs, as compared with traditional weaning directed by physicians, result in a reduction of ventilator days and shorter stays in the ICU and hospital.[37] The protocol should clearly define the method or screening tool to determine the patient's readiness to wean, the method and duration of the weaning trial, and when to terminate a weaning trial versus proceed with requesting an order for extubation. The weaning plan should include methods to facilitate respiratory muscle work along with adequate rest.[40,51] See the box, "QSEN Exemplar," for an example of teamwork and collaboration during the weaning process.

Assessment for Readiness to Wean (Wean Screen)

Before initiating the weaning trial, the patient is screened for readiness using parameters that have been associated with ventilator discontinuation success (Box 9-10). Weaning assessment tools are useful in assessing a patient's strengths and factors that may interfere with successful weaning. Patients are usually able to wean when the underlying disease process is resolving, they are hemodynamically stable, and they are able to initiate an inspiratory effort.[40] Therefore assessment of the neurological, cardiovascular, and respiratory systems provides a sufficient screen in most patients requiring a ventilator for only a short period.

Patients who require long-term mechanical ventilation (>72 hours) may have more physiological factors that affect weaning, such as inadequate nutrition and respiratory muscle deconditioning. A tool that provides a more comprehensive or multidimensional assessment of weaning readiness, as well as a baseline score from which to measure patient progress once

BOX 9-9 EVIDENCE-BASED GUIDELINES FOR WEANING FROM MECHANICAL VENTILATION

1. Identify causes for ventilator dependence if the patient requires ventilation for longer than 24 hours.
2. Conduct a formal assessment to determine a high potential for successful weaning:
   - Evidence of reversal of underlying cause of respiratory failure
   - Adequate oxygenation (PaO_2/FiO_2 >150-200; positive end-expiratory pressure <5-8 cm H_2O; FiO_2 <0.4-0.5) and pH (>7.25)
   - Hemodynamic stability
   - Able to initiate an inspiratory effort
3. Conduct a spontaneous breathing trial (SBT). During the SBT, evaluate respiratory pattern, adequacy of gas exchange, hemodynamic stability, and comfort. A patient who tolerates a SBT for 30 to 120 minutes should be considered for permanent ventilator discontinuation.
4. If a patient fails an SBT, determine the cause for the failed trial. Provide a method of ventilatory support that is nonfatiguing and comfortable. Correct reversible causes, and attempt a SBT every 24 hours if the patient meets weaning criteria.
5. Assess airway patency and the ability of the patient to protect the airway to determine whether to remove the artificial airway from a patient who has been successfully weaned.
6. In postsurgical patients, provide anesthesia/sedation strategies and ventilator management aimed at early extubation.
7. Develop weaning protocols that the nurse and respiratory therapist can implement.
8. Consider a tracheostomy when it becomes apparent that the patient will require prolonged ventilator assistance. Patients with the following conditions may benefit most from early tracheostomy:
   - High levels of sedation
   - Marginal respiratory mechanics (e.g., tachypnea) associated with work of breathing
   - Psychological benefit from ability to eat and speak
   - Enhanced mobility to promote physical therapy efforts and psychological support
9. Conduct slow-paced weaning in a patient who requires prolonged mechanical ventilation. Wean a patient to 50% of maximum ventilator support before daily SBT. Then initiate SBTs with gradual increase in duration of the SBT.
10. Unless evidence of irreversible disease exists (e.g., high cervical spine injury), do not consider a patient to be ventilator dependent until 3 months of weaning attempts have failed.
11. Transfer a patient who has failed weaning attempts but is medically stable to a facility that specialize in management of ventilator-dependent patients.

