Neonatal and Pediatric Mechanical Ventilation

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OUTLINE

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SUMMARY

KEY TERMS
- Bronchomalacia
- Bronchopulmonary dysplasia
- Choanal atresia
- Cleft palate
- Extracorporeal membrane oxygenation
- Meconium aspiration syndrome
- Neonate
- Patent ductus arteriosus
- Pediatric
- Prophylactic therapy
- Rescue therapy
- Tracheoesophageal fistula
- Tracheomalacia

LEARNING OBJECTIVES
On completion of this chapter, the reader will be able to do the following:

1. Discuss the clinical manifestations of respiratory distress in newborn and pediatric patients.
2. Identify differences in the level of noninvasive ventilatory support.
3. Describe device function and settings for different mechanical ventilatory support strategies.
4. Identify the primary and secondary goals of ventilatory support of newborn and pediatric patients.
5. Explain some key areas of assessment that influence the decision on whether to initiate ventilatory support.
6. Recognize the indications, goals, limitations, and potentially harmful effects of continuous positive airway pressure (CPAP) in a clinical case.
7. Describe the basic design of nasal devices used to deliver CPAP to an infant.
8. Compare and contrast a mechanical ventilator equipped with a CPAP delivery system to a freestanding CPAP system.
9. From patient data, recognize the need for mechanical ventilatory support in newborn and pediatric patients.
10. Identify the essential features of a neonatal and pediatric mechanical ventilator.
11. Explain how the advanced features of a ventilator enhance its usefulness over a wide range of clinical settings.
12. Relate the major differences between older-generation neonatal ventilators and modern microprocessor controlled mechanical ventilators.
13. Distinguish demand flow from continuous flow, and discuss other modifications that have been made to the basic infant ventilator.
14. Select appropriate ventilator settings based on the patient’s weight, diagnosis, and clinical history; also discuss strategies and rationale for ventilator settings.
15. Discuss newborn and pediatric applications, technical aspects, patient management, and precautions for the following ventilatory modes: pressure-control ventilation, volume-control ventilation, dual-controlled ventilation, pressure-support ventilation, airway pressure release ventilation, and neurally adjusted ventilatory assist.
16. Discuss the rationale and indications for high-frequency ventilation in newborn and pediatric patients.
17. Compare the characteristics and basic delivery systems of the following high-frequency ventilation techniques: high-frequency positive pressure ventilation, high-frequency jet ventilation,
18. Explain the physiologic and theoretic mechanisms of gas exchange that govern high-frequency ventilation, and defend the mechanism believed to be most correct.

19. Explain how settings of a given high-frequency technique are initially adjusted, the effect of individual controls on gas exchange, and strategies of patient management.

20. Discuss the physiologic benefits of inhaled nitric oxide therapy, and suggest recommended treatment strategies.

**RECOGNIZING THE NEED FOR MECHANICAL VENTILATORY SUPPORT**

Mechanical ventilation of newborn and pediatric patients involves the use of devices that recruit and maintain lung volumes, improve gas exchange and lung mechanics, assist in overcoming the resistive properties of an artificial airway, and reduce the work of breathing. These devices may provide continuous positive airway pressure (CPAP), assist spontaneous ventilation (e.g., noninvasive positive-pressure ventilation [NIV], bilevel positive airway pressure [BiPAP] units), or support part or all of the patient’s ventilatory requirements (e.g., invasive mechanical ventilation).

Currently, there are no well-defined, disease-specific criteria available to guide the decision on when to initiate mechanical ventilatory support in newborns and pediatric patients in respiratory distress. In fact, many institutions with desirable outcomes prefer to implement ventilatory support before the onset of severe respiratory illness, making the process of initiating support even more complicated. The ongoing clinical management of patients requiring ventilatory support also remains an elusive practice that is based more on experience and clinician preference and less on experimental data obtained from large randomized controlled trials.

This chapter focuses on the best available clinical and experimental evidence for initiating and managing neonatal and pediatric respiratory support. For the purpose of this discussion, a **neonate** will be defined as any infant born less than 44 weeks of age and **pediatric** will include any patient beyond the neonatal period and up to adolescence.

Clinicians caring for neonatal and pediatric patients must understand the etiology and pathophysiology of the various diseases and conditions that affect the airways and lung parenchyma of these patients.

They must also be knowledgeable of the theory of operation and limitations of the different ventilatory support devices used. Additionally, clinicians must be able to interpret physiologic data derived from the history and physical assessment, laboratory studies, and radiographic findings to evaluate properly the effectiveness of the ventilatory support provided. Keep in mind that although these data are a critical part of the decision-making process, other factors must be considered when initiating mechanical ventilation in these patients. In many cases, the approach to initiating ventilatory support may have to be individualized for neonates and pediatric patients because anatomic structure, size, and disease severity can vary widely from one patient to the next.

**Clinical Indications for Respiratory Failure**

*Respiratory failure* is defined as the inability to establish or maintain adequate gas exchange. Respiratory failure can present at birth and persist throughout the neonatal period or following a catastrophic event. Many pediatric patients encounter respiratory failure as a chronic condition (e.g., bronchopulmonary dysplasia). Although lung disease is the most common cause of respiratory failure, there are many extrinsic factors that can predispose patients to this “life-threatening” event. For example, hemodynamic conditions and congenital cardiac anomalies can also contribute to respiratory failure. Neonatal and pediatric patients have smaller lungs, greater airway resistance, lower lung compliance, less surface area for gas exchange, and lower cardiovascular reserve than do adults, making them more vulnerable to rapid deterioration. In fact, respiratory failure is a major cause of cardiac arrest in neonatal and pediatric patients. As such, clinicians must act quickly to limit the potential adverse outcomes associated with respiratory failure by observing clinical signs and symptoms. These factors can guide timely intervention well before respiratory failure develops into cardiopulmonary arrest.

