Chapter 25

ACUTE RESPIRATORY FAILURE AND VENTILATORY MANAGEMENT

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INTRODUCTION

Respiratory insufficiency in combat casualties is seen in many different guises depending on the mechanism and the time of appearance (Exhibit 25-1):

- In some casualties, respiratory insufficiency is an immediate and direct consequence of physical trauma to the lungs or the chest wall. An example might be a soldier who is shot through the chest and sustains a massive tension pneumothorax that will cause death within a few minutes unless immediate care is rendered by a medic or a medical officer.
- In other casualties, respiratory insufficiency might develop days after direct trauma to the chest. An example might be a soldier who sustained a gunshot wound of the lung and subsequently develops pneumonia and empyema.
- Unfortunately, some casualties may develop respiratory insufficiency that is, in part, secondary to the therapies given for an injury in some other part of the body. An example might be a soldier who develops pulmonary edema following excessive fluid overload that occurred during the treatment of massive blood loss resulting from a traumatic amputation of a leg.

The most common type of respiratory insufficiency that military anesthesiologists and intensivists (ie, critical care specialists) are likely to see, however, occurs within several days to 1 week following an injury that did not directly involve the lungs or chest wall: the adult respiratory distress syndrome (ARDS). This condition is now recognized as an expression of the systemic inflammatory response syndrome (SIRS) and is related to sepsis.

This chapter is intended to provide practical help to the medical officer who is confronted with casualties whose conditions are of varying degrees of severity, for whom decisions must be made promptly to avoid further morbidity from cerebral hypoxia or

EXHIBIT 25-1

ETIOLOGY OF RESPIRATORY FAILURE IN COMBAT AND CIVILIAN PATIENTS

Pulmonary aspiration

Foreign-body obstruction of the airway

Soft-tissue obstruction of the airway:
- Prolapse of tongue due to unconsciousness
- Prolapse due to facial bone fracture
- Edema and hematoma formation

Pulmonary injury:
- Hemothorax
- Pneumothorax
- Hemopneumothorax
- Lung contusion

Mediastinal emphysema

Rib fractures

Flail chest

Laryngeal fracture (with or without separation)

Tracheobronchial laceration or separation

Acute lung injury secondary to:
- Blast overpressure injury
- Chemical war gas
- Infection
- Hypermetabolism
- Inhalation of combustion products

Hypoventilation secondary to:
- Cervical spinal cord trauma
- Cranial trauma
- Exposure to nerve agent
- Diaphragmatic hernia
- Thoracic wall injury
- Central nervous system depression from drugs or hypoxemia

Adapted with permission from Grande CM, Stene JK, Bernhard WN. Airway management: Considerations in the trauma patient. *Crit Care Clin.* 1990; 6(1):43.

other visceral organ injury. The chapter emphasizes (1) the recognition of respiratory insufficiency and (2) the provision of mechanical ventilation for casualties with respiratory insufficiency at medical treatment facilities from a third-echelon hospital to a medical center in the continental United States.

EPIDEMIOLOGY OF RESPIRATORY FAILURE IN COMBAT CASUALTY CARE

Combat casualties do not commonly present with respiratory insufficiency. Data from the Vietnam War indicate that probably somewhat less than 5% of those killed in action had injuries primarily to the respiratory system exclusive of those who exsanguinated from injuries to the pulmonary vasculature.[1] Respiratory failure at the hospital level may be more common. The total number who die of wounds is about 3% to 4% of hospitalized casualties; it is probably reasonable to assume that all of these casualties—at some time during their hospital stay—would require respiratory support. Data from the Vietnam War indicate that approximately 0.4% of hospitalized casualties died of what would now be called ARDS.[2] Today, many more casualties would probably receive prophylactic respiratory support to prevent respiratory insufficiency, so it is not unreasonable to infer from these data that perhaps 10% of all hospitalized combat casualties are at risk of developing respiratory insufficiency. Furthermore, if nerve agents or enhanced blast munitions were to be used against U.S. military forces in a future hostile action, a much higher percentage of the casualty population will likely need immediate respiratory support. For planning purposes, the Department of Defense's Deployable Medical Systems (DEPMEDS) treatment files predict that in a major, high-intensity war, 17% of hospitalized casualties will require ventilatory support.[3]

Although the civilian experience depends on the type of hospital surveyed, probably no more than several percent of hospitalized patients develop respiratory insufficiency. This assumes that about 5% of patients who are admitted to civilian hospitals are transferred to the intensive care unit (ICU), and that of these, one half require respiratory support for more than 24 hours.[4]

ETIOLOGY OF PULMONARY INSUFFICIENCY

Mechanical Trauma

The battlefield is replete with potential mechanisms for pulmonary injury and the development of respiratory failure. Militarily relevant sources of direct mechanical trauma include penetrating, blunt, and blast injuries to the lungs and the chest wall. In addition, many soldiers with severe penetrating injuries to the brain are not killed outright and require intubation for airway control and mechanical ventilation. The injury to the brain results in destruction of the respiratory centers; therefore, ventilatory drive is absent. During the Vietnam War, this was probably the most common indication for using respiratory support.

Penetrating Injury

Penetrating thoracic injuries may, in turn, cause secondary conditions that result in respiratory insufficiency. Pneumothorax reduces the ability to carry out normal gas exchange. The lack of a negative pressure gradient across the lung parenchyma leads to alveolar collapse and a resulting intrapulmonary shunting of blood. When a sufficient amount of tissue collapses, arterial hypoxemia ensues. *Tension* pneumothorax occurs when air enters the pleural space during inspiration but is unable to escape during exhalation. The progressive accumulation of air produces a shift of the mediastinal structures toward the unaffected side of the thorax. This event worsens gas exchange by compressing the unaffected lung and reduces venous return to the heart, resulting in shock. Hemothorax and hemopneumothorax are found in approximately 50% of casualties with penetrating wounds of the lung.[5] This fact is not surprising, given the close anatomical proximity of bronchioles and arterioles within the lung parenchyma. Small fragments that strike the heart may cause blood to collect within the pericardial space. The increased pressure within the pericardium restricts the ability of the ventricles to accept blood (tamponade). Cardiac output and blood pressure progressively decrease as stroke volume is reduced. If the tamponade is not reversed, cardiovascular collapse and death result.

Blunt Injury

Blunt chest trauma with rib fractures and potential *flail chest* (ie, multiple rib fractures that result in segmental chest wall instability) can occur in a

variety of battlefield settings. Underlying pulmonary contusion may be a far more important cause for respiratory failure than the mechanical dysfunction associated with multiple rib fractures.

Blast Injury

Blast injuries from any type of explosion can be categorized as primary, secondary, and tertiary. *Primary* blast injury is due to the direct effect of the pressure wave on the body, and is not common. *Secondary* blast injury occurs when projectiles and debris from the explosion strike the victim. Finally, *tertiary* blast injury, which is the major cause of pure blast injury, occurs when the individual is physically displaced by the bulk flow of gases away from the site of the explosion. The displacement leads to collision with environmental objects and subsequent trauma.[6] A more complete discussion of the medical consequences of blast injury can be found in *The Medical Consequences of Conventional Warfare: Ballistic, Blast, and Burn Injuries*.[7]

The pressure wave from an explosion moves away from the epicenter at supersonic speeds, whether through air or water. As primary blast injury is seen predominantly in the gas-containing organs, the ears, lungs, and gastrointestinal tract are the most readily affected. The ear is the most sensitive, with rupture of the tympanic membrane occurring at pressures above 35 kPa (kilopascals). While injury to the ear causes pain and long-term morbidity, pulmonary damage causes the greatest

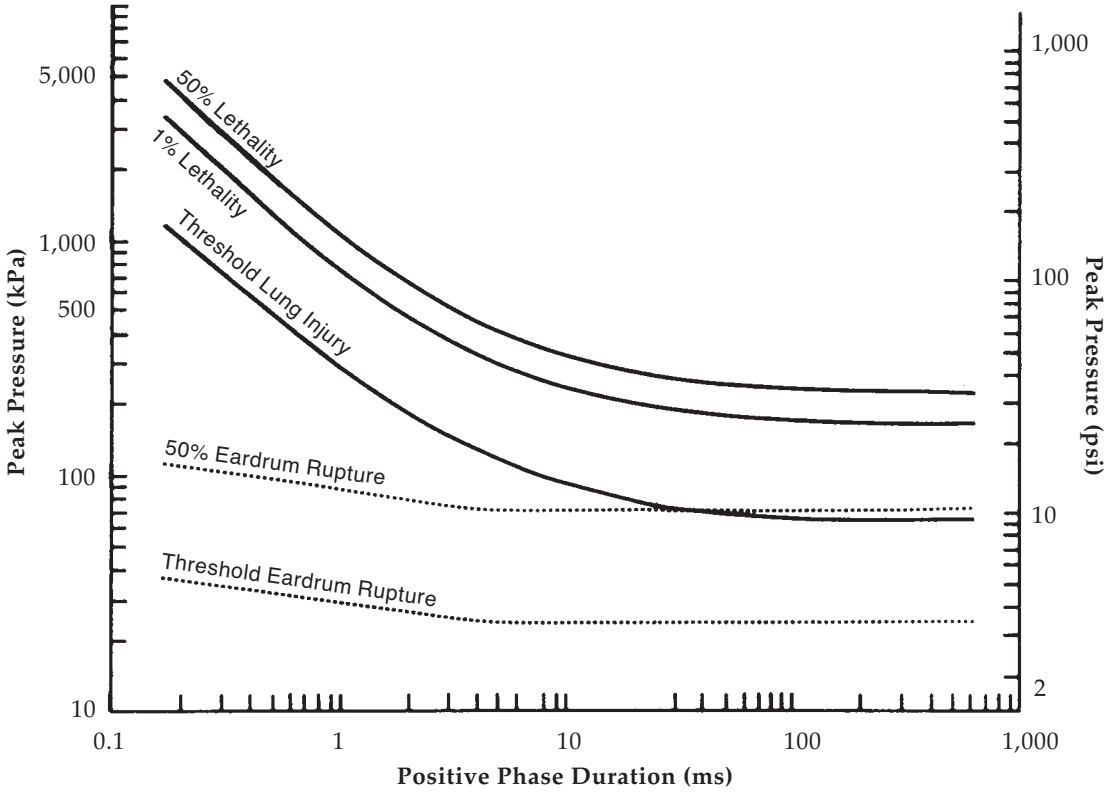

Fig. 25-1. The probabilities of eardrum rupture and death from a pulmonary blast injury. These are shown as functions of both the peak pressure above atmospheric pressure (measured in either kilopascals [kPa] or pounds per square inch [psi]) and the duration of the overpressure (in milliseconds [ms]). For example, a blast wave with a duration of 1 ms that has an overpressure of 5 psi can be expected to cause rupture of the tympanic membranes in a few people. If the overpressure reaches 15 psi, one half of the individuals in the exposed population can be expected to have ruptured tympanic membranes. Much greater pressures are required before pulmonary injury occurs. A blast wave with a duration of 1 msec and a peak overpressure of 50 psi can be expected to cause pulmonary injury in a few people. Doubling that pressure is likely to cause a fatal outcome in about one half of the exposed population. The same lethality is to be expected with a blast wave that lasts 1 s and has a peak overpressure of 12.5 psi. Note that both axes are logarithmic. Adapted from Bowen T, Bellamy RF, eds. *Emergency War Surgery NATO Handbook*. 2nd US rev. Washington, DC: US Department of Defense, Government Printing Office; 1988: 76.

immediate morbidity and mortality (Figure 25-1). Diffuse pulmonary contusions with a marked increase in lung water lead to ventilation–perfusion mismatch and arterial hypoxemia. Disruption of alveoli with the development of alveolar–pulmonary venous communications create the potential for air emboli, which is the most common cause of sudden death in such casualties. The gastrointestinal tract may be disrupted in portions where gas has collected. The colon tends to suffer the most gastrointestinal blast injuries, which are manifested by subserosal and intramural hemorrhage. Gastrointestinal blast injury may result in delayed rupture, days after the traumatic event.[8]

The clinical presentation of each of these injuries depends on the amount of energy transmitted to the various tissues in excess of the tissues' ability to dissipate the delivered forces. Significant pulmonary and gastrointestinal damage is unlikely to have occurred in the absence of tympanic membrane rupture. Arterial air emboli from alveolar–pulmonary venous communications typically present with signs and symptoms of cerebral dysfunction. However, direct trauma to the skull (secondary or tertiary blast injury) is more likely to be the cause of altered neurological function. Evidence of primary blast injury of the lung resembles the diffuse pulmonary contusion seen in blunt chest trauma. Chest pain with tachypnea, use of accessory respiratory muscles, and hemoptysis are common findings. The protean manifestations of *barotrauma* (injury resulting from elevated peak airway pressure) are frequently seen in these patients. Pulmonary contusion, pneumothorax, pneumomediastinum, subcutaneous emphysema, and pulmonary interstitial emphysema can be confirmed with a chest X-ray examination after the patient has been stabilized. Symptoms of blast injury to the lung may be delayed 24 to 48 hours, making differentiation from other forms of acute respiratory failure difficult.

The principles of management are detailed in other textbooks. Briefly summarized, the salient treatment required for improved survival rates of casualties with thoracic injuries is as follows:

- Remove the bloody secretions from the airway.
- Seal open chest wounds.
- Evacuate blood and air from the pleural space.
- Treat hypovolemia.
- Remove blood from the pericardial sac.
- Aggressively treat empyema.

Casualties with blast injuries require additional care. This follows the basic principles of trauma care, with the following caveats directed toward potential complications from the blast:

- Excessive volume resuscitation may complicate the pulmonary contusion present in most of these patients.
- Positive pressure ventilation may increase the severity of barotrauma and increase the possibility of air embolism.
- If oxygenation is inadequate with nasal cannula or mask, continuous positive airway pressure (CPAP) delivered via a tight-fitting mask or endotracheal tube may improve oxygenation without worsening barotrauma.

Thermal Trauma

Although thermal injuries will be encountered on the battlefield, this mode of injury is uncommon in conventional land warfare, occurring in fewer than 10% of all casualties (see Chapter 1, Combat Trauma Overview, Figure 1-4). Thermal injuries are, however, much more common in that subpopulation of soldiers who compose the crew of armored fighting vehicles, and are even more common in sailors and air personnel. In fact, fire and associated smoke inhalation are the most common causes of combat injury on warships.[9] Crewed vehicles—whether ships, tanks, or aircraft—are loaded with munitions and fuel and pose a constant hazard of explosions, fire, and thermal injury secondary to battle damage. Burns to the skin are what we usually think of when thermal injuries are discussed, and in fact, circumferential, full-thickness burns of the thoracic wall may sometimes restrict chest-wall motion to the degree that ventilation is compromised. Escharotomy may be required to improve the restrictive defect. However, the more frequent and more injurious form of thermal injury is damage to the respiratory system caused by inhaling steam and the gaseous and particulate products of combustion such as hydrogen chloride and phosgene from plastic, formaldehyde from wood, and hydrogen cyanide from polyurethane.

Below the level of the vocal cords, thermal injury from heated air is usually prevented by efficient upper-airway cooling. Stridor, hoarseness, and difficulty with phonation all suggest the need to evaluate the upper airway. Injury to the hypopharynx may lead to delayed airway obstruction, making prompt endotracheal intubation a lifesaving maneuver.

These injuries demonstrate a typical acute-lung-injury pattern of increased alveolar-capillary permeability with noncardiac pulmonary edema. Iatrogenic factors may complicate the lung injury: the massive fluid requirements for resuscitation from thermal burns and associated traumatic injuries will invariably worsen pulmonary function. Patients may develop respiratory failure 1 to 2 weeks following thermal injury. When delayed respiratory failure occurs, it is usually caused by infectious complications either within the lung (pneumonia) or at a remote site, giving rise to SIRS, which is the major cause of death.

Inhalation of the toxic products of combustion can induce parenchymal lung damage. The effects may be immediate or delayed for hours. A common, serious pathophysiological effect of inhaling combustion products is carbon monoxide intoxication. The marked affinity of carbon monoxide for hemoglobin (> 200-fold higher than that of oxygen) significantly reduces the oxygen-carrying capacity of blood. A carbon monoxide concentration of 0.1% in ambient air leads to a carboxyhemoglobin concentration of 50%. Carbon monoxide also shifts the oxyhemoglobin dissociation curve to the left, which further reduces oxygen delivery (Do_2) to the tissues. Patients will complain of headache, nausea, and exertional fatigue with moderate carbon monoxide exposure. Damage to the central nervous system (CNS) may occur in up to 30% of affected individuals unless they receive prompt therapy. The classic "cherry red" coloration is not seen with carboxy-hemoglobin concentrations lower than 40%. A far more common manifestation is cyanosis.[10] Lactic acidosis from reduced Do_2 is a common finding with severe intoxication.