FiO_2, Fraction of inspired oxygen; *PaO_2,* partial pressure of oxygen in arterial blood; *SBT,* spontaneous breathing trial.
Based on data from MacIntyre N. (2009). Discontinuing mechanical ventilatory support. In NR Macintyre and RD Branson (Eds.) *Mechanical Ventilation* 2nd ed., St. Louis: Saunders Elsevier, pp. 317-324; MacIntyre N, Cook D, & Ely E. (2001). Evidence-based guidelines for weaning and discontinuing mechanical ventilatory support: A collective task force facilitated by the American College of Chest Physicians; the American Association for Respiratory Care; and the American College of Critical Care Medicine. *Chest, 120,* 375S-395S; MacIntyre NR. (2004). Evidence-based ventilator weaning and discontinuation. *Respiratory Care, 49,* 830-836.

QSEN EXEMPLAR

Teamwork and Collaboration

Successful weaning of patients from mechanical ventilation requires a team approach. Led by a clinical nurse specialist (CNS), a hospital established a multiprofessional ventilator team composed of a pulmonologist, critical care nurses, step-down unit nurses, respiratory therapist, speech therapist, physical therapist, clinical pharmacist, case manager, social worker, chaplain, and home care personnel. Every patient in the facility who was mechanically ventilated for more than 3 days received a comprehensive evaluation by the CNS. The CNS met with the patient, family, physician, and unit staff to identify potential issues that could impact the weaning process. Patients meeting criteria were then presented at the weekly "Vent Team" meeting. Individualized weaning plans were developed for each patient. Additional concerns related to mechanical ventilation including nutrition, communication, mobility and function, pain and anxiety, infection risk, patient and family coping, end-of-life concerns, spirituality, and discharge preparation were addressed, and plans of care were modified as required. Patient outcomes included improved transitions between nursing units, reduction in ventilator days, reduction in ventilator-acquired pneumonia, and reduced length of stay. Team members proactively worked with patients and families to address end-of-life issues and to plan terminal weaning as appropriate. A team approach was used to transition patients who required ongoing mechanical ventilation for conditions such as amytrophic lateral sclerosis and spinal cord injury to the care of the family provider in the home setting.

BOX 9-10 ASSESSMENT PARAMETERS INDICATING READINESS TO WEAN

Underlying Cause for Mechanical Ventilation Resolved
- Improved chest x-ray findings
- Minimal secretions
- Normal breath sounds

Hemodynamic Stability; Adequate Cardiac Output
- Absence of hypotension
- Minimal vasopressor therapy

Adequate Respiratory Muscle Strength
- Respiratory rate <25-30 breaths/min
- Negative inspiratory pressure or force that exceeds −20 cm H_2O
- Spontaneous tidal volume 5 mL/kg
- Vital capacity 10-15 mL/kg
- Minute ventilation 5-10 L/min
- Rapid shallow breathing index <105

Adequate Oxygenation Without a High FiO_2 and/or a High PEEP
- PaO_2 >60 mm Hg with FiO_2 0.4-0.5
- PaO_2/FiO_2 >150-200
- PEEP <5-8 cm H_2O

Absence of Factors that Impair Weaning
- Infection
- Anemia
- Fever
- Sleep deprivation
- Pain
- Abdominal distention; bowel abnormalities (diarrhea or constipation)
- Mental readiness to wean: calm, minimal anxiety, motivated
- Minimal need for sedatives and other medications that may cause respiratory depression

FiO_2, Fraction of inspired oxygen; *PaO_2,* partial pressure of oxygen in arterial blood; *PEEP,* positive end-expiratory pressure.

weaning is begun, is the Burns Wean Assessment Program (BWAP). The BWAP has been scientifically tested in critically ill patients.[9] The BWAP evaluates nonpulmonary factors that impact weaning success, such as hematocrit; fluids, electrolytes, and nutrition; anxiety, pain, and rest; bowel function; and physical conditioning and mobility. Pulmonary factors assessed with the BWAP include RR and pattern; secretions; neuromuscular disease and deformities; airway size and clearance; and ABGs.

The nurse must collaborate with RT and physician, using data and weaning assessment tools to identify readiness for weaning and factors that may impede successful weaning.[37,52] When a patient continues to not be ready to wean or is not successful at a weaning trial, these factors should be assessed and optimized to promote patient success in future weaning endeavors.