**Neonate**

Neonates experiencing respiratory distress present with tachypnea, nasal flaring, and intercostal, substernal, and retrosternal retractions. The chest wall during infancy is composed primarily of cartilage, making the chest wall compliance much greater than that of the lungs. As the resistance and compliance worsen, neonates have to generate higher pleural pressures during inhalation, causing the “floppy” chest wall to collapse inward creating retractions. On exhalation, the neonatal chest wall lacks the necessary recoil to counteract the inward forces of the lungs, and thus the lungs are prone to premature collapse. Infants will attempt to maintain a back pressure in the lungs, to preserve the functional residual capacity, by narrowing the glottis and maintaining respiratory muscle activity (active exhalation). This results in vocalization during exhalation or “grunting,” which is often mistaken for infants’ crying. Grunting can usually be heard without auscultation and is a useful clinical sign of impending respiratory failure. The Silverman Anderson respiratory scoring system is a useful clinical tool to assess the degree of respiratory distress in neonates (Fig. 22.1).

Although this tool has been available in the clinical setting for nearly three decades, it has recently been reintroduced in a number of institutions to better evaluate patient response to settings changes during CPAP and mechanical ventilation.

Premature neonates can become apneic due to underdeveloped neural respiratory centers and may or may not be stimulated to reestablish spontaneous breathing. Infants that do not respond to gentle stimulation and caffeine therapy, often require immediate respiratory assistance using a manual resuscitator and when necessary, invasive mechanical ventilation.

**Pediatric**

Pediatric patients experiencing respiratory distress can present with some of the same clinical manifestations as neonates. However, larger pediatric patients have ossified or “stiffer” chest walls and are able to sustain longer periods of higher work of breathing (WOB) than neonates. Nonetheless, clinicians should be well versed in recognizing age-specific normal and abnormal respiratory and hemodynamic parameters prior to implementing mechanical respiratory support.
Determining Effective Oxygenation and Ventilation

Arterial blood gas (ABG) analysis is considered the gold standard for determining oxygenation, ventilation, and acid-base balance in neonates and pediatrics with respiratory failure. It is important to recognize that frequent ABGs can deplete the circulating blood volume of small patients. Noninvasive techniques (i.e., pulse oximetry [SpO₂] and transcutaneous CO₂ measurements) are alternative methods for trending gas exchange in most patients. Interpretation of ABG or noninvasive gas exchange values must also be coupled with data obtained from physical assessment and other clinical and laboratory data. For example, observing the color of the skin and mucous membranes can be used to assess tissue oxygenation; oxygen delivery and tissue perfusion can be evaluated clinically by noting capillary refill. Indeed, close attention to vital signs and physical assessment findings can help prevent deterioration of ABG/acid-base status.

Patients with certain congenital heart defects often require a high pulmonary vascular resistance to prevent excessive pulmonary blood flow and maintain adequate systemic circulation and cardiac output; thus, abnormal values are acceptable in this patient population before surgical correction. Additionally, allowing carbon dioxide levels to rise and pH levels to fall to abnormal levels has become a common standard for lung protection during mechanical ventilation. If necessary, an individualized or standardized approach for managing gas exchange during ventilatory support should be identified early in management.

Chest radiographic evaluation is another important tool that can add to the overall clinical assessment of patients with respiratory failure or those receiving respiratory support. Because lung volumes are difficult to measure in neonatal and pediatric patients, chest radiographs can provide valuable insight into the approximate level of lung expansion in patients susceptible to developing atelectasis or hyperinflation. Many clinicians use the chest radiograph to guide setting ventilator parameters. It is important to realize the limitations of chest radiographs and understand that frequent radiography may expose patients to unnecessary high levels of radiation. The following sections provide more in-depth information about clinical and laboratory indications for mechanical ventilatory support.

**GOALS OF NEWBORN AND PEDIATRIC VENTILATORY SUPPORT**

The goals of mechanical ventilatory support in newborn and pediatric patients are:

- To provide adequate ventilation and oxygenation
- To achieve adequate lung volume
- To improve lung compliance
- To reduce WOB
- To limit lung injury

One may argue that avoiding mechanical ventilatory support altogether or minimizing the duration of support should be the first goal because even short-term ventilation can result in ventilator-induced lung injury (VILI).

Maintenance of an appropriate functional residual capacity (FRC) ensures optimum lung mechanics, which leads to reduced WOB and reduced lung injury. To move gas into and out of the lungs, the patient must generate relatively high intrathoracic pressures to balance the resistive and elastic components that resist lung inflation. Positive-pressure ventilation can significantly reduce this burden and improve recovery in patients with lung disease. In fact, some patients receive chronic mechanical ventilatory support as a means of reducing caloric utilization by the respiratory muscles. These patients often are able to breathe spontaneously but at a significant caloric cost. Mechanical ventilatory support can help promote normal function and development of the respiratory system, especially in neonatal patients with lung disease.

**NONINVASIVE RESPIRATORY SUPPORT**

Continuous positive airway pressure (CPAP) is used in spontaneously breathing patients and may be applied with or without an artificial airway. CPAP provides a continuous distending pressure to
the lungs, which increases FRC and thus helps improve lung compliance ($C_V$). Often airway resistance (Raw) is also reduced and the patient’s WOB dramatically decreases with the use of CPAP. CPAP is most commonly applied noninvasively to the nasal airway opening and has become very popular over the last decade in the neonatal population as a strategy to avoid intubation and invasive mechanical ventilation. In this discussion, the term CPAP refers to nasal CPAP in neonates. CPAP often is recommended for patients who have adequate alveolar ventilation, and yet are hypoxic despite receiving an $F_{O_2}$ greater than 0.5. CPAP may be used to prevent atelectasis and to reduce WOB in patients who have been weaned and extubated from the ventilator.