The therapy for respiratory injury caused by thermal burn is primarily supportive. Supplemental oxygen and bronchodilators are used to treat hypoxemia and bronchoconstriction. Mechanical ventilation is required for patients with significant acute lung injury. The use of positive end-expiratory pressure (PEEP) increases the functional residual capacity (FRC, the remaining lung volume after passive exhalation) of the lung and allows the fraction of inspired oxygen (Fio_2) to be reduced to nontoxic levels. The U.S. Army Burn Center, Fort Sam Houston, Texas, reports that a mode of ventilation (high-frequency, percussive) that employs repeated cycles of very rapid (several hundred per minute) and small volume (less than the dead-space volume) ventilations may be more efficacious. The mortality rate found in such patients is markedly less (16.4%) than in patients with similar thermal insults who are treated with conventional mechanical ventilation (42.7%). The improved survival may be due to both (*a*) the low airway pressures found in high-frequency percussive ventilation (ie, there is less potential for barotrauma) and (*b*) improved removal of tracheobronchial secretions.[11]

Prophylactic steroids have not proven beneficial for inhalation injury and may increase the risk for subsequent infection. Likewise, antibiotic prophylaxis is no longer used; it may increase the risk for resistant bacterial infection, and has not been shown to reduce morbidity and mortality in clinical trials.[12]

The treatment of carbon monoxide poisoning involves supplying supplemental oxygen to reduce the half-life of carboxyhemoglobin. (With 100% oxygen, the half-life is reduced to 40 min, opposed to 250 min in room air.) Hyperbaric oxygen may be beneficial with severe intoxication; however, this modality is not likely to be available in the combat zone with the possible exception of aboard larger naval vessels.

Chemical Warfare

Almost all chemical war gases cause respiratory insufficiency and manifest a variety of effects that could lead to respiratory failure (see Chapter 30, Anesthesia for Casualties of Chemical Warfare Agents, for a discussion of the respiratory effects of war gases). The four major categories of war gases are nerve, blister, choking, and blood agents. Decontamination procedures are not addressed in this chapter; however, because acute respiratory failure is a common cause of death in affected individuals, medical personnel must ensure that the emergent nature of the situation does not lead to hasty action and inadvertent self-contamination.

Nerve Agents

Nerve agents have the potential to cause vast numbers of casualties. Soldiers must have sufficient personal protective equipment and adequate pharmacological prophylaxis. Military planners must emphasize that medical officers may need to care for mass casualties, all of whom may require mechanical ventilation.

Nerve agents exert their effects by preventing cholinesterase from hydrolyzing acetylcholine (previously released from the nerve terminal). This allows for repetitive stimulation of the postsynaptic receptor, which leads to rapid muscle fatigue. Inhalational or dermal exposures to nerve agents thus

present as a cholinergic syndrome, the severity of which depends on the amount of exposure. Respiratory effects of nerve agents include bronchoconstriction and bronchorrhea along with respiratory muscle dysfunction. Organophosphate-induced seizures may further complicate the respiratory embarrassment produced by these agents.

Treatment includes administering both atropine, which blocks the cholinergic receptors, and pralidoxime chloride (2-PAM Cl), which binds to organophosphate and most carbamate nerve agents, releasing the cholinesterase and allowing normal nerve function to resume. Airway control is of paramount importance, as copious secretions may prevent adequate ventilation and oxygenation. If respiratory muscle function is adequate, oral or nasopharyngeal suctioning may be all that is required. However, if respiratory muscle function is poor, endotracheal intubation and mechanical ventilation may be required. Care must be taken to ensure that the lungs are not exposed to the excessively high pressures that result from the intense bronchoconstriction that follows significant nerve agent exposure. Under these circumstances, barotrauma may occur before bronchospasm has subsided. Atropine, inhaled beta agonists such as metaproterenol or terbutaline, and inhalational anesthetics all reverse bronchospasm.[6]

Blister Agents

Blister agents (vesicants), of which the mustards are the best known, are alkylating chemicals that are thought to act by damaging cellular DNA. This leads to cell disruption and death; an intense inflammatory response causes blisters to form. Inhalation of vesicants produces severe tracheitis and bronchitis. Symptoms may not develop for 4 to 6 hours, requiring medical personnel to observe casualties for delayed effects. Bronchopneumonia frequently complicates the recovery of these patients. Progressive, irreversible respiratory insufficiency, developing over months to years after inhalation of mustard, has been described.[13] It appears to be caused by unrelenting fibrosis that leads to stenosis of the entire tracheobronchial tree.

Choking Agents

The war gas that caused the most flagrant example of respiratory insufficiency in World War I was phosgene, a choking agent. Choking agents cause a severe form of respiratory insufficiency and produce significant irritation of the lower respiratory tract. First used in World War I as a toxic agent, phosgene produces distal airway and alveolar inflammation, which eventually causes pulmonary edema. As with blister agents, the effects may be delayed for up to 12 hours; minimal symptomatology may precede the development of fulminant pulmonary edema.

Treatment is entirely supportive. Supplemental oxygen and positive-pressure ventilation may be necessary for severe cases. The effects are self-limiting, but due to the intense inflammatory response elicited, the patient's condition may evolve into ARDS.

Blood Agents

Blood agents such as hydrogen cyanide combine with intracellular cytochrome oxidase and halt cellular respiration. Exposure to large amounts of inhaled hydrogen cyanide causes death within minutes. Exposure to lesser amounts causes vertigo, nausea, vomiting, headache, and tachypnea. Seizures and cardiopulmonary arrest may rapidly ensue if treatment is delayed.

Treatment involves administering amyl nitrate by inhalation or sodium nitrite by vein. Nitrites oxidize the iron moiety of hemoglobin to produce methemoglobin. Methemoglobin scavenges cyanide from the circulating blood, which promotes a concentration gradient for the removal of cyanide from cytochrome oxidase. Intravenous sodium thiosulfate can be administered to provide free sulfur, which will convert cyanide to the far-less-toxic thiocyanate ion. Mechanical ventilation with supplemental oxygen will aid in the resuscitation of apneic casualties; however, expeditious treatment with nitrites is the mainstay of therapy.[6]

Nuclear Warfare

Thermonuclear weapons produce their devastating effects via the release of massive amounts of energy. This energy release produces casualties with three types of injuries: blast, thermal, and radiation. Blast and thermal injuries will be treated in a manner identical to that previously discussed. Neurovascular, gastrointestinal, and hematopoietic syndromes are associated with nuclear radiation injuries. Anesthesiologists and intensivists will encounter the neurovascular and gastrointestinal syndromes only rarely, as these are considered to be uniformly fatal. The hematopoietic syndrome will be the most common radiation injury requiring medical care.

INDIRECT MECHANISMS OF RESPIRATORY INSUFFICIENCY

Respiratory failure in the combat casualty may result from insults other than direct chest trauma. Three of the most frequently encountered etiologies are pulmonary aspiration, hydrostatic pulmonary edema, and permeability pulmonary edema.

Pulmonary Aspiration

Medical personnel should assume that every combat casualty has a full stomach. The combination of a traumatic injury and a full stomach puts soldiers at risk for aspirating blood or regurgitated stomach contents:

> The soldier may have eaten just before being wounded. Even if many hours have passed, pain and anxiety greatly delay gastric emptying. The important interval is not the time since the last meal, but rather the time from the last meal to the time of injury. Regurgitation and aspiration of stomach contents, particularly of volumes greater than 25 mL, with a pH less than 2.5, and in the presence of solid food, place the casualty at high risk for aspiration pneumonitis.[14(p28)]

Aspiration of gastric contents may be asymptomatic or cause several life-threatening syndromes. The underlying common denominator in aspiration is an altered level of consciousness. Many casualties will have head injuries that negate the protective airway reflexes, and many will require general anesthesia or have neurosurgical catastrophes that predispose to aspiration.[15] However, other predisposing conditions (ie, neuromuscular or gastrointestinal disorders) are not common in this population.

Silent aspiration occurs when a small volume of gastric contents is introduced into the trachea. This may occur somewhat frequently in a civilian medical population—elderly and obtunded—who are undergoing elective surgery, receiving therapeutic narcotics, whose level of consciousness is depressed, or while a nasogastric tube is being placed. In a combat situation, silent aspiration is probably less common.

When aspiration occurs, apnea or airway obstruction occur immediately. The acute chemical injury damages the tracheal bronchial tree and alveoli. Pathologic evaluation immediately after aspiration shows bronchiolar epithelial degeneration, pulmonary alveolar edema, and alveolar hemorrhage. Fibrinous exudate, focal atelectasis, and neutrophilic infiltrates occur later. After 24 hours, focal hemorrhage, atelectasis, empyema, fibrin-filled alveoli, and pneumonitis are established.[16] Bacterial pneumonitis can occur within 72 hours, and lung abscesses may occur, usually after days to weeks.

Injury occurs to both Type I and Type II pneumocytes. Type I pneumocytes undergo focal necrosis and the basement membrane is exposed. Fluid fills the alveolar and perivascular spaces. Type II cells are initially edematous, then degenerate. Hyaline membranes form by 48 hours. By 72 hours, alveolar cells regenerate and the inflammatory process abates.

Prevention is the best treatment for aspiration. When prevention fails or is not possible, then making the correct diagnosis, removing large particulate matter, and providing appropriate supportive measures will help improve morbidity and prevent mortality.[17]

Immediate endotracheal intubation is required when apnea occurs. Adequate ventilation should be documented by measuring arterial blood gases or by means of oximetry and capnography, which are discussed later in this chapter.

Airway protection during intubation with cricothyroid pressure should help prevent aspiration. The thumb and middle finger exert pressure lateral to the cricoid cartilage, while the index finger pushes directly down on it. If the technique is properly performed, this technique can prevent regurgitation when intragastric pressures rise to as high as 50 cm H_2O or more. Normal intragastric pressure is usually less than 16 cm H_2O so cricoid pressure should be protective in most instances. As soon as the patient is intubated, the cuff on the endotracheal tube is inflated before manual pressure is released.[18]

Airway obstruction may be due to laryngospasm, edema, mechanical obstruction by foreign material, bronchial inflammation, or bronchospasm. Laryngospasm can be broken with either continuous positive airway pressure or a rapid-acting depolarizing muscle relaxant. If the laryngospasm cannot be broken, surgical control of the airway is mandatory. Nebulized racemic epinephrine may improve airway edema.[15]

Suctioning often clears the airway of foreign debris, but if residual material remains, bronchoscopy will be required. In many instances, aggressive chest physiotherapy will help clear the small airways of particulate matter. This is contraindicated in patients with head trauma or spinal cord injuries, however.

The treatment of aspiration pneumonia should be prophylactic. Elevating the head of the bed to prevent gastroesophageal reflux, neutralizing gastric contents, and prophylactic intubation are of the utmost importance. Once aspiration has occurred, adequate oxygenation is mandatory.[17]

On the battlefield, multiply injured casualties will require intubation. Many will also require urgent or immediate operative intervention. Intubation will prevent further aspiration. Casualties with head injuries will require hyperventilation as well.

Bronchial lavage is no longer believed to be indicated because it may worsen hypoxia. Positive airway pressure should be used for diffuse pneumonitis; however, its use in localized aspiration must be tempered against augmenting ventilation–perfusion mismatch. PEEP may be preferentially distributed to normally compliant units and not improve the FRC in the affected lung region. It must therefore be used with caution.

Appropriate fluid resuscitation should not be tempered by the possibility of lung injury. Fluid overload should be avoided but aggressive resuscitation must not. Cardiovascular stability should be maintained with the proper use of inotropic agents when indicated. In more severe cases, a flow-directed pulmonary artery catheter may be beneficial because it allows optimization of the hemodynamic status of the patient, which should improve survival.

Glucocorticoids have no place in the treatment of aspiration. Glucocorticoids suppress host defense mechanisms and predispose the patient to infection. Prophylactic antibiotics are not used either, as they do not prevent pneumonia and tend to select out resistant organisms, thereby increasing morbidity and mortality.[17]

Hydrostatic Pulmonary Edema

Pulmonary edema is often associated with respiratory failure. The type of pulmonary edema must be determined before the condition can be properly treated. These types are *hydrostatic pulmonary edema*, in which fluid accumulates in the presence of a fairly normal blood–air barrier, and *permeability pulmonary edema,* of which ARDS is the classic manifestation. Hydrostatic pulmonary edema occurs because fluid accumulates in the presence of a fairly normal blood–air barrier. In permeability pulmonary edema, fluid accumulation occurs because the blood–air barrier is abnormal.[19]

Hydrostatic pulmonary edema is usually caused by an increase in the intravascular filtration pressure within the pulmonary microvasculature. The elevated filtration pressure increases the fluid flux into the interstitium. If the capacity of the lymphatic system to reabsorb this fluid is exceeded, then the fluid accumulates in the alveoli. Rarely, fluid may accumulate in two other situations: if plasma oncotic pressure is reduced or if the capacity of the lymphatic system to remove fluid is reduced. The intravascular pressure must rise above 7 to 10 mm Hg for water to flood the interstitial space.

With the increased flux of fluid into the interstitium, the fluid moves down a negative interstitial pressure gradient toward the bronchovascular cuffs. This is where the pulmonary lymphatics begin. If lymphatic uptake is decreased, fluid accumulates around the bronchoalveolar cuff until the interstitial pressure is greater than the airway pressure, at which time fluid enters the alveolus. This is typically seen when the pulmonary artery occlusion pressure (PAOP) exceeds 20 mm Hg.

In the civilian population, the causes of hydrostatic pulmonary edema include cardiac dysfunction, pulmonary venous occlusive disease, neurogenic pulmonary vasoconstriction, pulmonary arterial embolization with air, thrombus, or fat; however, these are rarely seen in young, healthy soldiers. In the combat casualty, the most common causes of hydrostatic pulmonary edema are iatrogenic. Asthma and foreign bodies that cause airway obstruction, and pulmonary lymphatic obstruction may also cause hydrostatic pulmonary edema. Clinically, the classic description is that of respiratory distress with pink, frothy sputum and arterial hypoxemia. In trauma patients, this occurs with massive head injury, chest injury, or large amounts of volume resuscitation.[20]

Patients will occasionally present less dramatically with complaints of mild chest tightness or a pressure sensation, a sensation of being unable to catch the breath, and cough. As the capillary pressure continues to increase, and as the fluid flux becomes more pronounced, the patient will become more symptomatic. The respiratory rate increases and the ventilatory pattern becomes labored. This increases the work of breathing required to achieve adequate lung expansion. Respiratory failure will occur because of muscular exhaustion.

Alveolar flooding is accompanied by arterial hypoxemia. Fluid-filled air spaces prevent the passage of gas into the capillary. These air spaces have low ventilation-to-perfusion ratios (ventilation–perfusion mismatch). As the mismatch increases, the effectiveness of hypoxemic pulmonary vaso-

constriction decreases and profound arterial hypoxemia will ensue. Formerly, oxygen was believed to need to diffuse through an abnormally thick layer of water before it reached the red blood cells, but compared to ventilation–perfusion mismatch, this is probably a minor impediment to the exchange of respiratory gases.

The fluid tends to accumulate first in the dependent portions of the lung. Because the pulmonary blood and air flows to the dependent areas are disproportionally small, this fluid accumulation exaggerates the normal propensity for alveoli in the dependent part of the lungs to be underventilated. The pulmonary blood flow is redistributed to less edematous, nondependent alveoli. The FRC decreases with increased critical closing pressure of the alveoli. Other unrelated conditions that decrease FRC (eg, increased intraabdominal pressure, upper abdominal or thoracic incision, and the supine position) can compound the hypoxemia.

The treatment of hydrostatic pulmonary edema must both reverse the changes caused by fluid accumulation and eliminate the underlying disorder that promotes fluid accumulation. The supportive therapy should minimize arterial hypoxemia and decrease the work of breathing. Supplemental oxygen and positive airway pressure are the mainstays of treatment. Administration of supplemental oxygen through nasal prongs or a face mask is useful. However, endotracheal intubation is often required as respiratory failure progresses. If the arterial partial pressure of oxygen (Pa_{O_2}) does not increase after supplemental oxygen therapy is added, then continued hypoxemia is caused by groups of alveoli with low ventilation–perfusion ratios. Positive airway pressure is then delivered as PEEP or CPAP. This will increase the Pa_{O_2} by increasing the FRC of the lungs. (The alveoli are now being ventilated because a higher pressure is being used to force respiratory gas into them.) To decrease the work of breathing, both positive airway pressure and assisted ventilation can be utilized. Positive airway pressure restores lung volume, increases compliance, and increases the cross-sectional area of the airways. It also reduces airway resistance by splinting the airway lumen open. Flow rates will decrease as ventilation becomes less labored, thereby converting a higher-resistance, turbulent flow into a lower-resistant, laminar flow pattern.[21]

Adult Respiratory Distress Syndrome

Lung injury has plagued successful resuscitation of the trauma patient. Lung injury associated with trauma was described in 1932,[22] and ARDS was defined in 1967.[23] For ARDS to occur, the capillary endothelium of the pulmonary vasculature must be injured. This results in dyspnea, hypoxemia, decreased pulmonary compliance, and bilateral pulmonary infiltrates. Pulmonary edema is present but not associated with an increased hydrostatic pressure; it is caused by increased permeability resulting from diffuse alveolar–capillary endothelial injury. ARDS is a common problem affecting previously healthy people. The mortality rate is high: despite vigorous supportive care, it can be greater than 40%.[24]

The syndrome is associated with a wide variety of disease states with multiple underlying etiologies, all demonstrating a single clinical pattern. ARDS can be caused by all types of shock (Exhibit 25-2). A 2.2% incidence of ARDS has been reported

EXHIBIT 25-2

ETIOLOGY OF SHOCK THAT INDUCES THE ADULT RESPIRATORY DISTRESS SYNDROME

- Burns
- Fat emboli
- Lung contusion
- Head trauma
- Near drowning
- Septic states
- Inhalation of toxic gases:
  - Smoke
  - Nitrous oxide
  - Ammonia
  - Chlorine
  - Phosgene
  - Cadmium
- Aspiration of gastric contents
- Drug ingestion
- Pancreatitis
- Multiple transfusions
- Disseminated intravascular coagulation
- Bowel infarction

In casualties with traumatic injuries.[25] The syndrome is more common in casualties with bilateral chest trauma and extensive lung injury than it is in casualties with a localized lung contusion.