Weaning Process (Weaning Trial)

Table 9-5 describes weaning methods. Patients are assessed and monitored throughout the weaning process; therefore the nurse must organize work to remain vigilant throughout the trial. Four methods of reducing ventilatory support are used: SIMV, PS, T-piece, or CPAP. Studies do not demonstrate one method to be superior to the others; however, they do show that weaning takes longer with SIMV.[5,20,40] Current evidenced-based practice guidelines recommend the use of a spontaneous breathing trial (SBT) for weaning. PS, T-piece, and CPAP qualify as spontaneous breathing modes, whereas SIMV, because of the provision of mandatory breaths, does not. The SBT for up to 2 hours (90 to 120 minutes) provides a direct assessment of spontaneous breathing capabilities and has been shown to be the most effective way to shorten the ventilator discontinuation process.[40]

The weaning procedure is explained to the patient and family in a manner that promotes reassurance and minimizes anxiety. The patient should be adequately rested and positioned optimally for diaphragm function and lung expansion, such as sitting. Baseline parameters are obtained: vital signs, heart rhythm, ABGs or pulse oximetry/$ETCO_2$ values, and neurological status. The patient is monitored during the weaning process for tolerance or intolerance to the procedure. Although the patient is required to increase participation in the WOB, caregivers must ensure that the patient does not become fatigued by the weaning effort and become compromised.[8,35] Box 9-11 provides a list of physiological parameters that are monitored to identify that the patient is not tolerating the weaning process. If these signs of intolerance develop, the weaning trial is stopped and mechanical ventilation is resumed at ventilator settings that provide full ventilatory support.[52]

Many respiratory and nonrespiratory factors can impact weaning success. Increased oxygen demands occur with infection, fever, anemia, pain, or asking the patient to perform another activity such as physical therapy during the trial, and can impair weaning. Other factors to assess for are decreased respiratory performance from malnutrition, overuse of sedatives or hypnotics, sleep deprivation, and abdominal distention. Factors involving equipment or technique, such as time of day or method for weaning, should also be examined. Psychological factors to evaluate include apprehension and fear, helplessness, and depression.[28,67] Each factor should be systematically assessed and optimized to promote weaning success.

TABLE 9-5 WEANING METHODS

	WEANING TECHNIQUE	
MODE	**DESCRIPTION**	**STRATEGIES**
Spontaneous Breathing Trial Gradual reconditioning through trials of spontaneous breathing effort	Every breath is spontaneous and patient performs all the work of breathing Attempt daily if patient passes wean screen Successful when patient remains stable for 90-120 minutes	Alternating periods of resting on full ventilator support with advancing periods of gradually reduced support Ratio of rest periods to time on trial based on patient's response Amount of time to liberate patient varies; may be days to weeks Indicated for significantly deconditioned patients
Pressure Support Provides inspiratory support to overcome resistance to gas flow through ventilator circuit and artificial airway	PS of 5 cm H_2O + 5 cm H_2O PEEP	Begin at level of PS that ensures normal RR and V_T Gradual reduction in PS in 2-5 cm H_2O increments Gradually lengthen intervals at reduced levels of support Discontinue when patient stable for 2 hours or longer at 5 cm H_2O PS
T-Piece Patient performs all the WOB No ventilator alarms for apnea, decreased VT, etc.	Remove patient from ventilator and provide humidified oxygen via a T-piece adaptor attached to the ETT or tracheostomy tube	May start with trial as short as 5 minutes Increase time on T-piece as tolerated with adequate rest periods (6-8 hours) on full ventilatory support Discontinue when patient stable on T-piece for at least 2 hours, often longer
CPAP Useful when patient requires PEEP to maintain oxygenation Patient performs all the WOB Ventilator will provide alarms for apnea, high RR, or low EVT	CPAP of 5 cm H_2O	CPAP of 5 cm H_2O May start with trial as short as 5 minutes Increase time on CPAP as tolerated with adequate rest periods (6-8 hours) on full ventilatory support Discontinue when patient stable on CPAP for at least 2 hours, often longer
SIMV	Not applicable, not a spontaneous breathing mode	Decrease the number of mandatory (machine) breaths in increments of 2 as tolerated Discontinue when patient stable on SIMV of 2-4 for 2 hours, often longer

cm H_2O, Centimeters of water; *CPAP,* continuous positive airway pressure; *ETT,* endotracheal tube; *EV_T,* exhaled tidal volume; *PEEP,* positive end-expiratory pressure; *PS,* pressure support; *RR,* respiratory rate; *SIMV,* synchronous intermittent mandatory ventilation; *V_T,* tidal volume; *WOB,* work of breathing.