**Box 22-1 Indications for Continuous Positive Airway Pressure (CPAP) in Newborns Via Nasal Prongs, Nasopharyngeal Tube, or Nasal Mask**

<table>
<thead>
<tr>
<th>Abnormalities on Physical Examination</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Increased work of breathing (WOB), as indicated by a 30% to 40% increase above the normal respiratory rate ($f$)</td>
</tr>
<tr>
<td>• Subternal and suprasternal retractions</td>
</tr>
<tr>
<td>• Grunting and nasal flaring</td>
</tr>
<tr>
<td>• Pale or cyanotic skin color</td>
</tr>
<tr>
<td>• Agitation</td>
</tr>
<tr>
<td>• Inadequate arterial blood gas (ABG) values:</td>
</tr>
<tr>
<td>• Inability to maintain a partial pressure of arterial oxygen ($PaO_2$) above 50 mm Hg with a fraction of inspired oxygen ($F_{O_2}$) of 0.6, provided minute ventilation $V_e$ is adequate, as indicated by a partial pressure of arterial carbon dioxide ($PaCO_2$) of 50 mm Hg and a pH of 7.25 or higher</td>
</tr>
<tr>
<td>• Poorly expanded and/or infiltrated lung fields on a chest radiograph</td>
</tr>
</tbody>
</table>
| • Presence of a condition thought to be responsive to CPAP and associated with one or more of these:
  • Respiratory distress syndrome (RDS)
  • Pulmonary edema
  • Atelectasis
  • Apnea of prematurity
  • Recent extubation
  • Tracheal malacia or other abnormality of the lower airways
  • Transient tachypnea of the newborn
  • Very low birth weight infants at risk for the development of RDS as an early intervention along with surfactant administration.
| • Administration of controlled concentrations of nitric oxide in spontaneously breathing infants |


**Neonatal Continuous Positive Airway Pressure in Neonates**

**Indications and Contraindications**

When introduced in 1971, CPAP was touted as the “missing link” because it could provide oxygen treatment while avoiding mechanical ventilation in the neonate. Used appropriately, CPAP is a less invasive and less aggressive form of therapy than other forms of ventilatory support. Newborns with retained lung fluid, atelectasis, insufficient surfactant production, or respiratory distress syndrome (RDS) are good candidates for CPAP. Such patients include very low-birth-weight (VLBW) and premature infants. CPAP also can be used successfully in infants with respiratory distress arising from other causes, including transient tachypnea of the newborn, meconium aspiration syndrome, primary pulmonary hypertension, pulmonary hemorrhage, paralysis of a hemidiaphragm, and following surgical repair of diaphragmatic hernias and congenital cardiac anomalies, congenital pneumonias, respiratory syncitial virus (RSV) bronchiolitis, apnea of prematurity, and congenital and acquired airway lesions.

Box 22-1 lists the indications for CPAP in neonates originally established by the American Association for Respiratory Care (AARC). Compared with standard oxygen therapy, CPAP reduces grunting, and tachypnea, increases FRC and arterial oxygen partial pressure ($PaO_2$), decreases intrapulmonary shunting, improves lung compliance, aids in the stabilization of the floppy infant chest wall, improves distribution of ventilation, and reduces inspiratory WOB. CPAP is believed to reduce the severity and duration of central and obstructive apneas by mechanically splitting the upper airway, promoting better alveolar recruitment, oxygenation, and stimulation of the infant to breath.

Although there is no consensus on how best to manage neonates on CPAP, two general approaches are currently being used to minimize the use of invasive mechanical ventilation and better protect the fragile neonatal respiratory system. Early CPAP involves implementing therapy in the delivery room or neonatal intensive care unit (NICU) only after the infant is stabilized and is effectively breathing on his or her own. This is performed prophylactically even if the neonate is not exhibiting respiratory distress or apnea. The goal is to recruit air spaces and maintain lung volumes early to promote gas exchange and reduce the likelihood that respiratory failure and apnea will occur. This approach is beneficial for premature infants that lack lung surfactant and are at risk for developing atelectasis. Breathing at low lung volumes can result in unnecessary lung injury (atelectrauma), which can hinder surfactant production. Many premature neonates can be managed successfully using CPAP without ever requiring endotracheal intubation and mechanical ventilation. If the patient does develop respiratory failure, then he or she is intubated, given lung surfactant, and then promptly weaned from the ventilator and extubated. This approach is a drastic departure from the previous approach that has been used over the last 20 years, in which neonates would be intubated and placed on a ventilator for weeks or even months until they were a certain size or weight. Centers that implement early CPAP report a very low incidence of infants developing chronic lung disease or bronchopulmonary dysplasia (BPD) because the lungs are not being subjected to the relatively large inflation pressures that are observed during mechanical ventilation. A recent clinical trial in premature neonates demonstrated that this early CPAP approach resulted in less need for intubation and fewer days of mechanical ventilation, and infants were more likely to be alive and free from the need for mechanical ventilation after a week than were neonates intubated for surfactant and supported with mechanical ventilation for at least 24 hours.