Pathophysiology of Alveolar Injury

The normal structural integrity of the alveolus is disrupted in patients with ARDS. Type I pneumocytes cover approximately 95% of the alveolar surface area while Type II pneumocytes cover the remaining 5%. Because of their greater number, Type I pneumocytes are more likely to be injured. Within 24 hours of injury, Type I pneumocytes are destroyed, leaving a denuded basement membrane. Type II pneumocytes, which appear to be more resistant to initial damage, then proliferate and form a continuous layer that covers the previously denuded basement membrane. In addition, the denuded surface may allow aggregates of plasma proteins, cellular debris, fibrin strands, and remnants of surfactant to adhere, which forms the characteristic hyaline membrane. Over the next several days, the alveolar septum thickens, and not only inflammatory cells but also reparative cells proliferate. Hyaline membranes organize, and microatelectasis occurs. The capillary endothelial damage becomes more apparent. Irregularities of the endothelial surface with focal cytoplasmic swelling can be seen, and fibrosis is apparent in the respiratory ducts and bronchioles.[26]

Starling's equation predicts the fluid flux across a semipermeable membrane. It describes the balance between hydrostatic and oncotic pressures acting in opposition across the endothelial barrier. There are two driving forces (the pressure within the capillary, P_c, and the pressure within the interstitium, P_i) and two retaining forces (the colloid osmotic pressure within the capillary, π_c, and the interstitium, π_i). The symbol σ, which is known as the reflection coefficient, represents the impermeability of the membrane to a given substance (ie, if $\sigma = 1$, the membrane is completely impermeable; while if $\sigma = 0$, the membrane is completely permeable). K is a constant representing the filtration coefficient.

Starling's equation states:

$$\text{fluid flux} = K[(P_c - P_i) - \sigma(\pi_c - \pi_i)]$$

Theoretically, the interstitial hydrostatic pressure is slightly negative due to the elastic properties of the lung; therefore, progressively more negative fluid pressures increase the flux into the alveolar space. The small amount of fluid filtered into the interstitial space is readily removed by the extensive pulmonary lymphatic system. Hydrostatic pulmonary edema occurs when the factors favoring fluid flux across the capillary membrane exceed those designed to protect the lungs from developing edema.[19]

The pulmonary edema that characterizes ARDS is caused by alteration of Starling's forces. This alteration is usually due to an abnormally high permeability coefficient; the abnormality is made worse when the capillary hydrostatic pressure increases. The latter abnormality may manifest as elevated pulmonary capillary wedge pressure. Decreased colloid oncotic pressure *can* be caused by severe hypoproteinemia from overhydration with intravenous crystalloid fluid, liver failure, or the nephrotic syndrome; however, it is rarely responsible for lung edema.

Finally, increased interstitial colloid oncotic pressure can occur with increased alveolar capillary membrane permeability (large protein molecules leak out into the interstitium where their oncotic properties attract fluid from the vascular bed). This may be the primary mechanism responsible for the development of pulmonary edema in ARDS. The altered permeability allows the free movement of fluid and protein from the intravascular space, to the interstitial space, to the intraalveolar space. Edema forms at a rate that exceeds the lymphatic system's ability to remove it.

The alteration of the integrity of the alveolar capillaries is believed to result from injuries due to the adverse effects of (*a*) tumor necrosis factor and leukokinin I, which are released from activated macrophages, and (*b*) oxygen free radicals, which are released from neutrophils. Intravascular activation of complement leads to stimulation of neutrophils, sequestration within the capillaries, and subsequent endothelial damage. Patients with ARDS have increased levels of serum C-5A, the activated fifth component of the complement cascade.[26]

Release of C-5A by the alternate pathway causes macrophages and neutrophils to aggregate. The neutrophils release oxygen free radicals; prostaglandins; and granules that contain protease, elastase, coagulase, pepsins and lysozymes. Damage to the basement membrane occurs and increased permeability follows. This allows nonhydrostatic pulmonary edema to form. Arachidonic acid metabolites affect vascular permeability, vascular tone, and airway reactivity, which causes further vasoconstriction and bronchoconstriction.

An additional factor resulting in alteration of the integrity of the alveolar membrane in casualties with massive CNS injury is massive sympathetic discharge, which affects the pulmonary veins. This, in turn, causes profound pulmonary hypertension, which mechanically disrupts the junctions between pulmonary capillary endothelial cells.

Pathophysiology Resulting in Abnormal Gas Exchange

The abnormal gas exchange that occurs in ARDS is due to the increase in extravascular lung water, which tends to close small groups of peripheral alveoli and cause intrapulmonary shunting. This process could be compounded over time by the progressive, extensive fibrosis that occurs in most of these patients. In one study,[27] researchers found that by using multiple, inert-gas elimination techniques in patients with ARDS, some degree of ventilation–perfusion mismatch occurred in 40% of patients. There was no evidence of a diffusion abnormality as it applies to respiratory gas exchange.

ARDS also causes changes in lung mechanics. Both the FRC and pulmonary compliance decrease. Loss of surfactant causes alveolar collapse, leading to further decreases in both FRC and pulmonary compliance. In addition to surfactant abnormalities, flooding of the alveoli decreases alveolar volume and causes further atelectasis. Compliance is further reduced by interstitial and alveolar edema, fibrosis, and the abnormal surfactant.

Clinically, during phase I, the patient will be dyspneic and will demonstrate a respiratory alkalosis with tachycardia and tachypnea (Table 25-1). A latent period, phase II, will occur, lasting from 6 to 48 hours after the injury, during which the patient will appear stable and may appear to be improving. During phase II, the patient will begin to demonstrate an increase in the work of breathing, with hyperventilation and hypocarbia; the lungs become less compliant due to both movement of fluid into the alveoli and loss of surfactant. Decreased alveolar volume and widespread atelectasis are the consequences of these changes (Table 25-2).

Physical examination will demonstrate scattered rales and evidence of the use of the accessory muscles of respiration. The initial chest X-ray examination will be normal, but subsequent radiographs will demonstrate increases in interstitial markings. With continued progression of the disease, the patient will develop acute respiratory failure (phase III). Lung compliance will be markedly decreased due to interstitial and alveolar edema, fibrosis, and abnormal surfactant. Loss of the normal surfactant leads to a decrease in the FRC and thus a further decrease in compliance. Further decreases in the FRC increase the alveolar–arterial oxygen difference and worsen the hypoxia.

The chest radiograph will demonstrate a diffuse interstitial pattern and, in fact, pneumonia is often difficult to rule out. At this time, bronchoalveolar lavage for diagnosis may be indicated to identify the pneumonic process.

The patient may improve at this time. The physical findings will resolve and the chest radiograph will return to normal. More commonly, however, the disease progresses with severe unresponsive hypoxemia, increased intrapulmonary shunting of blood, and metabolic and respiratory acidosis. The radiograph demonstrates honeycombed, patchy, and ill-defined nodular densities.

Treatment

Treatment of the patient with ARDS encompasses measures to (*a*) prevent death from respiratory failure, (*b*) minimize further lung injury, (*c*) provide general support, and (*d*) recognize and treat complications.

The goal of therapy is to provide adequate oxygenation at the lowest possible F_{IO_2}. High inspired oxygen concentrations, as well as high airway pressures, have the potential to significantly exacerbate the acute lung injury.

Initially, the lowest F_{IO_2} needed to provide an arterial hemoglobin saturation (Sa_{O_2}) of 90% or more is used. Continuous pulse oximetry is used to monitor therapy. Decreasing the F_{IO_2} to 50% is desirable, and to achieve it, end-expiratory pressure is frequently used. PEEP is begun at 5 cm H_2O and increased incrementally with careful attention to cardiac output, pulmonary compliance, and peak inspiratory pressure. The medical officer's clinical judgment may be all that is available to assure optimal oxygenation.

Mechanical ventilation is used to deliver increased F_{IO_2} and increased tidal volumes. Tidal volumes of 10 to 15 mL/kg should be used initially. Careful attention is required to prevent the increased peak inspiratory pressure from causing barotrauma.

The need for high peak inspiratory pressure may be dictated by decreased compliance of the ARDS lung. However, other causes that may give rise to high peak inspiratory pressure are endotracheal tube displacement, pneumothorax, increased pleu-

TABLE 25-1

CLINICAL FINDINGS IN ADULT RESPIRATORY DISTRESS SYNDROME

Phase	Findings
Phase I:	Normal auscultatory examination
Acute Injury	Normal chest X-ray examination
	Tachycardia
	Tachypnea
	Respiratory alkalemia on arterial blood gas testing
Phase II:	Minor abnormalities on auscultatory examination
Latent Period (6–48 h)	Minor abnormalities on chest X-ray examination
	Tachycardia and tachypnea persist
	Increased work of breathing
	Respiratory alkalemia
	Increased $P_{AO_2} - Pa_{O_2}$
Phase III:	High-pitched rales to auscultation
Acute Respiratory Failure	Diffuse infiltrates on chest X-ray examination
	Marked tachypnea and dyspnea
	Decreased lung compliance
	Worsening oxygenation and ventilation
Phase IV:	Severe, unresponsive hypoxemia
Terminal Abnormalities	Increased intrapulmonary shunting
	Severe metabolic and respiratory acidemia

$P_{AO_2} - Pa_{O_2}$: alveolar-arterial difference in partial pressure of oxygen

TABLE 25-2

PATHOPHYSIOLOGY OF THE ADULT RESPIRATORY DISTRESS SYNDROME

Findings	Mechanisms
Refractory hypoxemia	Increased intrapulmonary shunting
	Decreased FRC
	Release of inflammatory mediators
Diffuse infiltrates on chest X-ray examination	Alveolar edema
	Atelectasis
	Inflammatory cell infiltrates
Normal PAOP	Endothelial damage results in capillary leakage
Decreased pulmonary compliance	Edema
	Atelectasis
	Marked increase in extravascular lung water

FRC: functional residual capacity; PAOP: pulmonary artery occlusion pressure

ral effusion, increased lung edema, bronchospasm, mucous plugging, and high flow rates. Adjusting the tidal volume, the level of PEEP, or using paralytic agents to relax the patient's respiratory muscles may be required to decrease barotrauma. The mode of ventilation may be either intermittent, mandatory ventilation (IMV) or assist control (AC), both of which are discussed later in this chapter. Neither mode has been shown to be superior. The mode with which the operator has the most experience should be the mode used.

The use of glucocorticoids in the treatment of ARDS is not resolved. In the past, high-dose glucocorticoids have been advocated, but a prospective randomized trial published in 1987[28] reported that no benefit was seen from the use of glucocorticoids. However, a meta-analysis published in 1995[29] suggests that a subset of patients may benefit from corticosteroids administered during the fibroproliferative phase of the disease process.

Even with aggressive, intensive care, the mortality rate for ARDS remains approximately 50%. Of patients who survive the disease and its complications, most will become clinically asymptomatic. A small percentage, however, will have mild-to-moderate dyspnea on exertion. In the majority, the chest X-ray examination will return to normal, as will their lung volumes. Resting arterial blood gases will tend to be normal but with exercise, approximately half of these patients will have some decrease in their Pa_{O_2}. Surprisingly, the typical ARDS patient does not die of respiratory insufficiency but of complications of unrelenting sepsis with failure of other organ systems.[30]

Although not presently applicable to combat situations, the use of extracorporeal membrane oxygenation with low-frequency positive pressure ventilation may be of some benefit to patients with ARDS. Although several studies of note have shown no improvement in survival when extracorporeal membrane oxygenation was used for oxygenation, others have used the apparatus with low-frequency positive pressure ventilation to some success. Lung function improved in 73% of patients and 49% survived, compared with the previous mortality rate of 91%.[31] An intra–vena caval blood oxygen–extracorporeal carbon dioxide removal device is presently undergoing clinical trials. Preliminary data indicate that there is no improvement in survival compared with conventional therapy.[32]

DIAGNOSING RESPIRATORY FAILURE

Because the causes of respiratory failure are so varied, astute physicians always search for incipient respiratory failure in their patients. The term *acute respiratory failure* comprises disorders of oxygenation or ventilation or both. Its definition, likewise, has both oxygenation and ventilatory components:

- *hypoxemia*: while breathing room air, the partial pressure of oxygen (P_{O_2}) is 50 mm Hg or less; and/or
- *ventilatory failure:* the inability to ventilate sufficiently to maintain arterial blood partial pressure of carbon dioxide (Pa_{CO_2}) less than 50 mm Hg.[21]

Although disorders of oxygenation and ventilation frequently coexist, consideration of each as a separate entity allows for a clearer understanding of the pathophysiological processes that lead to respiratory failure.

Hypoxemia

The signs and symptoms of hypoxemia are the effects of, and the body's response to, insufficient D_{O_2} to tissues for metabolic function. Cyanosis is only detectable when the patient's blood contains at least 5 g of deoxygenated hemoglobin per deciliter of blood. Similar findings (ie, signs and symptoms of hypoxemia) are noted when the level of methemoglobin reaches 1.5 g/dL, which may be seen following treatment for cyanide toxicity.[6] Typically, CNS effects are prominent with significant hypoxemia. Confusion, restlessness, and loss of judgment are common manifestations. The lack of available oxygen leads to increased sympathetic tone as the body attempts to compensate for inadequate D_{O_2} to the tissues. Lactic acidosis with a compensatory respiratory alkalosis becomes prominent as cells convert to anaerobic metabolism to provide for energy needs.

The major causes of hypoxemia in the trauma patient are a direct result of abnormalities in ventilation–perfusion relationships within alveolar capillary units. The deoxygenated blood that perfuses the alveolar capillaries in areas of hypoventilation within the lung cannot be normally oxygenated. The oxygenation deficit in this circumstance can be corrected by supplemental oxygen. This will increase the relative amount of oxygen within the alveoli in the region of low ventilation.

Although a number of pathophysiological processes produce hypoventilation, atelectasis from direct trauma to the lung parenchyma is the most common. Overly aggressive volume resuscitation with increased lung water is also frequently encountered in the setting of trauma. The second mechanism for the development of arterial hypoxemia, that of right-to-left intrapulmonary shunting of blood, can be thought of as the extreme form of altered ventilation and perfusion. In this instance, pulmonary arterial blood traverses capillaries in regions of complete alveolar collapse or regions that receive no ventilation from airway obstruction. Supplemental oxygen does not correct the oxygenation deficit. Correction is accomplished by inflating these collapsed alveoli, and it is necessary to resort to either continuous positive airway pressure via face mask or endotracheal intubation to improve oxygenation.

In combat medical facilities, supplemental oxygen is delivered from a tank. Oxygen *sieves* (concentrators that can remove and concentrate oxygen from ambient air) will be available in future conflicts to supply supplemental oxygen. In addition to the signs and symptoms of hypoxemia, assessment of the patient's degree of oxygenation requires arterial blood-gas measurement and pulse oximetry. The echelon of combat medical care that will have blood-gas monitoring capabilities is unclear; however, transportable pulse oximetry is available and could be utilized at mobile army surgical hospitals (third echelon), combat support hospitals (third echelon), and possibly even at the clearing station (second echelon).

Ventilatory Failure

Ventilatory failure may result from a number of different pathological processes. The two disorders most commonly associated with respiratory insufficiency leading to ventilatory failure are (1) CNS depression from trauma or drug effect and (2) compromise of the respiratory function of the chest wall. Disorders of the chest wall (eg, flail chest) may occasionally lead to ventilatory failure manifested by hypercarbia. However, associated abnormalities of oxygenation tend to represent the more life-threatening condition. Clinical signs and symptoms of hypercarbia are similar to those noted with hypoxemia, but with these exceptions: cyanosis is absent while oxygenation is maintained, and obtundation is more common than agitation. As it does with failure of oxygenation, optimum determination of the adequacy of ventilation rests on the physician's interpretation of arterial blood-gas determinations. Portable capnography provides a reasonable estimate of arterial carbon dioxide content and may prove useful in situations in which blood gas determinations are not available.[21]

Combined Hypoxemia and Ventilatory Failure in Postoperative Casualties

Postoperative pulmonary complications are among the commonest causes of morbidity and mortality in this patient population. Of all the potential respiratory complications, atelectasis is easily the most common: it may account for up to 90% of postoperative respiratory complications.[33] The degree of involvement may range from a small, insignificant group of airways to complete collapse of an entire lung. Other less common but frequently more life-threatening complications include pneumonia, bronchospasm, pulmonary thromboembolic disease, and respiratory failure. It is possible to define the various abnormalities with a combination of radiographic, laboratory, and clinical features. Chest X-ray examinations, blood-gas measurements, and chest auscultation remain the most valuable techniques to define pulmonary abnormalities and determine the need for more-invasive measures such as bronchoscopy, endotracheal intubation, or pulmonary arteriography.[34]

Upper abdominal and thoracic surgery carry the greatest risk for postoperative pulmonary dysfunction. Splinting to ease pain reduces the patient's ability to cough and breathe deeply, both of which predispose to distal airway plugging and collapse. The splinting results in a reduced vital capacity that leads to hypoventilation, while the airway plugging causes atelectasis and hypoxemia. Another important factor, a direct result of general anesthesia, is depression of mucociliary transport. Abnormal ciliary action and production of mucus are both worsened by lengthening anesthesia time. Cigarette smoking potentiates this abnormality.