BOX 9-11 CRITERIA FOR DISCONTINUING WEANING

Respiratory
- Respiratory rate >35 breaths/min or <8 breaths/min
- Spontaneous V_T <5 mL/kg ideal body weight
- Labored respirations
- Use of accessory muscles
- Abnormal breathing pattern: chest/abdominal asynchrony
- Oxygen saturation <90%

Cardiovascular
- Heart rate changes more than 20% from baseline
- Dysrhythmias (e.g., premature ventricular contractions or bradycardia)
- Ischemia: ST-segment elevation
- Blood pressure changes more than 20% from baseline
- Diaphoresis

Neurological
- Agitation, anxiety
- Decreased level of consciousness
- Subjective discomfort

V_T, Tidal volume.

Extubation

If the patient demonstrates tolerance to the weaning procedure and can sustain spontaneous breathing for 90 to 120 minutes, then the second step to ventilator discontinuation, the decision to extubate (remove the ETT), may be made. Consideration must be given to the need for the airway for secretion clearance; therefore the patient must have a good cough and require suctioning no more than every 2 hours. If the patient has a tracheostomy, the patient may be liberated from the ventilator but the tracheostomy is maintained to facilitated airway clearance. If the decision is made to extubate, the ETT should be suctioned thoroughly before removal. Secretions that may have pooled above the cuff should be aspirated, the balloon of the ETT is deflated, and the ETT is removed during inspiration.[11,50,66] Once extubated, the patient is assessed for stridor, hoarseness, changes in vital signs, or low SpO_2, which may indicate complications.[63] Noninvasive ventilation may be used to avert reintubation in some patients.[41]

CASE STUDY

Mr. P., age 65 years, was transferred to the critical care unit from the emergency department after successful resuscitation from a cardiac arrest sustained out of the hospital. Initial diagnosis based on laboratory results and electrocardiography is acute anterior myocardial infarction. It is suspected that Mr. P. aspirated gastric contents during the cardiac arrest. He opens his eyes to painful stimuli. He is orally intubated and receiving mechanical ventilation. He is on assist-control ventilation, respiratory rate set at 12 breaths/min, FiO_2 of 0.40, PEEP 5 cm H_2O, V_T 700 mL. An arterial blood gas is drawn upon arrival to the critical care unit and shows the following values: pH, 7.33; $PaCO_2$, 40 mm Hg; HCO_3^-, 20 mEq/L; PaO_2, 88 mm Hg; and SaO_2, 99%. A decision is made to maintain the initial ventilator settings. The following day, Mr. P.'s chest radiograph shows progressive infiltrates. His oxygen saturation is dropping below 90% and he is demonstrating signs of hypoxemia: increased heart rate and premature ventricular contractions. Arterial blood gas analysis at this time shows pH, 7.35; $PaCO_2$, 43 mm Hg; HCO_3^-, 26 mEq/L; PaO_2, 58 mm Hg; and SaO_2, 88%. The physician orders the FiO_2 increased to 0.50, and PEEP increased to 10 cm H_2O.

Questions

1. What were the results of Mr. P.'s first arterial blood gas analysis? What factors are contributing to these results?
2. What factor is contributing to Mr. P.'s worsening condition the day after hospital admission?
3. Interpret the arterial blood gases done the day after the cardiac arrest.
4. Why did the physician change the ventilator settings after the second set of arterial blood gases?
5. What must the nurse assess after the addition of the PEEP? Why is this especially important for Mr. P.?