Another clinical approach implements elective intubation, prophylactic surfactant administration, short-term lung-protective ventilation, and rapid extubation to CPAP within hours of birth. (Note: This approach is also known as InSURE (Intubate, SURfactant, Extubation) and is implemented shortly following birth.) The InSURE approach assures that all premature infants will
receive at least one dose of surfactant, but it does not eliminate the potential that even short-term ventilation can result in some degree of lung injury. The InSURE approach has been associated with lower incidences of mechanical ventilation, air-leak syndromes, and BPD than an approach that administers surfactant and embraces prolonged mechanical ventilation support.11

The major question that remains is whether these two disparate approaches impact long-term survival and the development of chronic complications (e.g., BPD). Another important outcome related to these different approaches is the incidence of CPAP failure and subsequent ventilation requirements among neonates. Approximately 25% to 40% of infants with birth weights between 1000 and 1500 g may fail early CPAP and require intubation and mechanical ventilation, whereas 25% to 38% of infants with similar birth weights may fail CPAP using the InSURE approach.6 Some institutions use a combination of these approaches wherein smaller premature neonates (<28 weeks' gestational age), with lower surfactant production, will be supported initially using InSURE and all other larger neonates are supported using the early CPAP strategy. Both these strategies strive to minimize invasive mechanical ventilation and have redefined the approach to supporting premature infants at risk for developing respiratory failure. Minimally invasive ventilation strategies, such as CPAP, have likely been a major reason why premature neonates are able to survive at a lower gestational age and with fewer complications than ever before.

Any neonate that has recently been extubated from mechanical ventilation is at risk for developing hypoxemia, respiratory acidosis, and apnea. Extubation to CPAP, regardless of whether surfactant was administered, has been associated with a reduction in the incidence of respiratory failure and the need for additional ventilatory support in neonates.12

Infants with certain congenital heart diseases reportedly benefit from CPAP. Cardiac anomalies that increase pulmonary blood flow can reduce lung compliance (C(L)) and FRC, thus increasing WOB and worsening hypoxia. The most common defects associated with increased pulmonary blood flow are ventricular septal defects, atrial septal defects, atrioventricular (AV) canal, and patent ductus arteriosus. Positive intrathoracic pressure produced by a CPAP system can mechanically reduce pulmonary blood flow while restoring FRC.5,13

Use of CPAP is not appropriate and is potentially dangerous in infants who show signs of nasal obstruction or severe upper airway malformation, such as choanal atresia, cleft palate, or tracheoesophageal fistula.13 CPAP has been used in patients with bronchitis, but its use in these patients has been controversial and may be contraindicated in some cases.5,13,16 More recent evidence suggests, however, that CPAP can result in favorable outcomes in infants affected with bronchitis by reducing carbon dioxide levels and eliminating the need for mechanical ventilation.17

Patients who have severe cardiovascular instability, severe ventilatory impairment (pH < 7.25, PaCO2 > 60), refractory hypoxemia (PaO2 < 50 torr or >0.6 FIO2), frequent apnea that does not respond to stimulation or intravenous caffeine therapy, or are receiving high levels of sedation may require intubation and mechanical ventilation rather than CPAP.13 CPAP or any form of noninvasive positive pressure to the airway should not be used in infants with untreated congenital diaphragmatic hernia; these infants should be intubated to prevent gastric insufflations and distention and further compromise of the heart and lungs.13 Some surgeons discourage the use of CPAP in infants after any surgical procedure involving the gastrointestinal tract.

Most commonly, CPAP is applied via nasal prongs; however, some clinicians prefer to use CPAP in intubated neonates while weaning from mechanical ventilation to observe whether the infant is experiencing apnea. Prolonged support using this approach should be discouraged whenever possible because it is has been associated with increased need for reintubation after the breathing tube has been removed.12

Application of Nasal CPAP

Because newborns are obligate nose breathers, nasal CPAP can be applied in several ways. Previously, a short endotracheal tube (ET) was placed into one of the nares and taped to the face to provide nasopharyngeal tube CPAP. A snug-fitting set of short binastral prongs is the most commonly used interface. Neonatal nasal masks are also available and are gaining popularity among some clinicians. It is common practice to alternate between these two nasal airway interfaces. Both devices are effective, but the distending pressure they provide can be lost when the infant cries or breathes through the mouth. Binastral prongs and masks may be more beneficial than a nasopharyngeal tube because they are less invasive and provide the least amount of resistance to gas flow and, hence, lower imposed (or resistive) WOB, and facilitate mobilization and oral feeding.14,18 Additionally, short binastral prongs were found to be more effective than using single nasopharyngeal tube in reducing the rate of re-intubation in premature neonates supported with CPAP.19

Nasal prongs and masks must be fitted correctly so that they do not leak or cause trauma to the patient. Nasal prongs and masks usually are made of a latex-free material, such as silicone. Molded into a manifold or attached to one, the prongs are placed just inside the infant's nares (Fig. 22-2). Prongs are available in a variety of sizes. The fit of the prongs is critical; they must fit snugly into the nares but must not be so tight as to cause skin blanching. If a nasal mask is used instead of prongs, it, too, must be carefully selected for proper size and fit. Masks can cause pressure injury to the skin if improperly fitted, and they may not seal if they are too large or small.

The entire CPAP apparatus is stabilized on the patient's head with headgear or a “fixation” system consisting of a bonnet, cap, or straps (Fig. 22-3). Many commercial system configurations are available. The correct size of straps and head coverings must be carefully chosen and adjusted so that no part of the infant's head is subjected to squeezing or occlusive pressure points. After the CPAP delivery device has been attached to the CPAP manifold, the CPAP level and FIO2 are set. The CPAP system is assessed frequently to ensure effective delivery. The patient's nose is checked regularly for signs of pressure necrosis, and the prongs must be checked routinely for patency.

The CPAP system functions primarily to regulate gas flow during inhalation and exhalation and to maintain a consistent
pressure at the nasal airway opening. The CPAP system consists of five essential components:
1. A heated/humidified blended gas source
2. A nasal interface
3. A patient circuit
4. The pressure regulation mechanism
5. A means for monitoring and limiting the airway pressures

Numerous studies have compared differences in gas exchange, WOB, and requirement for (re)intubation with the available CPAP devices; however, it is important to note that no single device has been shown to be superior to another when major outcomes (i.e., mortality and morbidity) in neonates are considered (Key Point 22-1).