Surgical procedures also predispose to premature closure of the distal airways. Supine positioning, increased abdominal girth and breathing at reduced lung volumes are nonpulmonary factors that contribute to premature airway closure. Interstitial edema, airway obstruction from secretions and bronchoconstriction, and loss of surfactant are pulmonary factors that tend to promote airway closure and atelectasis.[34]

A routine program of respiratory therapy before, during, and after surgery can reduce the number of

postoperative respiratory complications and improve patient outcome when they do arise. The first few hours after the operation are the most critical for preventing respiratory complications. Alveolar collapse, with the resulting loss of FRC, may be prevented or reversed by deep breathing. Maneuvers that utilize forced exhalation (eg, blow bottles) do not effectively prevent airway collapse and may, in fact, promote this problem if an ineffective inspiration precedes the forced exhalation. Deep-breathing exercises, with inspiration to total lung capacity, are easily the most effective, cost-efficient means to prevent and reverse atelectasis (and hence, most pulmonary complications). Incentive spirometers allow patients to participate in their therapy and free the respiratory therapist to perform other duties. Most studies have found that intermittent positive-pressure breathing is, at best, equal to, and in most cases, inferior to incentive spirometry as a prophylactic measure to prevent postoperative pulmonary complications.[33]

The likelihood that a patient will develop postoperative respiratory complications is directly related to the type of surgical procedure and the duration of surgery and anesthesia; the patient's smoking history, preexisting pulmonary disease, obesity, and overall physical condition; and other factors.[35] For the most part, soldiers are in excellent physical condition and do not demonstrate significant preexisting lung disease.

MANAGING RESPIRATORY FAILURE

Although exsanguination is the most common cause of early death among combat casualties, medical officers must not forget that the first step to be taken in the management of any trauma victim is to assure a patent airway.[36] Respiratory failure must be recognized and treated promptly. Upper airway obstruction may not be obvious on initial assessment in the field, and may recur anytime during the medical-evacuation process. All military medical personnel should be trained in simple maneuvers to relieve upper airway obstruction. Should a hypovolemic trauma casualty require endotracheal intubation and positive-pressure mechanical ventilation, it is especially important that cardiovascular resuscitation be continued, to mitigate against the possible adverse effects of the positive pressure.

Supportive Care

Respiratory care begins with the use of supplemental oxygen. The most common methods to employ this drug are nasal cannulae and face masks. The F_{IO_2} actually achieved with a nasal cannula is dependent upon the patient's inspiratory flow rate and respiratory rate. A variety of masks are available for oxygen therapy. Higher percentages of F_{IO_2} may be obtained by use of the partial rebreathing mask or the nonrebreathing mask. The latter is the type most frequently applied to patients following general anesthesia, and is available in deployable medical facilities, including field echelons. The use of such a mask, which utilizes *three* unidirectional valves, ensures that carbon dioxide will not be rebreathed.

The next most common form of respiratory care used for patients is incentive spirometry. The success of this therapy in expanding the patient's FRC depends on both achieving adequate inspiratory volume and sustaining maximum inspiratory effort. Although preoperative teaching is often done in the setting of elective surgery, this obviously will not be possible in the combat casualty setting.

Intratracheal suctioning is important in maintaining the patency of the proximal airway and in preventing secretions from obstructing the alveoli. This is particularly true in the casualty with a penetrating chest wound, where the potential exists for bleeding into the airway. The casualty can drown in his own blood. Intratracheal suctioning is not entirely benign: it can be associated with complications such as trauma to the tracheal mucosa with resultant hemorrhage, hypoxia, and the vasovagal response leading to bradycardia and hypotension.

Endotracheal Intubation

Although most patients who require endotracheal intubation will also require mechanical ventilatory support, there are clinical situations in which intubation should proceed independent of the criteria defining respiratory failure. Intubation is required to provide airway protection and to prevent aspiration in a patient who is semicomatose. The treatment of upper-airway obstruction following direct maxillofacial trauma and the prevention of complete airway obstruction following thermal burn injuries are two other clinical examples. A full discussion of the methods of endotracheal intubation is found in Chapter 3, Airway Management.

Prolonged endotracheal intubation brings with it many possible complications. The posterior

arytenoid cartilages are the most common sites for laryngeal damage, which is caused by the anterior bend imposed on the endotracheal tube at this point. Although the definition of prolonged translaryngeal intubation and the timing of tracheostomy are not yet answered in the medical literature, most clinicians consider 14 to 21 days to be a safe period before performing a tracheostomy.[37]

The complications of an elective tracheostomy are generally greater than those of translaryngeal intubation, although the overall incidence of complications is still low (3%). Early stomal bleeding is common; bleeding after 48 hours is more worrisome because it may indicate a catastrophic complication: a fistula between the trachea and the innominate artery. Tracheal–innominate artery fistulae occur infrequently but have a high mortality.

An increased incidence of bacteremia and nosocomial pneumonia are other problems associated with tracheostomy. Studies[38] have shown that up to 87% of patients with tracheostomies experience chronic aspiration. For example, tracheal stenosis is a common, clinically significant complication. The reported incidence varies, but exceeds that seen in patients with endotracheal tubes.[37] The most common site for tracheal stenosis is the stoma itself. A 75% reduction in the diameter of the trachea may occur before symptoms are manifested. The patient who is developing tracheal stenosis—who experiences stridor at rest—probably has a tracheal diameter less than 5 mm.

PHYSIOLOGICAL EFFECTS OF MECHANICAL VENTILATION

Once an endotracheal tube is used for ventilation, the patient's natural mechanism for the warming and humidification of inspired air are bypassed. Immediately following intubation and the institution of mechanical ventilation, patients may experience physical and psychological discomfort, which results in coughing and agitation. Most patients—especially those who are awakening, alert, or mildly obtunded—attempt to "fight" the ventilator.[39] Nearly constant medical supervision is required, and restraints may be necessary to avoid self-extubation.

Pulmonary Effects

Positive pressure applied during inspiration results in an alteration of the normal ventilation–perfusion matching. During spontaneous ventilation, most pulmonary blood flow and ventilation occurs in the dependent parts of the lungs. This relationship is changed during mechanical ventilation such that the greatest ventilation now occurs in the superior aspects of the lung. This is most likely the result of changes in the diaphragmatic shape and motion following positive airway pressure. The result is an increase in the physiological dead space following the institution of mechanical ventilation.

The use of positive-pressure ventilation may also create changes in the lung mechanics. Reductions in the FRC and pulmonary compliance may result in an increase in the work of breathing, and increased minute ventilation requirement.

The application of expiratory distending pressure results in an improvement of gas exchange and pulmonary compliance secondary to an increase in lung volumes. The mechanism of the increase in FRC is not clearly known but probably is the result of one or more of the following:

- an increase in the transpulmonary pressure;
- "recruitment" of atelectatic alveoli, resulting in their reexpansion; or simply
- the maintenance of alveolar patency, which was achieved during inspiration.

It is unlikely that the application of PEEP can open atelectatic alveoli that were not reexpanded during positive-pressure ventilation. Expiratory distending pressure also causes a decrease in the closing volume below the FRC, which mitigates against airway collapse during exhalation.

Conversely, an increase in lung volume may be detrimental if the FRC exceeds the normal volume. Excessive increases in FRC reduce pulmonary compliance and increase the pulmonary vascular resistance and dead space. Contrary to earlier beliefs, applying expiratory distending pressure actually *increases* total lung water, although oxygenation usually improves.

Cardiovascular Effects

The effects of expiratory distending pressure can be divided into (*a*) the effects on pulmonary vascular resistance and (*b*) the effect on cardiac output. The pulmonary vascular resistance is lowest when the FRC is normal. If the FRC is returned to normal by the recruitment of atelectatic alveoli, and venous

admixture is decreased, hypoxic pulmonary vasoconstriction will be reduced. In contrast, if the alveolar pressure is increased beyond normal (thoracic overinflation), pulmonary capillary pressure will rise, resulting in an increase in the pulmonary vascular resistance.

The varying effect of expiratory distending pressure in different patients depends on thoracic compliance, which governs the transmission of pressure to the pleural space and great vessels. When the pulmonary compliance is low (eg, in a patient with ARDS), a minimal amount of expiratory distending pressure is transmitted to the pleural space. Thus, these patients frequently tolerate high levels of PEEP relatively well. If, however, the thoracic compliance decreases from abdominal distention or postoperative atelectasis, much of the expiratory distending pressure will be transmitted to the pleural space. This results in a potentially significant decrease in both venous return and cardiac output. The treatment is to increase the peripheral venous pressure, usually by volume administration. The extent to which this complication occurs may also be minimized by permitting spontaneous breaths through the use of IMV. Negative-pressure, spontaneous breaths serve to minimize the effect of expiratory distending pressure by augmenting venous return.

The effect of expiratory distending pressure on ventricular afterload differs between ventricles. An increase in right ventricular afterload is not predictable and depends upon the amount of increase in the pulmonary vascular resistance. At higher levels of PEEP (> 15–20 cm H_2O), right ventricular dilation occurs resulting in a shift of the intraventricular septum and possibly contributing to a decreased left ventricular stroke volume and cardiac output.[40] In this setting, the PAOP or pulmonary capillary wedge pressure (PCWP) may not reflect the true left ventricular end diastolic volume. As a result of an increased pleural pressure, the left ventricle may be compressed (ie, assisted). A failing left ventricle performs better when the afterload has been reduced.

Renal Effects

High levels of expiratory distending pressure have been associated with decreased urinary output. Animal studies comparing continuous, positive airway pressure to spontaneous breathing (ie, IMV) with PEEP failed to implicate reduced cardiac output as the etiologic factor.[41] Two possible explanations are (1) an increase in venous pressure, resulting in a decreased glomerular filtration rate and (2) an alteration of the distribution of intrarenal blood flow.

COMPLICATIONS FROM MECHANICAL VENTILATORY SUPPORT

A variety of complications may occur during mechanical ventilation. Two of the most important are cardiovascular depression and barotrauma. First, positive-pressure ventilation necessarily increases intrathoracic pressure. The increased intrathoracic pressure reduces venous return to the heart, and, therefore, right ventricular preload. This has two effects: (1) the reduction in preload results in a decrease in cardiac output; and (2), right-ventricular afterload increases, probably as a result of direct compression of the pulmonary vasculature. These two detrimental effects act in concert to reduce cardiac output, and both effects tend to be magnified by hypovolemia and PEEP.[21]

The second major complication of positive-pressure ventilation is barotrauma. The spectrum of barotrauma extends from pulmonary interstitial emphysema through pneumomediastinum, pneumothorax, and subcutaneous emphysema.[42] Current theories on barotrauma relate the initial event as alveolar rupture with dissection of air along the vascular sheath to the mediastinum. Once air enters the mediastinum, it may track along the fascial planes into the pleural space, subcutaneous tissue, or retroperitoneum.[43] It is the development of pneumothorax, and in particular, of tension pneumothorax, that represents the greatest threat to the patient. Peak airway pressure is believed to be the primary factor related to the development of barotrauma. No barotrauma was found in one series of patients whose peak airway pressure could be maintained at less than 50 cm H_2O.[42] Although experts debate the maximal pressure that can be tolerated without barotrauma, the practice of minimizing peak airway pressure to forestall the development of barotrauma is standard for managing patients with respiratory failure.

The finding of pulmonary interstitial emphysema on chest X-ray examination should alert the clinician to the real possibility that the patient may soon develop a pneumothorax. Because pneumothorax may present abruptly, the diagnosis should be made on the basis of clinical examination. The combination of respiratory distress, hypotension,

hyperresonance, diminished breath sounds, and tachycardia should lead the clinician to evacuate the presumed pneumothorax with a large-bore needle placed in the second or third intercostal space in the midclavicular line on the affected side. Following immediate decompression by needle drainage, a tube thoracostomy should be performed, with the tube connected to a Heimlich valve or a water seal to preclude reaccumulation of air in the pleural space.

ETHICAL DILEMMAS IN USING LIMITED LIFE-SUSTAINING THERAPY

"Battlefield military medicine has a threefold mission: to save lives, to alleviate suffering, and to return soldiers to duty."[15(p192)] The question exists as to whether mechanical ventilation is a useful component of the medical officer's armamentarium for the treatment of combat casualties. In other words, is it likely that the casualty who requires mechanical ventilation will be returned to the battlefield? Probably not, with these two exceptions: when perioperative ventilation is used for residual anesthetic effects, and when mechanical ventilation is used to support casualties of chemical war gases. However, the mission of battlefield medicine is also to save lives, and the use of mechanical ventilation to accomplish this goal is without dispute.

When the number of mechanical ventilators is limited, medical officers will be required to decide which patients will receive them. This decision may be based on several considerations. Probably the most important, and least-biased, factors are (*a*) the expected reversibility of the inciting injury and (*b*) the predicted patient outcome. For example, the casualty who develops ARDS will most likely require at least 2 to 3 weeks of mechanical ventilatory support. In addition, the mortality rate exceeds 40%.[24] If this patient receives a ventilator, several others with an expected quick recovery may either sustain neurological injury or death if no respirator is available for respiratory support. A second example is the patient who develops multiple organ failure. If three or more organ systems have failed for at least 3 days in the ICU, the mortality exceeds 95% to 97%.[44]

Another factor that must be considered when a limited number of mechanical ventilators is available is the medical officer's experience and skill in managing critically ill patients *nonmechanically*.

MECHANICAL VENTILATION AND COMBAT CASUALTIES

The medical officer working in the combat casualty environment must (*a*) understand the types of pulmonary injuries that may occur and (*b*) be able to evaluate the need for mechanical ventilatory support. The decision to use a mechanical device to assist ventilation is primarily based on the patient's inability to sustain sufficient gas exchange. The most common reason for requiring positive-pressure ventilation is refractory hypoxemia. Historically, this use of positive-pressure ventilation dates back to 1938 for the treatment of pulmonary edema.[45]

Military anesthesiologists and critical care specialists must understand the design of mechanical ventilators, be able to choose the proper mode of ventilation, and be able to select the appropriate initial ventilatory settings. These required settings include tidal volume, rate, peak gas flow, inspiratory-to-expiratory ratio, and the fractional concentration of oxygen. A further challenge to medical officers is the lack of sophisticated monitoring for casualties who are mechanically ventilated. Thus, a good understanding of pulmonary physiology and astute clinical-assessment skills are required to determine if the patient is appropriately ventilated and oxygenated.

In contrast to the Vietnam War, where the capability for mechanical ventilation was very limited, in future conflicts, state-of-the-art ventilators may be available to save the lives of casualties who would otherwise die. During the months following the 2 August 1990 invasion of Kuwait by the Iraqi army, the Department of Defense purchased many types of mechanical ventilators, which were sent to the Saudi Arabian theatre. An unexpected problem arose, however: although many different kinds of ventilators were available, most of them were unfamiliar to young physicians new to the military.

Additional problems relating to the provision of mechanical ventilation in the field were the need for oxygen, oxygen blenders, PEEP valves, and spirometers. The principal problem on the battlefield is the availability of compressed oxygen. During the Vietnam War, oxygen was provided principally in cylinders, with replenishment by liquid oxygen. In future conflicts, there are no plans for pipeline oxygen supply in the land hospitals; however, there should be an increased availability of liquid oxy-

gen. Use of oxygen sieves may become commonplace. The logistics of continual provision of filled oxygen tanks is significant.

Indications for Mechanical Ventilation

A combat casualty's need for mechanical ventilatory support generally falls under one of three categories: impaired oxygenation, inadequate ventilation (eg, the central ventilatory drive or the functional integrity of the thoracic cage is impaired), or an excessive amount and/or inefficiency of respiratory work. A number of clinical problems are associated with more than one of these deficiencies (Exhibit 25-3).

Several of these indications are self-explanatory; however, a few merit specific comments. Oxygen itself may be toxic to the pulmonary system when administered in a high quantity. If a peripheral oxygen saturation above 90% cannot be obtained within a reasonable amount of time (< 12 h) by the use of a face mask–nonrebreathing apparatus ($FIO_2 > .80$), then endotracheal intubation and mechanical ventilation should be instituted. Improved oxygenation may be achieved by manipulation of the FIO_2, mean airway pressure, or the pattern of ventilation.