SUMMARY

Skills in establishing and maintaining a patent airway, providing oxygen therapy, initiating mechanical ventilation, and ongoing patient assessment are essential for critical care nurses. Care of the patient requiring mechanical ventilation is common practice in the critical care unit; therefore it is essential that the nurse apply knowledge and skills to effectively care for these vulnerable patients.

CRITICAL THINKING EXERCISES

1. Based on your knowledge of clinical disorders, identify different clinical conditions that could cause problems with the following steps in gas exchange:
   a. Ventilation
   b. Diffusion
   c. Perfusion (transportation)
2. Your patient has the following arterial blood gas results: pH, 7.28; PaO_2, 52 mm Hg; SaO_2, 84%; $PaCO_2$, 55 mm Hg; HCO_3^-, 24 mEq/L.
   a. What is your interpretation of this arterial blood gas?
   b. What clinical condition or conditions could cause the patient to have these arterial blood gas results?
3. Your patient requires mechanical ventilation for treatment. The pressure alarm keeps going off for a few seconds at a time, even though you have just suctioned the patient. What nursing assessments and potential actions are warranted at this time?
4. You are caring for a patient who has been mechanically ventilated for 2 weeks. Physically, the patient meets all the criteria to begin weaning from mechanical ventilation. What parameters should the nurse monitor to assess tolerance of weaning?
5. Your patient is being ventilated with noninvasive positive-pressure ventilation with a nasal mask. The patient is mouth breathing and the ventilator is alarming low exhaled tidal volume. What interventions should the nurse take to ensure the patient receives adequate ventilation?