**Key Point 22-1** The success by which CPAP is applied to neonates is probably based more on the clinicians’ abilities to understand the system and identify pathophysiologic changes in response to settings changes than is the type of CPAP device or nasal interface being used.

Over the last three decades, the most common CPAP system that has been applied noninvasively has been accomplished using a mechanical ventilator. Ventilator CPAP has also been referred to as conventional CPAP in the clinical setting. Ventilator CPAP is convenient because it has traditionally been used following extubation from mechanical ventilation and does not rely on having to use a separate device to apply therapy. Many ventilator manufacturers have designed specific noninvasive CPAP modes into the ventilator platform exclusively for neonates. The clinician can set the CPAP level, and the exhalation valve regulates the pressures accordingly. Another advantage of these modes is that the ventilator may be able to deliver noninvasive “backup breaths,” based on a preset apnea interval when the neonate has stopped breathing. One potential limitation of ventilator CPAP is that the demand flow system may not be able to respond adequately to changes in patient respiratory efforts because pressure is being measured back at the ventilator and not at the patient’s airway. Additionally, new evidence suggests that the expiratory resistance of newer neonatal ventilator’s exhalation valves may impose additional resistance and hence increase the WOB during spontaneous breathing.

Two commercially available, freestanding CPAP devices that use fluidic gas principles are widely used to provide CPAP to infants. The Hamilton Arabella (Fig. 22-3, A and B) and the CareFusion Infant Flow SiPAP System (Fig. 22-4) are favored by some clinicians. The overall function of these fluidic devices to generate CPAP at the airway is similar. Both devices have a fully integrated flow controller, a delivery circuit, different-sized nasal prongs that attach to a gas delivery manifold, and a bonnet with Velcro straps to secure the manifold and prongs. The manifold incorporates a fluidic flip-flop mechanism at the infant’s nasal airway opening to regulate flow to match the infant’s inspiratory demand and provide a consistent pressure level (see Fig. 22-3, B). The controller provides gas flow with an adjustable FIO₂ and also monitors CPAP pressure. Turbulence caused by continuous flow has been eliminated in these systems to make exhalation easier and reduce the WOB. The manufacturers have attempted to provide
better-fitting, easier-to-secure nasal prongs to make CPAP more effective and patient care easier. These devices are particularly effective at reducing WOB in VLBW infants. CPAP is better tolerated in these patients, which helps to avert the need for positive-pressure ventilation.21

Bubble CPAP (B-CPAP) is a technique for delivering CPAP via a simple freestanding system (Fig. 22-5). It has been used in certain forms for more than 30 years and is again gaining favor over other CPAP techniques. In the United States, two B-CPAP devices are now available (B&B Medical, Fischer Paykel), but most centers that implement B-CPAP have done so using homemade systems consisting of a blended and humidified gas source, ventilator circuits, nasal prongs, pressure manometer, and a water column. The blended gas flow is adjusted at 5 L/min, and the CPAP level is regulated by varying the depth of the distal ventilator circuit below the water surface (i.e., 5 cm = 5 cm H$_2$O CPAP). Higher pressures than those anticipated by the submersion depth of the distal tubing have been observed when using higher flows,22,23; thus clinicians will use the lowest flow possible to maintain constant bubbling throughout the respiratory cycle. An additional safeguard involves measuring the pressure at the nasal airway interface using a pressure manometer and limiting excessive pressures with a safety “pop-off” valve during B-CPAP.

Tiny vibrations or oscillations in the airway pressure created by gas “bubbling” through the water column may assist in enhancing gas exchange and lung recruitment. Anecdotal reports made by clinicians have observed the chest walls of intubated premature neonates supported by B-CPAP oscillating at frequencies similar to those provided by high frequency ventilation,24 but these ventilation effects have never been quantified in neonates using “leaky” nasal prongs.

Initial pressures for CPAP are commonly set at about 4 to 6 cm H$_2$O.25 The CPAP level requirements are likely to fluctuate throughout the course of treatment, and the optimal level is one that results in adequate lung inflation without overdistending the lung parenchyma.26 If little clinical improvement is seen, the level is gradually increased to 10 cm H$_2$O in increments of 1 to 2 cm H$_2$O.26 The response is considered adequate when the required F$_{O_2}$ is 0.6 or less and the PaO$_2$ is at least 50 mm Hg.26 Adequate oxygenation usually is accompanied by reduced WOB, as manifested by a 30% reduction in the respiratory rate and a decrease in retractions, grunting, and nasal flaring. In some cases the chest radiograph indicates improvement by showing better aeration and increased lung volumes (Key Point 22-2).27 Continuous noninvasive monitoring of transcutaneous CO$_2$ pulse oximetry, and Silverman Respiratory Scores can provide reliable trending of physiologic response in patients when adjustments are made to the CPAP level.

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**Key Point 22-2** Regardless of the type of nasal prongs or nasopharyngeal (NP) tube used for CPAP, the clinician must always verify the patency of the device and strive to reduce injury by frequently assessing the proper fit.

**Complications of CPAP**

CPAP can cause pulmonary overdistention and can lead to ventilation/perfusion mismatching, decreased pulmonary blood flow, increased pulmonary vascular resistance, and decreased cardiac output.28 Marked overdistention can increase WOB and cause CO$_2$ retention.29 Air-leak syndromes have also been reported.30 The clinician must be aware that the CPAP system can cause abdominal distention and gastric insufflation, which can lead to aspiration if not detected and corrected early.31 Perforations of the gastrointestinal tract, although rare, are possible.32 Excessive pressure from the application devices can injure the nose and nasal mucosa, and inadequate humidification can contribute to injury.