Although patients in cardiovascular collapse or shock may not meet the arterial blood-gas criteria for respiratory failure, supportive care for these patients includes using mechanical ventilation to reduce work demands placed on a patient with inadequate cardiac output. Data from animal studies support this approach, as mortality was reduced in the group of animals in shock that were supported with mechanical ventilation.[39]

EXHIBIT 25-3

INDICATIONS FOR MECHANICAL VENTILATION

Impaired Oxygenation
- Direct thoracic trauma with resulting pneumothorax
- Hemothorax or pulmonary contusion
- Hypovolemia and shock
- Pulmonary edema
- Progressive atelectasis
- Pneumonia or aspiration pneumonitis
- Adult respiratory distress syndrome

Impaired Ventilation
- Postoperative state with residual anesthesia
- Head injury
- Chemical warfare injury
- Quadriplegia from spinal cord trauma
- Diaphragmatic injury
- Fractured ribs or flail chest

Excessive Respiratory Work
- Multiple trauma
- Compensation for severe metabolic acidemia
- Severe bronchospasm
- Sepsis or multiple organ dysfunction

Functional Design of Mechanical Ventilators

The type of ventilator is designated by its mechanism of cycling (ie, the event that terminates inspiration). There are three principal types of cycling: pressure, volume, and time. Both volume and time allow for a constant level of tidal volume. The tidal volume varies during use of pressure-cycled ventilators whenever pulmonary compliance or airway resistance change. An important component of the ventilator's design is its driving power (ie, what empowers positive pressure); either compressed gas (usually oxygen) or electrical power may be used. Most modern ventilators, and all that are designed for use in ICUs, are electrically powered, have an internal battery for back-up power, and are often controlled by a microprocessor. For medical officers, this advantage is significant: it reduces the amount of compressed oxygen that must be supplied to support field hospitals.

Simplistic, mechanical ventilators have only a few components: a gas-controller device, blender, humidifier, PEEP generator, and the circuit tubing. The gas-controller device provides flow either on demand, based on input from a sensor, or by continuous flow.

The limitations of specific ventilators are discussed later in this chapter.

Pressure Cycled

In pressure-cycled ventilators, inspiration ends when a preset pressure is reached in the proximal airway, as sensed by a manometer placed within the ventilator circuit. The Bennett PR-2 is an example of this type of ventilator, and is available in military warehouses. It is one of the first kinds of ventilators

sent to Saudi Arabia in 1990. However, a pressure-cycled ventilator is used mainly in the patient who is comatose, quadriplegic, or postoperative from general anesthesia; or who has a large leak around the endotracheal tube and an anesthesiologist or clinician who is experienced in reintubation is unavailable. Many limitations discourage the use of the PR-2.

Volume Cycled

In volume-cycled ventilators, the inspiratory phase ends when a preset volume is delivered to the patient. This is the most common type of cycle mechanism on modern ventilators and on all machines classified as "ICU ventilators." Ventilators of this type that were sent to the Persian Gulf War to support U.S. and coalition forces include the Bear 33, Lifecare PLV-100 and PLV-102, Bennett MA-1, and the Puritan-Bennett Companion. A safety feature of an appropriately set peak airway pressure prevents excessive pressure in the airway if either pulmonary compliance or airway resistance change suddenly. When this "pop-off" pressure is reached, the remainder of the tidal volume vents out of the circuit and remains undelivered to the patient. Volume cycling also has some limitations, including the need for a good seal around the endotracheal tube, and an inspiratory time that is unresponsive to the patients' own respiratory pattern.

Time Cycled

In time-cycled ventilators, the inspiratory phase ends when the selected time is reached. Ventilators of this type include the Servo 900C (Siemens-Elema, Solna, Sweden); the Evita (Dragerwerk, Bonn, Germany); and the Univent-750 (Impact Instrumentation, West Caldwell, N.J.). The Univent-750 was designed as a transport ventilator and large quantities were sent to the Persian Gulf during Operation Desert Storm. Similar to the volume-cycled ventilator, the time-cycled ventilator delivers a relatively constant tidal volume. Tidal volume is determined by either (*a*) the inspiratory time and the peak inspiratory flow rate or (*b*) the minute ventilation and the respiratory rate. For example, if the inspiratory time is 1 second, and the peak flow rate is 60 L/minute, then the set tidal volume will be 1,000 mL. The Evita, also a time-cycled ventilator, is an extremely flexible and capable ICU ventilator and was supplied in large quantity to receiving hospitals in the U.S. Army Seventh Medical Command, in Germany.

Flow Cycled

Pressure-support ventilation is generally viewed as a mode of ventilation and may be performed on either volume-cycled or time-cycled machines. Nevertheless, the mechanism for termination of inspiration (cycling) is unique in that flow to the patient stops when the patient's intrinsic inspiratory flow falls below a certain percentage of the initial effort.

This topic is discussed more fully below.

Modes of Positive-Pressure Ventilation

There is no uniformly accepted terminology for the different ventilatory modes. The application of positive airway pressure can be divided according to the timing during inspiration or exhalation, or during both phases. When comparing the following modes of ventilation, one general concept should be remembered: positive intrathoracic pressure may contribute to decreased venous return and, therefore, to inadequate cardiac output. The degree to which this complication becomes clinically significant depends on the duration and level of positive airway pressure, whether any spontaneous (negative-pressure) breaths occur, and the patient's preceding intravascular volume and cardiac status.

Inspiratory Positive Pressure

Controlled Mechanical Ventilation. In the controlled mechanical ventilation (CMV) mode, the ventilator initiates positive-pressure inspiratory cycles independent of the patient's spontaneous efforts. This method is indicated when the patient is apneic secondary to heavy sedation, paralysis, or coma; or when neuromuscular pathology, including quadriplegia, has occurred. A problem arises when the patient tries to breathe spontaneously: the patient may compete with the ventilator. The result will be a patient who is at best uncomfortable, and at worst subject to pulmonary overinflation and barotrauma. All inspiratory cycles are positive pressure, thus decreased venous return and decreased cardiac output are common. If the patient does not initiate spontaneous efforts, then the entire work of breathing is provided by the ventilator. This allows the patient to rest, but also may lead to atrophy of the diaphragm and intercostal muscles.

Assist-Control Ventilation. Again, all inspiratory cycles are positive pressure with the AC mode of ventilation. Any breaths taken above the rate set

by the physician must be triggered by a spontaneous effort. Competition between the patient and the ventilator is therefore avoided, which reduces the potential for barotrauma. The primary advantages of AC ventilation are that (1) respiratory muscles rest when the peak flow is high enough to decrease the patient's work of breathing and (2) the patient determines the P_{CO_2} and the pH. Unfortunately, respiratory alkalosis is frequently observed in critically ill patients. When AC ventilation is used, the medical officer must remember that the set respiratory rate is only a back-up if the patient's spontaneous rate falls below that frequency. The actual frequency of full tidal-volume breaths will be determined by the patient's spontaneous rate or the set, mechanical rate—whichever is the greater number.

Intermittent, Mandatory Ventilation. The IMV mode allows unlimited, negative-pressure, spontaneous breaths between the delivered mechanical breaths. As a result, this mode can be used across a spectrum of minimal to complete ventilatory support, offering flexibility to the physician whose patient load requires a large number of ventilators or who is attempting to wean a patient from the ventilator. IMV is indicated primarily during weaning. However, when the respiratory rate and tidal volume are set high enough to provide most of the patient's minute ventilation requirement, the patient can rest, and the work of breathing can be reduced. The use of IMV has several advantages (Exhibit 25-4).

Synchronized, Intermittent, Mandatory Ventilation. The synchronized, intermittent, mandatory ventilation (SIMV) mode was designed to improve patient comfort and to prevent the stacking of breaths (ie, repeated inhalations without full exhalation) that may lead to barotrauma. As with IMV, the set rate is the minimum number of positive-pressure breaths that the patient will receive regardless of his intrinsic central respiratory drive. However, in SIMV, the patient's spontaneous breathing and the ventilator are coordinated by means of a circuit manometer, which detects the onset of a spontaneous breath. Within a "window" during which the ventilator should cycle (eg, every 6 s when the set rate is 10 breaths per minute), if the patient makes a spontaneous effort, the mechanical breath will be triggered and superimposed at the beginning of the patient's respiratory cycle, rather than randomly throughout. This timing should help avoid barotrauma by synchronizing the ventilator-delivered breath with the inspiratory effort of the patient. The only significant disadvantage of the IMV or SIMV ventilatory mode is the potential increase in work of breathing imposed by the demand valve incorporated into the ventilator.

EXHIBIT 25-4

ADVANTAGES OF INTERMITTENT, MANDATORY VENTILATION

- Distribution of ventilation is more normal
- Physiological dead space is reduced
- Detrimental effects on the cardiovascular system are minimized
- Some degree of respiratory muscle tone is maintained
- Respiratory alkalemia is minimized

Pressure Support Ventilation. The pressure support (PS) mode of ventilation is triggered by the negative flow created by a patient's spontaneous breath. The initial use of this mode was the intermittent, positive-pressure breathing (IPPB) device. The level of pressure support set by the physician provides for positive airway pressure for the duration of spontaneous inspiration. Depending on the type of ventilator employed, the pressure returns to the end-expiratory pressure level when the inspiratory flow drops below a preset absolute rate or percentage of the initial inspiratory flow rate. This pressure augmentation results in reduced work of breathing, which compensates for the increased resistance from the endotracheal tube, ventilator circuit, and mechanical demand valve (which may be difficult to trigger). The total respiratory rate, tidal volume, and length of inspiration remain under the patient's control; the result is markedly increased patient comfort. Pressure support is usually used in conjunction with SIMV, which provides back-up ventilation in the event that patient-initiated efforts become inadequate (eg, excessive sedation, metabolic encephalopathy).

The disadvantages of pressure-support ventilation are that (1) the inspiratory cycle uses positive pressure and (2) the delivered tidal volume varies when changes in either airway resistance or pulmonary compliance occur.

The initial assist-pressure setting can be estimated at approximately one half the peak airway pressure. An in-line expiratory spirometer should be used to measure spontaneous breaths (5–7 mL/kg exhaled tidal volume is a reasonable goal). None of the ventilators that were sent to the Persian Gulf War in 1990 had this capability.

Inverse-Ratio Ventilation. As a patient's respiratory gas exchange becomes progressively inadequate, increasing the inspiratory-to-expiratory ratio to greater than 1 may lead to significant improvement (by elevating the mean airway pressure). The most likely mechanism for this improvement is an increase in lung volume resulting from the recruitment of atelectatic alveoli through the generation of intrinsic end expiratory pressure or so-called "auto-PEEP." Inverse-ratio ventilation may be accomplished in the pressure or volume control ventilatory modes. The pressure control variety is achieved by maintaining the set pressure limit for a fixed period of time. The volume control mode of IRV results when the inspiratory flow rate is reduced to the point whereby inspiratory time exceeds expiratory time.[39] Because this pattern of ventilation is nonphysiological, sedation is usually required for the patient.

High-Frequency Ventilation. High-frequency ventilation can be provided in three modes: high-frequency, positive-pressure ventilation; high-frequency jet ventilation; and high-frequency oscillation. The advantages of high-frequency ventilation are that the mean and peak airway pressures decrease, resulting in minimized risks of barotrauma and cardiovascular impairment. The Food and Drug Administration approves the use of high-frequency ventilation in adults for only one condition: bronchopleural fistula. The results of high-frequency ventilation in patients with ARDS have been very disappointing, but there may be some use for it in treating inhalation injury.[11]

Expiratory Positive Pressure

The application of positive pressure during exhalation, whether it occurs in the spontaneous or the mechanically ventilated patient, is called expiratory distending pressure (EDP). The two principal methods of delivery are (1) CPAP, which uses positive airway pressure throughout the spontaneous ventilatory cycle, and (2) PEEP, which uses positive pressure during the exhalation fraction of the cycle.

Expiratory distending pressure can be applied via either a flow resistor or a threshold device placed in the expiratory limb of the ventilatory circuit. The result is that expiration is retarded, prohibiting full exhalation, which, in effect, maintains alveolar distension. The flow resistor accomplishes this by changing the size of the orifice for gas flow, whereas the threshold device allows no expiratory flow until the pressure exceeds the set valve pressure. The threshold device is preferred because it can accommodate a sudden increase in expiratory pressure (eg, during coughing) without allowing a build-up of pressure in the ventilator circuit.

Continuous, Positive Airway Pressure

Large, patient-initiated decreases in airway pressure are associated with increased work of breathing. CPAP may reduce the work of breathing by giving an inspiratory assist, which leads to an almost passive inspiratory cycle. Continuous, positive airway pressure delivered via a face mask may have a limited role in the treatment of combat casualties because of the frequent occurrence of obtundation, which increases the risk for pulmonary aspiration of gastric contents. With proper patient selection, however, continuous, positive airway pressure has certain advantages: (1) the maintenance of the patient's ability to cough, (2) the maintenance of the patient's ability to warm and humidify the inspiratory gases, and (3) the avoidance of complications associated with endotracheal intubation. The primary disadvantage is gastric distension (and the concomitant increased risk of aspiration), which can be minimized by the placement of a nasogastric sump.

Clinical Use of Positive End-Expiratory Pressure

The use of PEEP merits specific attention not only because this technique is popular but also because the misconceptions regarding its use are numerous. The only valid reason for using PEEP is to increase lung volume—specifically, the FRC. The FRC is composed of the residual volume and the expiratory reserve volume. This lung capacity provides for a reservoir of oxygen-filled alveoli and permits gas exchange between inspirations. The FRC is frequently decreased following trauma or surgery (Table 25-3).

In most patients, the length of the expiratory cycle far exceeds the inspiratory. If alveoli are permitted to close during the expiratory time, then intrapulmonary shunting, resulting in hypoxemia, may occur. This premise was used to defend the "super" PEEP that was used during the 1970s, when PEEP levels greater than 20 cm H_2O were titrated until the shunt fraction was decreased to the lowest possible level.[46] No prospective study has demonstrated improved patient outcome with the use of these high levels of PEEP, leading most clinicians to use PEEP in restricted amounts (eg, < 15 cm H_2O).[47]

TABLE 25-3

CAUSES OF DECREASED FUNCTIONAL RESIDUAL CAPACITY

Cause	Effect
Residual anesthetic	Shifts the CO_2 response curve or alters central respiratory control, resulting in shallow tidal volume breathing
Abdominal distension	Compresses the diaphragm and the lower pulmonary segments
Splinting of chest-wall movements secondary to thoracic pain	Pain prevents deep breathing, which results in collapsed alveoli
Excessive airway secretions or obstruction	Work of breathing increases as the airway is blocked

INITIATING MECHANICAL VENTILATION

The initial settings that are selected for mechanical support depend on the indication for which the patient requires ventilatory support. Exhibit 25-5 is a brief guide to setting the ventilator.

Tidal Volume and Respiratory Rate

The recommended initial settings for nonrespiratory failure–ventilatory support are a tidal volume of 10 to 15 mL/kg and a respiratory rate of 7 to 10 breaths per minute, resulting in a minute ventilation of 70 to 150 mL/kg/min. The patient who is comatose or who remains under the influence of sedatives will generally be normocarbic at 80 mL/kg/min. Higher rates should be used for the initial setting when

- hypocapnic therapy is needed to treat increased intracranial pressure,
- a severe metabolic acidosis needs immediate compensation,
- a large amount of sodium bicarbonate has been administered,
- a thermal burn has created hypermetabolism, or
- pulmonary vascular occlusion has caused an increase in physiological dead space.

The selection of respiratory rate in the AC mode only determines the minimum amount of ventilation that will be ensured if the patient loses his respiratory drive. Thus, the selected setting should provide for approximately 70% to 80% of the minute ventilation required for the patient to maintain normocarbia. To obtain the maximal benefit from SIMV, once the patient is stable, the respiratory rate should be set at the minimum amount necessary to prevent respiratory acidosis.