REFERENCES

1. Ackerman MH, Mick DJ. Instillation of normal saline before suctioning in patients with pulmonary infections: a prospective randomized controlled trial. *American Journal of Critical Care*. 1998;7:261-266.
2. American Thoracic Society. Guidelines for the management of adults with hospital-acquired, ventilator-associated, and healthcare-associated pneumonia. *American Journal of Respiratory and Critical Care Medicine*. 2005;171:388-416.
3. Baumgartner CA, Bewyer E, Bruner D. Management of communication and swallowing in intensive care. *AACN Advance Critical Care*. 2008;19:433-443.
4. Bouza C, Garcia E, Diaz M, et al. Unplanned extubation in orally intubated medical patients in the intensive care unit: a prospective cohort study. *Heart and Lung*. 2007;36:270-276.
5. Brochard L, Rauss A, Benito S, et al. Comparison of three methods of gradual withdrawal from ventilatory support during weaning from mechanical ventilation. *American Journal of Respiratory and Critical Care Medicine.* 1994;150:896-903.
6. Brook AD, Ahrens TS, Schaiff R, et al. Effect of a nursing-implemented sedation protocol on the duration of mechanical ventilation. *Critical Care Medicine*. 1999;27:2609-2615.
7. Brower R, Matthay M, Morris A, et al. Ventilation with lower tidal volumes as compared with traditional tidal volumes for acute lung injury and the acute respiratory distress syndrome: the acute respiratory distress syndrome network. *New England Journal of Medicine*. 2000;342:1301-1308.
8. Burns SM. AACN protocols for practice: weaning from mechanical ventilation. In: Burns SM, ed. *AACN Protocols for Practice: Care of the Mechanically Ventilated Patient*. 2nd ed., pp. 97-160. Boston: Jones & Bartlett; 2007.
9. Burns SM, Fisher C, Tribble SS, et al. Multifactor clinical score and outcome of mechanical ventilation weaning trials: Burns wean assessment program. *American Journal of Critical Care*. 2010;(19)5:431-440.
10. Celik SA, Kanan NA. A current conflict: use of isotonic sodium chloride solution on endotracheal suctioning in critically ill patients. *Dimensions of Critical Care Nursing*. 2006; 25(1):11-14.
11. Centers for Disease Control and Prevention. Guidelines for preventing health-care associated pneumonia. *MMWR. Morbidity and Mortality Weekly Report*. 2004;53:(RR-3).
12. Chulay M, Seckel MA. Suctioning: endotracheal or tracheostomy tube. In: Lynn-McHale Weigand DJ, ed. *AACN Procedure Manual for Critical Care*. 6th ed., 80-104. St. Louis: Elsevier Saunders; 2011.
13. Colonel P, Houze MH, Vert H, et al. Swallowing disorders as a predictor of unsuccessful extubation: a clinical evaluation. *American Journal of Critical Care*. 2008;17:504-510.
14. DePew CL. Subglottic secretion drainage: a literature review. *AACN Advanced Critical Care*. 2007;18:366-379.
15. Dezfulian C, Shojania K, Collard HR, et al. Subglottic secretion drainage for preventing ventilator-associated pneumonia: a meta-analysis. *American Journal of Medicine*. 2005;118:11-18.
16. Drakulovic MB, Torres A, Bauer TT, et al. Supine body position as a risk factor for nosocomial pneumonia in mechanically ventilated patients: a randomised trial. *Lancet*. 1999;354:1851-1858.
17. Durbin CG Jr. Indications for and timing of tracheostomy. *Respiratory Care.* 2005;50:483-487.
18. Durbin CG, Jr. Techniques for performing tracheostomy. *Respiratory Care.* 2005;50:488-496.