**Noninvasive Positive-Pressure Ventilation in Neonates**

As noted in the previous discussion, a large percentage of neonates supported by CPAP still develop severe respiratory failure requiring endotracheal intubation and mechanical ventilation. Recent evidence suggests that invasive mechanical ventilation contributes to the development of BPD and other complications in neonates.32 It is unclear whether this is related exclusively to the VILI or whether the presence of the ET in the airway is also a contributing factor.

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![Diagram of the Bubble CPAP delivery system. (Redrawn from Aly H, Miller JD, Patel K, El-Mohandes AA: Does the experience with the use of nasal continuous positive airway pressure improve over time in extremely low birth weight infants? Pediatrics 114 (3):697, 2004.)](image-url)
factor. Endotracheal intubation is a traumatic and painful procedure that a neonate can experience, especially if proper sedation levels are not achieved. Intubation is accompanied by significant hemodynamic instabilities, airway injury, colonization of the trachea, reduced ciliary movement, secretions, high resistance to air flow, and increased WOB.\(^{33}\)

Also known as “CPAP with a rate,” Noninvasive positive pressure ventilation (NIV) is an established form of ventilatory support in adults and pediatric patients. It is accomplished by using superimposed positive-pressure inflations with CPAP. NIV is becoming a new intermedary approach between CPAP and invasive mechanical ventilation to reexpand atelectatic areas, improve gas exchange, reduce respiratory distress, prevent apnea, and potentially avoid the need for invasive mechanical ventilation. NIV is used as an initial form of respiratory support and following extubation from invasive mechanical ventilation.\(^{34}\) Like CPAP, NIV assists spontaneous breathing patients only, and thus neonates with persistent apnea cannot be supported by this method of mechanical ventilator support. Further, not all neonates can be supported by NIV alone, and intubation is indicated for severe ventilatory impairment (pH <7.25, PaCO\(_2\) >60), refractory hypoxemia (PaO\(_2\) <50 torr on >0.6 F\(_{2}O\(_{2}\)) , and frequent apnea that does not respond to stimulation or intravenous caffeine therapy.\(^{35}\) The same complications that arise during CPAP and mechanical ventilation can be observed during N-IPPV.

Most commonly, NIV is applied using short binaural prongs or a nasal mask and a fixation technique similar to that of CPAP. As improvements in nasal airway interfaces and ventilator devices have evolved, clinicians have begun implementing different forms of NIV in neonates with little experimental evidence to support their use. The following section discusses the most common methods and approaches for applying NIV in neonates.

**Nasal Intermittent Mandatory Ventilation in Neonates**

Nasal synchronized and intermittent mandatory ventilation, or “nasal IMV” (IMV or N-IMV), is the most commonly used form of NIV in neonates, and pressure control is the most common mode for providing NIV in neonates. Like nasal CPAP, it requires placement of a nasopharyngeal tube or snugly fitting nasal prongs or mask. In addition to the CPAP effect of the ventilator, the patient’s spontaneous breaths are assisted by patient-triggered or machine-triggered, time-cycled, positive-pressure inflations. Although ventilators, equipped with proximal flow sensors, have been used for patient-triggered nasal IMV, appropriate triggering is difficult to obtain because of the large leaks that can occur between the patient airway and nasal interface.

Traditionally, the most commonly used device for patient-triggered nasal IMV in neonates has been using as the Starsynchn abdominal capsule used with the Infrasinos Infant Star ventilator (Infrasinos Mallinkrodt, Inc., St. Louis, Mo). The Infant Star ventilator is no longer being supported by the manufacturer; thus machine-triggered, N-IMV breath types are being used with apparent success. In a recent publication, there were no clinically relevant differences in WOB and gas exchange in neonates comparing patient-triggered with machine-triggered breath types during N-IMV.\(^{36}\) Suggested initial N-IMV settings in neonates are inspiratory pressure 16 to 20 cm H\(_{2}\)O, positive-end expiratory pressure (PEEP) 4 to 6 cm H\(_{2}\)O, inspiratory time of 0.35 to 0.45 seconds, rate 40 to 60 breaths/min and F\(_{2}O\(_{2}\) adjusted to keep saturations 90% to 96%.\(^{37}\) Subsequent adjustments in peak inspiratory pressure (PIP) are adjusted to improve chest rise and ventilator rate is adjusted to maintain CO\(_{2}\) levels. Pressure-support ventilation is typically not provided to assist spontaneous breaths due to large airway leaks and ineffective triggering.

Compared with standard CPAP approaches, N-IMV has been shown to improve chest wall stabilization and synchrony, reduce WOB and apnea, and promote better gas exchange. These physiological differences are likely related to the use of higher mean airway pressures and the ability to provide active stimulation and “sighs” to recruit airspaces and prevent apneic episodes during N-IMV.\(^{38}\) In two separate clinical trials, premature neonates supported with patient-triggered N-IMV had fewer requirements for endotracheal intubation and less BPD than those supported by CPAP.\(^{37,38}\) Additionally, there were no reported risks of gastrointestinal insufflation or perforation related to the use of N-IMV in neonates. It has been suggested that the reduction in BPD may be related to the absence of an ET and the natural pressure release created at the neonate’s mouth and nasal airway, which may limit excessive pressure transmission to the distal airways during N-IMV.