Calculation of the tidal volume should be set on the patient's *actual* weight if the weight above the estimated lean amount is the result of obesity. Increased weight gain following fluid resuscitation does not necessitate increasing the tidal volume. Not all the set tidal volume will be delivered to the patient's alveoli because of dead space (anatomical and artificial) and volume loss (from the compliance of the ventilator circuit tubing). When the patient reaches a peak airway pressure of 40 to 60 cm H_2O during ventilation, the average amount of volume lost is 3 to 4 mL/cm H_2O pressure.[12]

Fraction of Inspired Oxygen

Many of the ventilators sent to the Persian Gulf in support of Operation Desert Storm in 1990 required an additional, external oxygen blender to provide oxygen in a relatively safe range (> room air [.21] and < 1.0). All F_{IO_2} settings—even on a sophisticated ICU ventilator—should be confirmed by an oxygen analyzer that is placed within the inspiratory limb of the patient's breathing circuit. The initial setting for F_{IO_2} will depend on whether pulmonary injury has occurred. In a postoperative patient who has sustained only an extremity or lower-abdominal wound, a slight increase in the F_{IO_2} to .40 will usually suffice. If significant pulmonary injury has occurred, then an initial F_{IO_2} of 1.0 should be selected. All attempts should be made to use the lowest concentration of oxygen that provides at least 90% oxygen saturation

EXHIBIT 25-5

MODES AND SETTINGS OF POSITIVE PRESSURE VENTILATION

Volume-Cycled Ventilators

Apnea	*Spontaneous Ventilation*
Control Mode	*Assist-Control/Synchronized, Intermittent, Mandatory Ventilation*
1. TV: 10–15 mL/kg	1. TV: 10–15 mL/kg
2. Not applicable	2. Sensitivity: –2 to –2.5 cm H_2O
3. Respiratory rate: 8–12 breaths per min	3. Respiratory rate: AC: set default rate to 6–8 breaths per min in case of apnea SIMV: to rest the patient, ensure that 80% of breaths are mechanically provided
4. Not applicable	4. PF: 3.5–4.0 • minute ventilation

5. Expiratory time should exceed inspiratory time by 2:1
6. Keep PAP < 30–40 cm H_2O
7. Add PEEP in 2- to 4-cm H_2O increments if hypoxemia persists on F_{IO_2} > 50%
8. Set PL at 15–20 cm H_2O above PAP during a normal breath or < 80 cm H_2O

Pressure-Cycled Ventilators

1. TV: starting at 20 cm H_2O, increase peak pressure until tidal volume is adequate by clinical examination or arterial blood gas criteria
2. Respiratory rate: provides for a minimum amount if patient becomes apneic. Set to provide 70%–80% of minute ventilation
3. F_{IO_2}: an in-line O_2 analyzer is strongly recommended
4. Patient interaction: the spontaneously breathing patient may not tolerate this type of ventilator, necessitating either weaning or changing to a different mode of ventilation

Time-Cycled Ventilators

1. TV: set the inspiratory time and PF to provide the calculated tidal volume
2. Respiratory rate: ensures a minimal amount of ventilation
3. As the PF determines the TV, it is necessary to check the inspiratory-to-expiratory time ratio with each rate change

AC: assist-control ventilation; F_{IO_2}: fraction of inspired oxygen; PAP: peak airway pressure; PEEP: positive end-expiratory pressure; PF: peak flow; PL: pressure limit; SIMV: synchronized,intermittent, mandatory ventilation; TV: tidal volume

of the arterial hemoglobin. High oxygen concentrations are toxic to the lungs and may result in numerous complications. These include a deterioration in ventilation–perfusion matching, absorption atelectasis, decreased mucociliary clearance, and ARDS.[48] Decreasing the F_{IO_2}, utilizing a pulse oximeter as a monitor, may be safely done in increments of 5% to 10% every 5 minutes.

Inspiratory Flow Rate

The peak inspiratory flow rate is particularly important. If it is set too high, ventilation is maldistributed throughout the lungs. If set too low, the expiratory time may be shortened, creating a problem for patients with obstructive airway disease. In all cases, the peak inspiratory flow rate

must exceed the patient's intrinsic demand if further respiratory work by the patient is to be avoided. A setting that is 3.5- to 4-fold greater than the patient's minute volume requirement is usually adequate. When increasing the peak flow rate, the physician will generally observe a rise in the peak airway pressure. Occasionally, the peak airway pressure will decrease, signifying that intrinsic (or auto-) PEEP is occurring. This will resolve with lengthening of the expiratory time.

Additional Settings

The remainder of the mechanical ventilator settings are often overlooked by medical officers but will be set by a respiratory therapist. These include the inspiratory-to-expiratory ratio, the selection of an inspiratory waveform (square, ramp, sine), and a variety of alarms. The most important alarm, which is accompanied by a release mechanism, is the peak airway pressure. Initially, this should be set for approximately 15 to 20 cm H_2O higher than the peak pressure measured during unlabored, mechanical breathing. These additional settings are important for the intensivist when a patient is particularly difficult to ventilate.

Stabilizing the Patient in Acute Respiratory Failure

The approach to initiating mechanical support in the patient in acute respiratory failure differs from the previous discussion. The initial settings recommended above are merely a guideline for initiating mechanical support. As a result of either hypermetabolism or increased dead-space ventilation, a combat casualty's minute ventilation requirement may be as high as 25 to 30 L/min. Respiratory muscle fatigue appears to be a common component of acute respiratory failure regardless of the etiology.[49] The initial goal is to ensure a good quality of rest and recovery. Using a constant tidal volume of 10 to 15 mL/kg, the respiratory rate should be increased until the patient is comfortable and rested. When these high levels of mechanical support are required, bedside supervision by an intensivist is necessary to avoid (*a*) excessive elevation of airway pressure and (*b*) air-trapping, which results from an inadequate expiratory time. Although the AC mode may allow the greatest decrease in the patient's work of breathing, a very tachypneic or anxious patient may cause excessive triggering and stacking of ventilations. Occasionally, the patient may need to be sedated and paralyzed so that hemodynamic compromise can be minimized and the elimination of carbon dioxide can be improved.

Patients who do not respond initially to treatment for sepsis or shock should be considered for early intubation and mechanical ventilatory support. Considerable oxygen can be consumed by the diaphragm alone (as much as 30%–40% of the body's total amount) in the state of shock or acute respiratory failure.[50]

Following a period of stabilization, a progressive amount of work should be assumed by the patient to prevent atrophy and promote conditioning.[51]

MONITORING THE EFFECTIVENESS OF VENTILATION AND OXYGENATION

Monitors used in patients with acute respiratory failure may be characterized as invasive or noninvasive, and intermittent or continuous. A monitor may either follow only the progression of disease or give concomitant information regarding the effect of therapy.

Oxygen Concentration

The F_{IO_2} delivered to the patient is one of the most important and easiest parameters to measure. Once the F_{IO_2} is known, other indices—all of which serve as markers for the severity of pulmonary dysfunction—can be calculated:

- the difference between the partial pressure of oxygen within the alveoli and that present within the arterial blood ($P_{AO_2} - Pa_{O_2}$); normal is 5 to 10 mm Hg;
- the ratio of arterial to alveolar partial pressure of oxygen (Pa_{O_2}/P_{AO_2}); normal is 0.75; and
- the ratio of arterial partial pressure of oxygen to the fraction of inspired oxygen (Pa_{O_2}/F_{IO_2}); normal is 250 mm Hg.

Pulse Oximetry

The pulse oximeter is a continuous monitor that detects the percentage of total hemoglobin saturated with oxygen. This is achieved by using a photoelectric tube to measure the amount of light transmitted at two different wavelengths. Only the arterial component of the blood in the tissue is analyzed. This is done by simultaneously measuring pulsatile flow and subtracting the background absorption, which is the nonpulsatile venous and capillary blood. Because of a different stereochemistry of hemoglobin molecules, saturated and un-

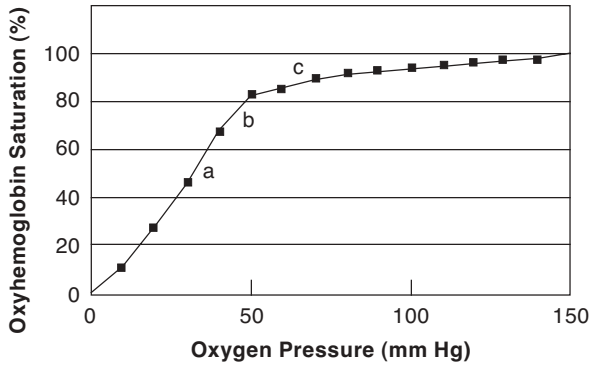

Fig. 25-2. The sigmoid shape of the oxyhemoglobin dissociation curve, with its nonlinear relation between the partial pressure of oxygen dissolved in blood and the percentage of oxygen saturation of hemoglobin, must be borne in mind when interpreting blood gas data. The shape and position of the curve can be estimated in terms of these three points, which fix the relation between partial pressure and saturation: (**a**) the oxygen pressure at which hemoglobin is 50% saturated (P_{50}), (**b**) the oxygen pressure of mixed venous blood, which is normally 75% saturated ($S_{mv}O_2$), and (**c**) the partial pressure at which hemoglobin is 90% saturated.

saturated hemoglobin display unique absorption spectrums. To use this information clinically, the physician must know the three key points on the oxyhemoglobin dissociation curve (Figure 25-2):

a. the P_{50}, the P_{O_2} at which 50% of the hemoglobin molecules are saturated, which normally occurs at 27 mm Hg;
b. the mixed venous saturation ($S_{mv}O_2$) (75%), corresponding to a P_{O_2} of 35 to 40 mm Hg; and
c. the "knee" of the curve at 90% saturation of hemoglobin molecules, at a P_{O_2} of approximately 60 mm Hg.

This technology has several limitations: pulse oximetry (*a*) does not compute carboxyhemoglobin or methemoglobin as desaturated hemoglobin, (*b*) is inaccurate in instances of severe anemia ($\leq$ 7 g/dL), and (*c*) does not measure venous oxygen saturation because venous blood is only slightly pulsatile. The latter situation may occur when large amounts of PEEP are applied. One important point is that the patient's P_{O_2} may decrease from 400 to 100 mm Hg, representing a significant change in pulmonary function, yet the pulse oximeter saturation display does not change.

The Persian Gulf War was the first conflict in which pulse oximeters were widely available for patient monitoring. Oximeters were present at all anesthetizing locations, within the ICUs, and in the emergency departments.

Gas-Exchange Indices

Partial Pressure of Arterial Oxygen

Factors other than the gas-exchange capability and efficiency of the lung may affect the Pa_{O_2}. Two clinical situations that exemplify this point are (1) shock, during which the venous P_{O_2} is markedly reduced, and (2) a state of increased V_{O_2}. The desaturating effect of venous hypoxemia is exacerbated in the presence of lung pathology, contributing to intrapulmonary shunting. Hence, when hypoxemia (low P_{O_2}) occurs, it is not a correct assumption that the patient's clinical problem is one of strictly pulmonary pathology. Conversely, if the P_{O_2} is completely normal for a given F_{IO_2}, the lungs are assumed to be functioning normally. The P_{O_2}, when measured by an arterial blood-gas determination, is the accepted standard for measuring oxygenation.

Arterial blood gases as a monitor of oxygenation have the limitation of reflecting the clinical state at only one moment. Furthermore, the Pa_{O_2} measures only the oxygen dissolved in the blood, which is only a small fraction of the total amount of oxygen delivered to the tissues. Most of the arterial oxygen content is that portion of oxygen bound to hemoglobin. However, the Pa_{O_2} is the major determinant of the position on the oxygen–hemoglobin dissociation curve and thus determines the arterial hemoglobin oxygen saturation.

Alveolar–Arterial Oxygen Tension Difference

The difference between the partial pressure of oxygen in the alveoli and in the arteries (P_{AO_2} – Pa_{O_2}) is a specific indicator of pulmonary pathology, but this index has a significant practical limitation: the clinician must know the F_{IO_2}. Multiple determinations of the calculated value for P_{AO_2} – Pa_{O_2} are accurate predictors of pulmonary injury *only* when the F_{IO_2} has remained constant. P_{AO_2} – Pa_{O_2} is calculated using the following equation:

$$P_{AO_2} - Pa_{O_2} = F_{IO_2}(P_B - P_{H_2O}) - P_{CO_2}/R.Q.$$

where P_B represents barometric pressure (760 mm Hg at sea level); P_{H_2O} represents partial pressure of fully saturated water vapor (47 mm Hg); and *R.Q.* represents the respiratory quotient, assumed to be 0.8 with a normal diet. The normal value for P_{AO_2} –

Pa_{O_2} is less than 10 mm Hg for a healthy, young adult.

The Ratio of Arterial to Alveolar Oxygen Tension

The arterial-to-alveolar oxygen tension ratio (Pa_{O_2}/P_{AO_2}) remains more stable with alteration in the F_{IO_2}, and this calculation can be employed to predict the expected Pa_{O_2} when the F_{IO_2} is altered. The normal value is 0.75.

The Ratio of Arterial Oxygen to Inspired Oxygen

The arterial-to-inspired oxygen ratio (Pa_{O_2}/F_{IO_2}) is the simplest index to calculate, as it does not require use of the alveolar gas equation. The normal value is greater than 250 mm Hg.

Right-to-Left Shunting

Right-to-left intrapulmonary shunting of blood leads most of the hypoxemia seen with severe respiratory failure. The shunt equation applicable to severe respiratory failure is

$$Qs/Qt = Cc_{O_2} - Ca_{O_2}/Cc_{O_2} - Cv_{O_2}$$

where *Qs* represents the flow to the shunt, *Qt* represents the total pulmonary blood flow, and Cc_{O_2} represents the oxygen content of pulmonary capillary blood, which equals (1.34 mL O_2/g Hb) • (Hb g/dL) • (% S_{O_2}). The pulmonary capillary blood is assumed to be fully saturated ($S_{O_2} = 100\%$). Ca_{O_2} represents the arterial oxygen content and Cv_{O_2} represents the pulmonary venous oxygen content whose hemoglobin oxygen saturations can be directly measured. This equation discounts the small contribution of dissolved oxygen in computing the various oxygen contents.

If $F_{IO_2} = 1.0$, then the following simplified shunt equation may be used:

$$Qs/Qt = P(A - a)_{O_2}/20$$

Monitoring Mechanical Ventilation

The importance of bedside patient observation cannot be overemphasized. Much information is gained by attentively watching the patient's spontaneous ventilatory efforts and use and coordination of the thoracoabdominal muscles of inspiration. In addition to the more-specific pulmonary pathologies, tachypnea is an early sign of shock or sepsis.

Airway Pressures

The peak and plateau airway pressures are monitored in all patients in respiratory failure who are supported by mechanical ventilation. From these measurements the dynamic and static compliance may be calculated as follows:

$$Cd = \text{change in volume}/(PAP - PEEP)$$

$$Cs = \text{change in volume}/(PLP - PEEP)$$

where *Cd* represents dynamic compliance, *Cs* represents static compliance, *change in volume* represents ventilator tidal volume, *PAP* represents peak airway pressure, *PEEP* represents positive end-expiratory pressure, and *PLP* represents plateau pressure. Actual tidal volume should be corrected by subtraction of the volume lost for circuit expansion (3–4 mL/cm H_2O peak airway pressure).

A single, discrete value cannot be given be given for the dynamic compliance, which is flow dependent. Static compliance has the range 75 to 125 mL/cm H_2O. The normal values are position dependent, and, for supine measurements, the normal value should be reduced approximately 30%.

The measurement of dynamic compliance is affected by the airway resistance, the compliance of the pulmonary parenchyma, and the compliance of the chest wall. The difference in peak and plateau pressures (normal = < 10 cm H_2O) indicates the difference between these two compliances and is independent of airway resistance.[52] If this difference is large, then therapeutic intervention should be directed toward minimizing or eliminating the contributing factors (Exhibit 25-6). Bronchospasm is a common cause of increased airway resistance during mechanical ventilation. Stan-

EXHIBIT 25-6

FACTORS CONTRIBUTING TO INCREASED AIRWAY RESISTANCE

Airway secretions
Kinked endotracheal tube
Narrow internal diameter of the endotracheal tube
Condensed water in the ventilator tubing
Bronchospasm

dard medical therapy includes inhaled beta agonists, inhaled anticholinergic agents, theophylline, and steroids.

If both the peak and plateau airway pressures are elevated and there is minimal difference between the dynamic and static compliances, then the compliance of either the pulmonary parenchyma or the chest wall has been reduced. Most commonly, this is the result of pulmonary injury (Exhibit 25-7). All medical officers caring for patients on mechanical ventilation must remember that the peak airway pressure is not infrequently elevated as a result of the selection of ventilatory settings (peak inspiratory flow, tidal volume, and respiratory rate), an example of iatrogenic noncompliance.

Capnography

Whenever possible, end-tidal carbon dioxide ($ETCO_2$) monitoring (capnography) should be used in every patient on mechanical ventilation. The initial widespread clinical use of capnography was to confirm the correct placement of an endotracheal tube. If direct visualization and placement of an endotracheal tube between the vocal cords is not achieved, capnography is the only other absolute confirmation of correct placement. A malpositioned endotracheal tube is a greater problem in a critically ill patient in the ICU than in an anesthetized, paralyzed patient in the operating room. Patients in the ICU are obviously intubated for longer periods of time, are more mobile, more interactive, often more agitated, and are not continuously observed at the bedside, compared to patients in the operating room.

EXHIBIT 25-7

CLINICAL CAUSES OF REDUCED PULMONARY COMPLIANCE

- Pulmonary edema
- Pneumonia
- Atelectasis
- Pulmonary fibrosis
- Pneumothorax or hemothorax
- Pulmonary aspiration
- Chemical pneumonitis

Self-extubation is a recognized complication in all ICUs, with an estimated occurrence of 11% to 13%.[53] In addition, because of patient movement and respiratory therapies, disconnections in the airway circuitry (ventilator tubing, humidifier, nebulizer) occur frequently. Capnography provides immediate detection of airway obstruction, extubation, or ventilator disconnection. The presence of a regularly occurring exhaled carbon dioxide waveform confirms the continued presence of an intratracheal tube. The use of pulse oximetry is not a replacement for monitoring these events, as pulmonary oxygen reserve may allow sufficient gas exchange for several minutes.