19. El Masry A, Williams PF, Chipman DW, et al. The impact of closed endotracheal suctioning systems on mechanical ventilator performance. *Respiratory Care*. 2005;50:345-353.
20. Esteban A, Frutos F, Tobin MJ, et al. A comparison of four methods of weaning patients from mechanical ventilation. Spanish Lung Failure Collaborative Group. *New England Journal of Medicine*. 1995;332:345-353.
21. Fink J. Humidity and aerosol therapy. In: Cairo JM, Pilbeam SP, eds. *Mosby's Respiratory Care Equipment*. 8th ed. St. Louis: Mosby; 2010.
22. Good VS, Luehrs P. Continuous end-tidal carbon dioxide monitoring. In: Lynn DJ, Lynn-McHale Weigand DJ, ed. *AACN Procedure Manual for Critical Care*. 6th ed., pp. 106-127. St. Louis: Elsevier Saunders; 2011.
23. Grossbach I, Chlan L, Tracy MF. Overview of mechanical ventilatory support and management of patient- and ventilator-related responses. *Critical Care Nurse*. 2011;31:30-45.
24. Grossbach I, Stranberg S, Chlan L. Promoting effective communication for patients receiving mechanical ventilation. *Critical Care Nurse*. 2011;31(3):46-61
25. Gulanick M, Myers JL. *Nursing Care Plans: Nursing Diagnosis and Intervention*. 7th ed. St. Louis: Mosby; 2011.
26. Habashi NM. Other approaches to open-lung ventilation: airway pressure release ventilation. *Critical Care Medicine*. 2005;33:S228-S240.
27. Hall JE, Guyton AC. *Textbook of Medical Physiology*. 12th ed. Philadelphia: Saunders Elsevier; 2011.
28. Happ MB, Swigart VA, Tate JA, et al. Family presence and surveillance during weaning from prolonged mechanical ventilation. *Heart and Lung*. 2007;36:47-57.
29. Happ MB, Tuite P, Dobbin K, et al. Communicating ability, method, and content among nonspeaking nonsurviving patients treated with mechanical ventilation in the intensive care unit. *American Journal of Critical Care*. 2004;13(3):210-220.
30. Heuer AJ, Scanlan CL. Medical gas therapy. In: Wilkins RL, Stoller JK, Kacmarek RM, eds. *Egan's Fundamentals of Respiratory Care*. 8th ed., pp. 868-920. St. Louis: Mosby Elsevier; 2009.
31. Hickling KG. Lung-protective ventilation in acute respiratory distress syndrome: protection by reduced lung stress or by therapeutic hypercapnia? *American Journal of Respiratory and Critical Care Medicine*. 2000;162:2021-2022.
32. Institute for Healthcare Improvement. Getting started kit: prevent ventilator associated pneumonia. *How to Guide.* Available at www.ihi.org; 2007
33. Jacobi J, Fraser GL, Coursin DB, et al. Clinical practice guidelines for the sustained use of sedatives and analgesics in the critically ill adult. *Critical Care Medicine*. 2002;30:119-141.
34. Jongerden IP, Rovers MM, Grypdonck MH, et al. Open and closed endotracheal suction systems in mechanically ventilated intensive care patients: a meta-analysis. *Critical Care Medicine*. 2007;35:260-270.
35. Kacmarek RM, Dimas S, Mack CW. *The Essentials of Respiratory Care*. St. Louis: Mosby; 2005.
36. Kollef MH, Levy NT, Ahrens TS, et al. The use of continuous I.V. sedation is associated with prolongation of mechanical ventilation. *Chest*. 1998;114:541-548.
37. Kollef MH, Shapiro SD, Silver P, et al. A randomized, controlled trial of protocol-directed versus physician-directed weaning from mechanical ventilation. *Critical Care Medicine*. 1997;25:567-574.