**Nasal “Sigh” Positive Airway Pressure in Neonates**

Nasal “sigh” positive airway pressure (SiPAP, CareFusion, Viasys, San Diego, Calif) (nasal SiPAP) (see Fig. 22-4) is a relatively new form of NIV that is being used more frequently to assist spontaneously breathing infants in the NICU. Nasal SiPAP is different from other forms of NIV because it allows the neonate to breathe continuously at CPAP and during a sustained “sigh” breath to recruit lung units at two different lung volumes. Simply put, the neonate is able to breathe at a high and a low CPAP setting. The sum of alveolar ventilation depends on both the neonate’s spontaneous minute ventilation and the minute ventilation created by nasal SiPAP when transitioning between the two preset CPAP levels. The higher CPAP level is generally set at 2 to 4 cm H\(_{2}\)O higher than the baseline CPAP pressure (4-6 cm H\(_{2}\)O), the breath hold at the higher CPAP level is set at 0.5-1 second, and the respiratory rate controls the frequency of the machine-triggered “sigh” breaths. The same nasal prongs, masks, and fluid-flip mechanism as the infant flow nasal CPAP is used during SiPAP. Preliminary clinical studies in neonates following extubation have demonstrated that SiPAP provides better gas exchange and results in less need for invasive mechanical ventilatory support than conventional CPAP without causing additional lung injury.\(^{40-42}\)

**Noninvasive Nasal High-Frequency Ventilation in Neonates**

Nasal high-frequency ventilation (N-HFV) has been used more commonly as a form of NIV in clinical practice over the last 5 years. Unlike N-IMV and nasal SiPAP, N-HFV uses smaller pressures, higher frequencies, and may be more lung protective than other NIV devices that apply higher pressure to the lungs. The most common ventilator that has been used to apply N-HFV is the Infrasinos Infant Star ventilator. The N-HFV is applied using either an nasopharyngeal tube or binaural prongs with fixation. Unfortunately, N-HFV is such a new form of NIV that there are very few published papers to suggest a strategy for long-term management of neonatal patients. Initial mean airway pressure is usually set to equal the previous level of CPAP, frequency is set at 10 HZ, amplitude is adjusted to obtain visible chest wall vibration and increased every 30 minutes by 4 to 6 units if necessary to maintain clinically appropriate chest wall vibration or blood gases.\(^{43}\) In one study, researchers demonstrated a significant
reduction in PaCO₂ in neonates that were transitioned from CPAP to short-term nasal HFV. Another short-term study showed that nasal HFV promotes better alveolar growth and development in preterm lungs than invasive mechanical ventilation. This research has stimulated a tremendous amount of interest in using nasal HFV as an initial form of ventilatory support for neonates failing CPAP and following extubation from mechanical ventilation. Although the widespread use of nasal HFV is not common, much-needed research will be required to evaluate whether nasal HFV reduces the need for intubation and the incidence of BPD in neonates with respiratory distress.

**CPAP and BiPAP in Pediatric Patients**

Although CPAP is used less often for pediatric patients than for adults, it is useful in children to restore FRC and reduce WOB with acute hypoxemia, neuromuscular disorders, and conditions that cause abdominal distention. It also is used to relieve the airway obstruction associated with obstructive sleep apnea or airway lesions like laryngotracheal malacia. The use of CPAP for many of these purposes follows the guidelines established for adults (see Chapters 13 and 19).

Some clinicians recommend the use of ventilator CPAP trials to help evaluate an intubated patient's readiness for extubation after a weaning period during mechanical ventilation. The intent of the CPAP trial is to evaluate spontaneous breathing. However, WOB can increase markedly when CPAP is provided through a small ET. When spontaneous breathing evaluations are performed, enough pressure support to overcome ET resistance should be provided, or ET resistance compensation, a feature found on some ventilators, should be used. This approach is explored in more detail in the chapter on weaning and discontinuation of mechanical ventilation (Chapter 20).

In addition, CPAP may be provided effectively through a tracheostomy tube. Patients who fatigue easily because of neuromuscular weakness or who are susceptible to lung collapse seem to tolerate CPAP by tracheostomy tube, especially if continuous or nearly continuous support is needed.

In nonintubated patients, nasal prongs, a nasal mask, or a full facemask can be used in children through the toddler years. Nasal prongs that are designed for neonates are unable to be used in larger infants and toddlers. Furthermore, these patients may not be able to trigger effectively the demand flow systems in these devices. These factors pose a unique challenge for clinicians and manufacturers considering CPAP in this patient population. Children who are 3 years old or older who require CPAP generally are encouraged to use a nasal mask. Some of these patients are better managed with a full facemask, especially if they require CPAP only intermittently. Pediatric patients with airway obstructions often benefit from CPAP. Certain obstructions, such as tracheomalacia or bronchomalacia, can make weaning from ventilation and extubation difficult. A decision may be made early in a patient's course to perform a tracheotomy and to apply CPAP on a 24-hour basis. Other patients with less severe obstructions may have difficulty breathing only when they are sleeping. These patients can avoid a tracheotomy and continue to rely on CPAP provided by nasal prongs. Many patients with obstructive lesions require surgery, and CPAP often is necessary until correction is complete.

Some patients have mechanical obstruction of the upper airway caused by soft tissue or excessive loss of muscle tone during sleep (i.e., obstructive sleep apnea [OSA]). OSA involves obstruction by either the tongue or the soft palate. Often CPAP can “stent” open these obstructions and dilate the oropharynx during sleep and reduce apnea.

The BiPAP system (Philips, Respironics, The Netherlands) delivers CPAP by nasal or full facemask to children and adolescents. This device is useful for patients with higher inspiratory flow rates and can overcome leaks at the mask by increasing flow. It also can monitor the tidal volume (VT) and minute ventilation, and it provides high and low alarms and high FiO₂ levels.