An idealized normal capnogram is shown in Figure 25-3. In contrast to pulse oximetry, monitoring of exhaled carbon dioxide can provide more information than just the $ETCO_2$ (recorded in mm Hg or % CO_2). From the shape of the waveform, we can infer several observations such as the adequacy of alveolar emptying and the magnitude of pulmonary dead space. A well-defined plateau tells us that alveolar emptying is adequate and pulmonary dead space is minimal; therefore, $ETCO_2$ closely matches $PaCO_2$.

The difference between $PaCO_2$ and $ETCO_2$ is normally 4 to 7 mm Hg for most monitors currently available. An increase in this gradient in a patient on mechanical ventilation suggests the further development of physiological dead space. Exhibit 25-8 lists frequent clinical causes of increased dead space ventilation.

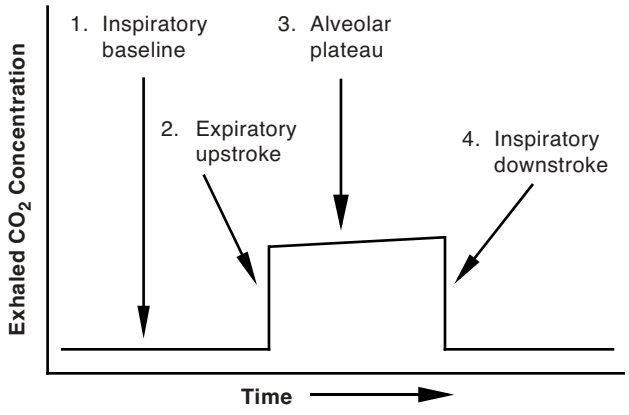

Fig. 25-3. Idealized capnogram waveform, with time plotted on the x axis and exhaled carbon dioxide concentration on the y axis. The numbered segments indicate the four phases of the ventilatory cycle. Adapted with permission from Benumof JL. *Anesthesia for Thoracic Surgery*. 2nd ed. Philadelphia, Pa: WB Saunders; 1995: 244.

Analysis of the carbon dioxide waveform also provides information regarding bronchospasm (the slope of the ascending segment) and the competency of the ventilator expiratory valve (baseline rebreathing of CO_2 > 2–4 mm Hg). One study[54] indicated that a rise or reappearance of exhaled carbon dioxide is one of the first indicators of the return of spontaneous circulation following a cardiopulmonary arrest. Additional studies have demonstrated that $ETCO_2$ monitoring may be a good indicator of both the adequacy of pulmonary blood flow and cardiac output, and also a good prognostic indicator of the success of cardiopulmonary resuscitation.[55]

Exhaled Volume Monitoring (Spirometry)

It is crucial that exhaled tidal or minute volume be monitored while mechanical ventilation is used. Several of the ventilators that were sent to the Persian Gulf War—including the Univent 750, Bear 33, Lifecare PLV-100 and Lifecare PLV-102—did not have built-in exhaled volume monitoring. Monitoring of exhaled volume is particularly important when using either a pressure-cycled or a time-cycled mechanical ventilator. The use of these ventilators does not ensure the maintenance of a constant minute ventilation and a stable PCO_2 level. Even during the use of volume-cycled ventilation, measurement of exhaled volume serves as an additional monitor warning of a disconnection, a malfunctioning inspiratory or expiratory valve, an endotracheal or tracheostomy cuff leak, or a marked change in the pulmonary compliance (eg, pneumothorax).

EXHIBIT 25-8

FACTORS CONTRIBUTING TO INCREASED DEAD SPACE

Pulmonary
- Bronchospasm
- Chronic obstructive pulmonary disease
- Excessive positive end-expiratory pressure

Circulatory
- Hypovolemia
- Hypotension
- Reduced cardiac output
- Pulmonary embolism (thrombus, air, fat)

Clinical Examination of the Patient–Ventilator Unit

The clinical examination of the patient receiving mechanical ventilation (ie, the patient–ventilator *unit*) is the most important "monitor" of all. The first observation to make is determining whether the patient is ventilating spontaneously. If the patient demonstrates a lack of ventilatory effort, it usually reflects one or more of the criteria discussed in Exhibit 25-9. If the patient is making spontaneous ventilatory efforts, then ensure that the ventilator is not in the CMV mode, as this setting does not allow for ventilation beyond that designated by the set rate.

It is equally important to evaluate how well the mechanical support is meeting the patient's ventilatory requirements. If purpose of mechanical ventilation is to better meet the patient's ventilation and oxygenation needs, then allowing the patient to perform an excessive amount of the work of breathing defeats the purpose of mechanical support. The amount of minute ventilation required to maintain normocarbia can provide useful information as to the amount of dead space ventilation and the overall metabolic state in the setting of trauma and serious infection.

The level of peak airway pressure is a major risk factor for the development of barotrauma. For this reason, should the airway pressures be increased or noted to be rising, it is prudent to evaluate the patient for reversible causes of airway pressure elevation.

Lastly, should the patient demonstrate agitation while receiving mechanical ventilation, one should always first assume that the problem rests not with the patient, but rather with the ventilator itself or the manner in which it is being employed. By fully evaluating the ventilator and assuring that there are no malfunctions or inappropriate ventilatory settings, the clinician can avoid a potential catastrophe.

Oxygen Delivery

The gas exchange capability of the lung, as assessed by the PaO_2, $AaDO_2$, or other indices, is only one of the components that determines DO_2 to tissues. The two other major components are the level of hemoglobin and the cardiac output. (The comments in this chapter are limited to respiratory insufficiency; the reader can find a more detailed discussion in Chapter 24, The Syndromes of Systemic Inflammatory Response and Multiple Organ Dysfunction.) However, a number of studies[56–59] give credence to the importance of the calculation of both DO_2 and VO_2 by use of a pulmonary artery

EXHIBIT 25-9

CRITERIA FOR OPTIMAL VENTILATOR USE

1. **Is the patient ventilating spontaneously?**

   If ***NO***, then

   a. The settings (TV, rate) for mechanical support exceed the patient's requirement.
   b. The patient's apneic threshold has been altered (eg, narcotics, benzodiazepines).
   c. pH compensation is needed for a severe metabolic alkalosis.
   d. The patient does not have an intact central ventilatory drive (eg, due to a closed head injury or coma).
   e. The patient's thoracic or diaphragmatic muscular function is inadequate (eg, due to nerve agents, extreme fatigue, malnutrition).

   If ***YES***, then

   a. Change from control mode to either AC or SIMV, and
   b. Evaluate how hard the patient is working while maintaining this spontaneous ventilation (eg, respiratory rate, diaphoresis, pulse, mental status).

2. **What is the minute ventilation requirement?**

   a. The requirement will vary frequently in the same patient based on the physical demands and the therapies being performed.
   b. The daily trend in mechanical ventilation provides information as to overall patient status in sepsis and trauma.

3. **Is the peak airway pressure too high (ie, progressively increasing or > 50 cm H_2O)?**

   If ***YES***, then

   a. Evaluate dynamic (TV/PAP – PEEP) and static (TV/plateau – PEEP) compliances.
   b. Evaluate whether bronchospasm or secretions are causing increased airway resistance.

4. **Is the patient fighting the ventilator?**

   If ***YES***, then evaluate for the following:

   a. Secretions and the need for suctioning,
   b. Bronchospasm, and
   c. Stacking of breaths or "auto-PEEP" because of too short an expiratory time or too high a sensitivity setting.

Remember: the patient is "fighting" for his life. *Do not sedate and paralyze the patient* until you are confident that the ventilator is not malfunctioning and that the settings chosen are appropriate for the patient's needs.

TV: tidal volume; AC: assist control; SIMV: synchronized, intermittent, mandatory ventilation; PAP: peak airway pressure; PEEP: positive end-expiratory pressure

catheter. Several clinical states including sepsis and ARDS are characterized by what appears to be a pathological oxygen-supply dependency. Systemic oxygen need may reach levels as high as 21 mL/kg/min.[60] (Arterial and mixed-venous oxygen contents and the cardiac index must be measured simultaneously to allow these calculations.)

The usefulness of augmenting cardiac output has been difficult to demonstrate in several clinical trials. In both the surgical setting and the setting of myocardial infarction,[61] pushing cardiac output with inotropic drugs has not been associated with improved outcome. These studies are difficult to perform, since critically ill patients are sometimes hard to match—but meta-analysis and further clinical trials will elucidate the true effects of artifically increasing oxygen delivery. At this time, the question remains only partially answered and is the source of considerable controversy among practitioners across the country.[62,63]

Chest Radiography

Daily chest X-ray examinations are an accepted ICU monitor for patients in respiratory failure. Several studies have validated the importance of daily chest X-ray examinations in critically ill unstable or mechanically ventilated patients: approximately 14% to 15% of chest X-ray examinations demonstrate unexpected findings that lead to a change in management.[64,65]

Most chest X-ray examinations taken of critically ill patients are made by a portable device at the bedside. These films are taken from an anterior-posterior rather than the standard posteroanterior orientation. This is important for several reasons. The distance from the X-ray beam to the intrathoracic structures is not constant, and objects that are far from the film (cardiac silhouette) are magnified. Critically ill patients, particularly those in respiratory failure, are often tachypneic, and obtaining an end-inspiratory chest film is difficult because they cannot hold their breath. It should be remembered that a chest X-ray examination cannot properly evaluate upper-airway obstruction; however, careful attention not infrequently reveals tracheal abnormalities. One example of this is tracheal or left main bronchus deviation when a traumatic aortic dissection occurs. The information that can be obtained from a chest X-ray examination depends on the respiratory status of the casualty: whether the patient's ventilations are spontaneous or mechanically assisted.

Spontaneously Ventilating Patients. Chest radiography is used to evaluate the patient for pulmonary parenchymal injury. The presence of a radiological density may represent collapse of alveoli (eg, atelectasis); or alveolar consolidation from water (pulmonary edema), blood (aspiration or bronchoalveolar hemorrhage); or infected secretions (pneumonia). These fluids are indistinguishable radiologically, and clinical examination or other diagnostic tests must be conducted to discern the actual cause. The chest X-ray examination may also detect pathology in the pleural space (eg, pneumothorax, hemothorax, or effusion). Frequently, chest X-ray examinations done in this patient population are performed in the supine position, and a pneumothorax may be missed. One helpful radiological sign is the "deep sulcus sign," a clear costophrenic angle that often extends well below the normal location for the diaphragm.

Additionally, radiological assessment of the heart, great vessels, and pulmonary vasculature offers information pertinent to the patient's intravascular volume status, the presence of congestive heart failure, or the possible occurrence of a pulmonary embolism. The latter should be suspected when a pleura-based, wedge-shaped infiltrate (Hampton's hump) or regional oligemia (Westermark's sign) are seen on a chest roentgenogram.

Mechanically Ventilated Patients. Patients who require mechanical ventilation may benefit from additional radiographic monitoring for potential complications. A daily chest X-ray examination is a good method to use for confirming a safe location for an endotracheal tube. Although other monitors (such as capnography) exist to confirm intratracheal placement, a chest X-ray examination will reveal the exact location of the cuff and the tip of the endotracheal tube. To prevent either intubation of the right main bronchus or accidental extubation, the optimal distance from the carina to the tip of the endotracheal tube is 3 to 7 cm. The carina is used as a reference point because the relationship of the endotracheal tube tip to the clavicle changes, depending on the angle at which the film was exposed. Tracheostomy tubes are much more secure, and less frequent radiological monitoring is required.

Mechanical ventilation may be associated with barotrauma. This is more common when peak airway pressures exceeding 40 to 50 cm H_2O have been employed. Barotrauma may take several forms including pneumothorax, pneumomediastinum, pneumoperitoneum; or subcutaneous emphysema of the chest wall, neck, or even the eyes. Patients in respiratory failure who require high levels of ventilatory support, particularly once evidence of barotrauma is present, need frequent, routine chest X-ray examinations, as well as an additional one whenever a clinical deterioration in either hemodynamic or respiratory status occurs. A small pneumothorax in a patient being supported with positive-pressure ventilation may at any time become a tension pneumothorax with the accompanying hemodynamic compromise.

Hemodynamic Monitoring

Although arterial, central venous, and pulmonary artery–catheter monitoring are important in monitoring the critically ill patient, a full discussion of hemodynamic monitoring is beyond the scope of this chapter. Further information may be found in Chapter 5, Physiological Monitoring.

WEANING FROM MECHANICAL VENTILATION

Weaning the patient from mechanical ventilation refers to shifting to partial ventilatory support while observing the patient's response to the increased work of breathing associated with spontaneous ventilation. Weaning is frequently unnecessary in patients who are mechanically supported for a brief period of time following surgery or another medical illness and who have not suffered from malnutrition. These patients should not have developed respiratory muscle fatigue.

Discontinuation of mechanical ventilation should be considered only if the underlying cause of the respiratory failure (eg, treated pneumonia, resolved sepsis, arousal from coma) has been reversed. Discontinuation is likely to prove possible only if the following preconditions have been met:

- the central respiratory drive is intact, ensuring normocarbia;
- muscular strength is adequate to maintain a clear airway and provide for full spontaneous breathing;
- the patient is hemodynamically stable (ie, shock is resolved, intravascular volume restored); and
- carbon dioxide production is not elevated.

Medical officers, however, must consider not only the casualty's condition but also the need for aeromedical evacuation to a higher echelon of care. The U.S. Air Force is reluctant to evacuate casualties on ventilators and, in fact, refused to do so during the Persian Gulf War. (See Chapter 27, Military Medical Evacuation, for a more complete discussion of this subject.)

Many criteria have been published to evaluate a patient's ability to be successfully weaned from mechanical ventilatory support (Exhibit 25-10). Some of these criteria are subjective and thus require good clinical acumen as to the physiological capability of the patient. Even objective criteria that can be measured and quantified have been questioned, as there are patients who do successfully wean from mechanical support despite their not meeting the weaning criteria.[66,67] In addition, certain of the quantitative measurements are frequently not performed properly and thus may give false information. Nevertheless, combining criteria into a decision tree (Figure 25-4) is usually helpful in deciding if and when to wean. Application of the decision tree presupposes that the patient is not sedated to the point where the ventilatory drive is blunted.

Several of these indicators (negative inspiratory force, minute ventilation, and maximum voluntary ventilation) were evaluated in one study.[68] If the minute ventilation was less than 10 L, then the maximum voluntary ventilation was 2-fold greater than the minute volume, and the negative inspiratory force was greater than –30 cm H_2O. All patients were successfully extubated in this study. These parameters are probably of greater use in short-term ventilator use than they are in prolonged, when the clinical judgment of the physician is as valid as the measured criteria.

Patients who required prolonged (> 30 d) mechanical ventilation have been shown to have a hospital mortality identical to patients who required only short-term mechanical ventilation.[69] Thus, these patients deserve a well-conceived plan for the goal of successful extubation and separation from mechanical ventilatory support.

EXHIBIT 25-10

CRITERIA FOR WEANING FROM VENTILATORS

Respiratory or Metabolic Demand or Both

- Respiratory rate < 25
- Minute ventilation < 10 L/min
- Dead space ventilation < 30%–40% of total ventilation

Oxygenation

- Pa_{O_2} > 60 mm Hg on F_{IO_2} < 50%
- PEEP < 5 cm H_2O
- Intrapulmonary shunt < 30%
- Adequate hemoglobin concentration (7–10 g/dL)

Respiratory Muscle Strength

- Tidal volume > 5 mL/kg
- Vital capacity > 10 mL/kg
- Negative inspiratory force > –30 cm H_2O
- Maximal voluntary ventilation > 2-fold the resting minute ventilation

Pa_{O_2}: partial pressure of arterial oxygen; PEEP: positive end-expiratory pressure

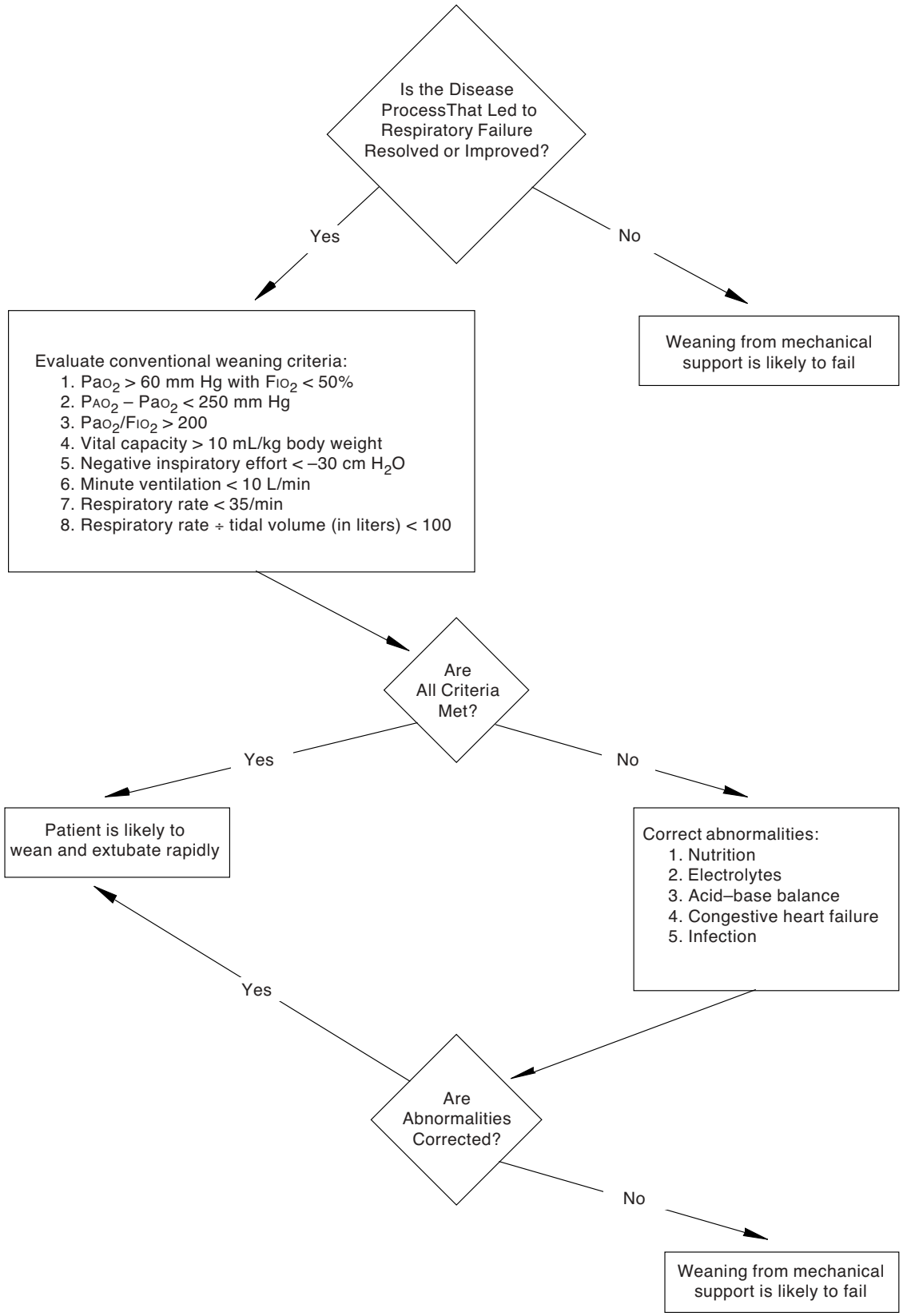

Fig. 25-4. Decision tree for weaning from mechanical ventilation.