38. Luer J. Sedation and neuromuscular blockade in the mechanically ventilated patient. In: Burns SM, ed. *AACN Protocols for Practice: Care of the Mechanically Ventilated Patient.* 2nd ed., pp. 253-284. Boston: Jones & Bartlett; 2007.
39. MacIntyre N. Discontinuing mechanical ventilatory support. In: MacIntyre NR, Branson RD, eds. *Mechanical Ventilation*. 2nd ed., St. Louis: Saunders Elsevier; 2009.
40. MacIntyre N, Cook D, Ely E. Evidence-based guidelines for weaning and discontinuing mechanical ventilatory support: a collective task force facilitated by the American College of Chest Physicians; the American Association for Respiratory Care; and the American College of Critical Care Medicine. *Chest*. 2001;120:375S-395S.
41. MacIntyre NR. Evidence-based ventilator weaning and discontinuation. *Respiratory Care*. 2004;49:830-836.
42. Malone AM. The use of specialized enteral formulas in pulmonary disease. *Nutrition in Clinical Practice*. 2004;19:557.
43. Miller CA. Respiratory function. In: Miller CA, ed. *Nursing for Wellness in Older Adults*. 6th ed. Philadelphia: Lippincott, Williams & Wilkins; 2012.
44. Muscedere IP. Comprehensive evidence-based clinical practice guidelines for ventilator associated pneumonia prevention. *Journal Critical Care*. 2007;23:126-137.
45. Niel-Weise BS, Snoeren RL, van den Broek PJ. Policies for endotracheal suctioning of patients receiving mechanical ventilation: a systematic review of randomized controlled trials. *Infection Control and Hospital Epidemiology*. 2007;28:531.
46. O'Neal PV, Grap MJ, Thompson C, et al. Level of dyspnoea experienced in mechanically ventilated adults with and without saline instillation prior to endotracheal suctioning. *Intensive and Critical Care Nursing*. 2001;17:356-363.
47. Parrish CR, Krenitsky J, Willcutts K. Nutritional support for mechanically ventilated patients. In: Burns SM, ed. *AACN Protocols for Practice: Care of the Mechanically Ventilated Patient*. 2nd ed., pp. 191-244. Boston: Jones & Bartlett; 2007.
48. Patak L, Gawlinski A, Fung NI, et al. Communication boards in critical care: patients' views. *Applied Nursing Research*. 2006;19:182-190.
49. Patton KT, Thibodeau GA. Anatomy of the respiratory system. In: Thibodeau GA, Patton KT, eds. *Anatomy and Physiology,* 7th ed., pp. 777-817. St. Louis: Mosby Elsevier; 2010.
50. Pierce LNB. Invasive and noninvasive modes and methods of mechanical ventilation. In: Burns SM, ed. AACN *Protocols for Practice: Care of the Mechanically Ventilated Patient*. 2nd ed., pp. 59-89. Boston: Jones & Bartlett; 2007.
51. Pierce LNB. *Management of the Mechanically Ventilated Patient*. 2nd ed. Philadelphia: Saunders; 2007.
52. Pilbeam SP. Discontinuation of and weaning from mechanical ventilation. In: Pilbeam SP, Cairo JM, eds. *Mechanical Ventilation: Physiological and Clinical Applications*. 4th ed., pp. 443-471. St. Louis: Mosby; 2006.
53. Pilbeam SP, Cairo JM. *Mechanical Ventilation: Physiological and Clinical Applications*. 4th ed. St. Louis: Mosby; 2006.
54. Richmond AL, Jarog DL, Hanson VM. Unplanned extubation in adult critical care. Quality improvement and education payoff. *Critical Care Nurse*. 2004;24:32-37.
55. Ridling DA, Martin LD, Bratton SL. Endotracheal suctioning with or without instillation of isotonic sodium chloride solution in critically ill children. *American Journal of Critical Care*. 2003;12:212-219.
56. Safdar N, Dezfulian C, Collard HR, et al. Clinical and economic consequences of ventilator-associated pneumonia: A systematic review. *Critical Care Medicine*. 2005;33:2184-2193.
57. Schutz SL. Oxygen saturation monitoring with pulse oximetry. In: Lynn-McHale Weigand DJ, ed. *AACN Procedure Manual for Critical Care*. 6th ed., 121-137. St. Louis: Elsevier Saunders; 2011.
58. Shilling A, Durbin CG. Airway management. In: Cairo J, ed. *Mosby's Respiratory Care Equipment*. 8th ed. St. Louis: Mosby Elsevier; 2010.
59. Siela D. Pulmonary aspects of obesity in critical care. *Critical Care Nursing Clinics of North America*. 2009;21(3):301-310.
60. Simmons KF, Scanlan CL. Airway management. In: Wilkins RL, Stoller JK, Scanlan CL, Kacmarek RM, eds. *Egan's Fundamentals of Respiratory Care*. 9th ed., pp. 694-765. St. Louis: Mosby Elsevier; 2009.
61. Skillings K, Curtis B. Tracheal tube cuff care. In: Lynn-McHale Weigand DJ, ed. *AACN Procedure Manual for Critical Care*. Philadelphia: Saunders; 2010.
62. Skoretz SA, Flowers HL, Martino R. The incidence of dysphagia following endotracheal intubation. *Chest*. 2010;137: 665-673.
63. Sole ML, Byers JF, Ludy JE, et al. A multisite survey of suctioning techniques and airway management practices. *American Journal of Critical Care*. 2003;12:220-230.
64. Sole ML, Xiaogang S, Talber S, et al. Evaluation of an intervention to maintain endotracheal tube cuff pressure within therapeutic range. *American Journal of Critical Care*. 2011;20:109-118.
65. St. John RE. End-tidal CO_2 monitoring. In: Burns SM, ed. *AACN Protocols for Practice: Noninvasive Monitoring*. 2nd ed. Boston: Jones & Bartlett; 2006.
66. St. John RE, Seckel MA. Airway management. In: Burns SM, ed. *AACN Protocols for Practice: Care of the Mechanically Ventilated Patient*. 2nd ed. Boston: Jones & Bartlett; 2007.
67. Tracy MF, Chlan L. Nonpharmacologic interventions to manage common symptoms in patients receiving mechanical ventilation. *Critical Care Nurse*. 2011;31:19-29.
68. Weber J, Kelly JH. Thorax and lungs. *Health Assessment in Nursing*. 4th ed., Philadelphia: Lippincott, Williams & Wilkins; 2010.
69. West JB. *Respiratory Physiology: the Essentials*. 9th ed. Baltimore, MD: Lippincott Williams & Wilkins; 2011.
70. Wilkins RL. Gas exchange and transport. In: Wilkins RL, Stoller JK, Kacmarek RM, eds. *Egan's Fundamentals of Respiratory Care.* 9th ed., pp. 238-268. St. Louis: Mosby Elsevier; 2009.