The first step in weaning a patient who has been dependent on mechanical ventilatory support is to optimize the patient's general status (Exhibit 25-11). This may seem obvious, but it is not fully appreciated and is frequently overlooked completely—especially when too much attention is directed toward purely respiratory indices. Even if the patient demonstrates no respiratory distress, it is unwise to wean a patient who appears to be in any form of shock. By definition, the term "shock" means that the supply of blood and nutrients (oxygen and glucose) to organs or tissues has not met the demand. If such a patient were required to sustain full spontaneous ventilation, the increase in oxygen demand could worsen the balance between systemic oxygen delivery and systemic oxygen need. For example, in sepsis, oxygen utilization by the diaphragm can be very high and, in fact, can constitute nearly 30% to 40% of the body's total V_{O_2}.[50]

Several clinical conditions may lead to an elevated production of carbon dioxide, necessitating a higher minute ventilation to maintain normocarbia. Patients who are febrile, agitated, in pain, or who have suffered traumatic injuries, thermal burns, or seizures produce increased carbon dioxide. Excessive carbon dioxide production may also arise iatrogenically as a result of excessive carbohydrate administration. (For a more complete discussion, see Chapter 23, Metabolic Derangements and Nutritional Support.)

It is often impossible to determine the exact cause of a patient's failure to wean, but recent discussion has focused on respiratory muscle fatigue and disuse atrophy as two important causes. It may be clinically impossible to distinguish between these two causes. Respiratory muscle fatigue has been suspected clinically for some time and electromyographic evidence supports this concern.[70] The clinician must remain alert to mechanical factors that exacerbate rather than aid respiratory muscle fatigue. Most intensivists know that increased airway resistance results from using a small endotracheal tube ($\leq$ 7.0 mm internal diameter). A tracheostomy, by increasing the diameter and shortening the length of the airway, may reduce the resistance. Occasional patients may wean best by simply proceeding to extubation, totally eliminating the resistance of the artificial airway. (This is becoming less necessary as advanced mechanical ventilators with multiple ventilatory modes are developed.)

The three principal weaning modalities are (1) a progressive reduction of mechanical support through the use of IMV, (2) T-piece trials, whereby the patient is disconnected from the ventilator and attached to a T-piece for progressively longer intervals, and (3) pressure-support weaning. The literature does not support the superiority of any one method.[67]

T-piece trials should be initiated in 5-minute intervals, with the physician or nurse at the bedside to evaluate any significant changes in the patient's respiratory rate, pulse, or subjective complaints of excessive work. T-piece trials use intermittent periods of vigorous breathing intended to increase the patient's respiratory endurance. Rest periods are

EXHIBIT 25-11

FACTORS THAT MAY PREVENT DISCONTINUATION OF MECHANICAL VENTILATION

The patient must not be malnourished. Diaphragmatic muscle loss is nearly linearly related to the degree of malnutrition.[1,2]

Significant acid–base and electrolyte abnormalities must be corrected, including metabolic alkalosis that demands hypoventilation to normalize the serum pH.

Hypophosphatemia may also contribute to respiratory muscular weakness.

Left-ventricular failure leading to reduced pulmonary compliance may be an occult cause of persistent failure to wean from mechanical support.

Airway secretions may be a significant problem to the patient after extubation, during which time small-airway obstruction leads to atelectasis and bronchospasm.

Sources: (1) Bell RM, Bynoe RP. Nutrition and respiration. *Prob Crit Care*. 1987;1(3):413–434. (2) Lewis MI, Belman MJ. Nutrition and the respiratory muscles. *Clin Chest Med*. 1988;9(2):337–348.

important to the success of this method. The definition of quality respiratory rest (ie, the respiratory muscles are resting) has been clouded by recent studies showing significant amounts of respiratory work with AC ventilation. In addition, the optimal rest time between periods of T-piece work has not been answered in the literature. Our practice has been to allow for a minimum of one hour between exercise periods and rest the patient with full ventilatory support overnight.

SIMV is another ventilatory strategy commonly used in weaning. Quite simply, the number of mechanical breaths per minute provided to the patient are gradually reduced over several days to weeks. As the number of mechanical breaths is reduced, the patient must assume a greater amount of the work of breathing. The potential disadvantage of this strategy is the increase in work of breathing due to the resistance inherent in the inspiratory demand valves used in modern mechanical ventilators.[71] This work may be sufficient to cause the weaning attempt to fail in some patients (eg, those patients with severe obstructive lung disease or significant respiratory muscle atrophy).

Another relatively new strategy for weaning has been the use of pressure support ventilation, either in conjunction with or in place of SIMV. Pressure support ventilation permits the mechanical flow of fresh gas after the patient initiates a breath. Because the patient retains control of the total respiratory rate and the depth and duration of assisted breaths, they are generally more comfortable while receiving mechanical ventilatory support. This mode offsets the increased work of breathing inherent in the ventilator circuit due to resistance to gas flow in the circuit. Most authorities do not recommend reducing the SIMV rate below four breaths per minute unless pressure support ventilation is utilized concomitantly.

Several general comments should be made regarding medical plans for ventilatory weaning:

- Adequate rest periods are essential to prevent respiratory muscle fatigue.
- The physician must constantly assess the patient's progress during weaning.
- Arterial blood gas measurements are no substitute for clinical judgment.
- Hypercarbia is a late finding of inadequate respiratory muscle strength.
- The patient must be observed for tachypnea, paradoxical respiratory movements, use of accessory muscles of inspiration, and diaphoresis.

VENTILATORS AVAILABLE FOR COMBAT CASUALTY CARE

A large number of mechanical ventilators are in the inventories of the medical departments of the U.S. armed forces. The following section discusses the Puritan-Bennett PR-2 (manufactured by the Bennett Medical Equipment, Los Angeles, Calif.), which is pressure cycled; the Bear 33 (manufactured by Bear Medical Systems, Riverside, Calif.) and the Lifecare PLV-100 and PLV-102 (manufactured by Lifecare Corporation, Lafayette, Colo.), which are volume cycled; the Bennett MA-1 (manufactured by Bennett Medical Equipment, Los Angeles, Calif.), which is also volume cycled; and the Impact Uni-Vent Model 750 M (manufactured by Impact Instrumentation, West Caldwell, N.J.), which is time cycled.

These ventilators are available for use with deployed medical units.

Puritan-Bennett PR-2

The Puritan-Bennett PR-2 is basically a converted intermittent, positive-pressure breathing machine and requires a source of compressed gas to function. The PR-2 is unique among the ventilators under discussion here because it is pressure cycled. The patient's tidal volume cannot be directly set, but is a result of the pressure limit and the inspiratory time. The limitations of the PR-2 in providing mechanical ventilatory support to combat casualties are as follows:

- If the patient's compliance decreases or airway resistance increases (a change in the patient's position, secretions in the airway, bronchospasm, or patient agitation), the same settings of airway pressure will result in a decreased tidal volume and hypoventilation. Thus, we strongly advise that (*a*) exhaled tidal volume be monitored and (*b*) an alarm be attached.
- The patient may continue to spontaneously breathe; however, competition may occur as there is no mechanism to provide synchronization with the patient's efforts. This may result in high airway pressures and barotrauma.

- There is inaccurate control of F_{IO_2} from breath to breath when oxygen enrichment is used. The higher the patient's minute ventilation, the lower the F_{IO_2}.
- When the respiratory rate increases and the pressure is kept constant, inspiration may end prematurely because the constant flow rate results in higher peak airway pressures closing the inspiratory valve.
- To provide PEEP, an external valve must be attached to the exhalation limb.
- These ventilators have limited peak flow capabilities and may not be able to provide for the traumatized casualty whose minute ventilation demands are excessive.

Bear 33 and Lifecare PLV-100 and PLV-102

The Bear 33 and the Lifecare PLV-100 and PLV-102 ventilators are all portable, electrically powered, microprocessor controlled, and volume cycled. They do not require a separate source of compressed gas unless oxygen enrichment is used. All these ventilators can function in AC and SIMV modes, with some limitations. All have an internal battery and may be connected to an external battery as well. The limitations of these ventilators are as follows:

- "Apnea, breathing-circuit disconnections, leaks, exhalation-valve failures and occlusions may remain undetected...."[72(p107)] Thus, it is recommended that an exhaled volume monitor be used with these ventilators. There is no built-in capability for monitoring exhaled volume.
- The provision for enriched oxygen supply is passive and, except in the PLV-102, requires an externally attached H-valve mechanism. Three problems may be encountered:
  1. the potential misassembly of the H-valve, which may lead to obstruction during patient inspiration;
  2. the inaccuracy of the inspired oxygen concentration, which mandates the use of an oxygen analyzer (the F_{IO_2} will be ± 10% if the patient has a *stable* minute ventilation pattern, and greater if the patient is unstable);
  3. the automatic increase in tidal volume, which occurs with increased oxygen flow (in the Bear 33 and PLV-100), may cause increased airway pressure and barotrauma.
- "Substantial increases in work of breathing varied with each ventilator so that the work of breathing approached that of a patient with high airway resistance: Bear 33, 41%; Lifecare PLV-100 and PLV-102, 88%...."[72(p107)] While supported on these ventilators, the patient may appear dyspneic, and weaning by IMV is likely to be unsuccessful unless an external H-valve attachment is assembled.
- If a microprocessor failure occurs, the Bear 33 will not return to the previously set parameters, but will revert to "apnea settings" of a tidal volume of 500 mL and a respiratory rate of 16 breaths per minute.
- Lead–acid batteries are used in all these ventilators. The life of this type of battery will be shortened if it is allowed to remain discharged. If fully functioning and charged, the internal battery life is expected to be 1 to 3 hours.
- To provide PEEP therapy, an external valve needs to be attached on the exhalation limb.

Bennett MA-1

The Bennett MA-1 has been used for many years in ICUs. This ventilator is electronically powered and volume cycled (termination of inspiration). Its limitations are as follows:

- There is no built-in monitoring of exhaled volume.
- An externally attached H-valve apparatus must be used to allow IMV to be used. Hence, IMV is not synchronized and mechanical breaths may "stack" on top of spontaneous breaths.
- To provide PEEP, an external valve must be attached to the exhalation limb.

Impact Uni-Vent Model 750 M

The Impact Uni-Vent Model 750 is the newest ventilator to be accepted for military use. The Impact is a portable, electrically controlled, time-cycled, pressure-limited mechanical ventilator. It is microprocessor-controlled and is capable of control, AC, or SIMV ventilatory modes. It has an internal battery with a fully charged time capability of 9 hours. This mechanical ventilator does require a source of compressed gas to provide patient flow,

except during spontaneous breaths in the SIMV mode. The limitations are as follows:

- There is no built-in capability for exhaled volume monitoring.
- To institute PEEP therapy, an external PEEP valve needs to be attached and several control settings must be adjusted to allow monitoring of the amount of PEEP within the patient's circuit.
- The work of breathing is not well studied for this ventilator. An optional demand valve (connected to a source of compressed gas) is available, which provides an inspiratory flow of 60 L/min and is reported to provide a small pressure assist (support) of 2 to 3 cm H_2O.
- If the optional demand valve is not used, room air will be entrained through the antiasphyxiation port during the patient's spontaneous breaths. This will decrease the fractional concentration of oxygen provided to the patient.
- There is no measurement of the size of spontaneous breaths taken during SIMV.
- The digital display of peak inspiratory pressure is hard to quantitate in the most common range of pressures (10–50 cm H_2O).

Many of the mechanical ventilators projected for use in the combat casualty setting are different from those used in most peacetime hospitals. For this reason, it would be prudent for medical personnel who may be called on to care for patients with these ventilators to familiarize themselves with their operating capabilities.

SUMMARY

Respiratory failure of a magnitude that requires mechanical ventilation can be expected to occur in 10% to 15% of hospitalized casualties. The most likely causes are ARDS, severe brain injury, and severe torso injury during the postoperative phase. The diagnosis of respiratory failure depends on the observation of hypoxemia ($Pao_2 \leq 50$ mm Hg while breathing room air) and ventilatory failure ($Paco_2 >$ 50 mm Hg). Although these findings indicate the presence of severe pathophysiology, respiratory support with supplemental oxygen, incentive spirometry, and intratracheal suctioning may decrease the need for intubation and mechanical ventilation.

Mechanical ventilation has several beneficial effects, including eliminating the work of breathing for the casualty, maximizing the concentration of inspired oxygen, and ventilating collapsed and poorly ventilated alveoli. The latter changes cause a decrease in the amount of intrapulmonary shunting and an increase in the functional residual capacity of the lung. However, high airway pressure during expiration—especially in the presence of hypovolemia—may lead to decreased venous return to the heart, a fall in cardiac output, and a decrease in oxygen delivery. Barotrauma leading to tension pneumothorax is a potentially fatal complication of mechanical ventilation but is unlikely to occur if peak airway pressure is maintained below 50 cm H_2O.

Military anesthesiologists and intensivists must understand the design of deployable mechanical ventilators, be able to choose the proper mode of ventilation, and be able to select the appropriate initial ventilatory settings. These required settings include tidal volume, rate, peak gas flow, inspiratory-to-expiratory ratio, and the fractional concentration of oxygen. In addition, anesthesiologists and intensivists must be familiar with the logistics of oxygen supply.

Mechanical ventilators use three types of cycling: pressure, volume, and time. Both volume- and time-cycled ventilators allow for a constant level of tidal volume, which makes them more effective than pressure-cycled ventilators whenever pulmonary compliance or airway resistance is elevated. The initial settings that are selected for mechanical support depend on the indication for which the patient requires ventilatory support. Plausible initial settings are tidal volume, 10 to 15 mL/kg; respiratory rate, 7 to 10 breaths per minute; and inspired oxygen concentration, 40%. Both the respiratory rate and the oxygen concentration may have to be increased, and flow rates and end expiratory pressures adjusted, if pulmonary injury is present. A variety of parameters may be measured to judge the effectiveness of ventilation and oxygenation (eg, systemic Do_2 and the magnitude of the arterial-venous shunt), but useful information is also obtained from the subjective appearance of the casualty. In addition, in deployable hospitals, from the practical standpoint, an assessment as to the effectiveness of ventilation will depend on measurement of Pao_2 and $Paco_2$ measured with a blood gas analyzer, Sao_2 measured with a pulse oximeter, a chest radiogram, and the airway flow and pressure measurements from the controls and sensors of

the mechanical ventilator. The goal is to maximize oxygenation while minimizing airway pressures and inspired oxygen concentration.

Discontinuation of mechanical ventilation should be considered only if the underlying cause of the respiratory failure has been reversed. Multiple criteria have been advanced to guide the weaning process once the decision has been made to discontinue mechanical support. Among them are (*a*) Pa_{O_2} greater than 60 mm Hg when F_{IO_2} is less than 0.5, (*b*) the ability to produce a spontaneous vital capacity greater than 10 mL/kg, and (*c*) the ability to generate a negative inspiratory effort greater than 30 cm H_2O. None of these criteria are absolute. Premature discontinuation of mechanical ventilation and extubation to make possible rapid evacuation—especially when it is by air—is to be avoided in combat casualties.

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