Also, CPAP systems are available for home use in patients who require chronic support. These units are recommended for nasal CPAP using nasal prongs or a mask in older children and adults. Bilevel positive airway pressure units (e.g., Respironics BiPAP ST and S/T-D and Vision) also can be set to deliver CPAP. These units are recommended for adults and children older than 1 year who require little or no supplemental oxygen. Like home CPAP systems, these units are intended to be used with nasal prongs (pillows) or masks held in place by adjustable headgear. The BiPAP unit can easily be switched from CPAP to BiPAP without modifications for patients who need additional support (Case Study 22-1 and 22-2).

The use of BiPAP systems and NIV, using critical care ventilators, has gained considerable popularity in the pediatric intensive care unit (PICU) settings as an alternative to invasive mechanical ventilation to support spontaneously breathing patients with acute...
respiratory failure and acute exacerbations of chronic lung disease. It is also being used to support patients, following extubation, who are difficult to wean from the ventilator or are thought to have difficulties following extubation. Patients with acute exacerbation of asthma, acute respiratory distress syndrome (ARDS), cystic fibrosis, neuromuscular disorders, and respiratory infections (i.e., pneumonia) have been supported successfully using this approach.

Both BiPAP and NIV have been shown to improve gas exchange and reduce the need for invasive ventilation by 40% in pediatric patients with acute respiratory failure. Inspiratory positive airway pressures, or IPAP, settings are initially set very low and increased slowly to provide time for the patient to become comfortable and allow the clinician to assess accurately the reduction in WOB. The PEEP or EPAP settings are usually set between 4 and 10 cm H₂O. The maximum inspiratory pressure or IPAP level is dependent on patient size and lung pathology. Older pediatric patients may tolerate inspiratory pressure as high as 20 cm H₂O, but in all cases, a nasogastric or orogastric tube should be placed and small amounts of sedation should be considered to improve comfort. Gastric insufflation has been observed in pediatric patients (>1 year old) using inspiratory pressure greater than 15 cm H₂O with a full face mask. However, larger patients may tolerate higher inspiratory pressure, but the patient should have a gastric tube and be monitored frequently for abdominal distention. In patients who do not tolerate this strategy or continue to develop severe respiratory failure and poor gas exchange, invasive mechanical ventilation is indicated.

**CONVENTIONAL MECHANICAL VENTILATION**

Conventional mechanical ventilation or “invasive” mechanical ventilation involves the use of positive-pressure inflations in intubated patients who are breathing spontaneously or who are heavily sedated or paralyzed. Many of the techniques for managing neonatal and pediatric patients during conventional mechanical ventilation have been based on adult strategies. Neonatal and pediatric patients present with a multitude of respiratory diseases that warrant fundamentally different ventilator approaches. There have been no large randomized clinical trials in this patient population to suggest that a particular ventilator mode or ventilator brand is preferable over another in managing different lung disease. However, there is overwhelming agreement among clinicians that the management of such patients should be to avoid invasive ventilation whenever possible to minimize VILI. This section discusses the techniques most widely practiced in neonatal and pediatric ventilator management. Clinicians working with children should always keep guidelines in mind, but they should also be able to “think outside the box” when faced with the unique challenges these patients sometimes present.

**Indications for Ventilatory Support of Neonates**

New advances in noninvasive ventilatory support have resulted in less frequent use of invasive ventilation; however, mechanical ventilation is a “lifesaving” intervention that remains an essential tool for managing neonates with respiratory failure. Infant mortality caused by respiratory distress syndrome in the United States decreased from >268 in 100,000 live births in 1971 to 98 in 100,000 live births in 1985 and 17 in 100,000 live births in 2007. The decrease in mortality from 1971 to 1985 was, in large part, due to the development and widespread availability of mechanical ventilators designed to work well in neonates. Approximately 2% of neonates born in the United States require mechanical ventilatory support at or shortly after birth. About 75% of these patients are either very low birth weight (VLBW) neonates (i.e., weighing <1500 g) or are low birth weight (LBW) neonates (weighing 1500-2500 g). As a result, ventilator care of the newborn often is an integral part of the broader management of premature infants.

Most newborns who require full ventilatory support are placed on infant ventilators or infant-through-adult ventilators specifically designed to respond to even the smallest patients. The indications for ventilation of neonates are listed in Box 22-2.

Infants with very low Apgar scores who do not respond to initial resuscitation efforts may require early intubation and ventilatory support. Intubation and mechanical ventilatory support are usually necessary when an infant has been diagnosed with congenital anomalies that are likely to interfere with normal ventilatory function (e.g., diaphragmatic hernia, cardiac structural defects). The decision to provide mechanical ventilation for infants who do not have any of the previously described conditions is more subjective. The degree of respiratory distress is a valuable indicator even when ABG values are within acceptable ranges. Intercostal
and substernal retractions, grunting, and nasal flaring are classic warning signs of impending ventilatory failure. Increasing supplemental oxygen requirements may be a sign that gas exchange is worsening. These indicators, along with the patient and maternal histories, often are more persuasive for initiation of ventilatory support than are laboratory data. Infants who are intubated for reasons other than respiratory failure often are sedated and mechanically ventilated for at least a short period. In many such cases, airway protection is the only indication for intubation. In other situations, sedation is required to alleviate discomfort during certain procedures. Whenever ETs are placed, the WOB from airway resistance can increase markedly. Even when these patients have sufficient drive to breathe spontaneously, minimum positive pressure or pressure support can help overcome ET resistance and help prevent lung collapse.

Indications for Ventilatory Support of Pediatric Patients
In contrast to premature infants, term infants and older pediatric patients have a wider variety of conditions requiring mechanical ventilatory support. One of every six term infants and children who are admitted to an intensive care unit (ICU) requires some form of mechanical ventilation. Airway obstruction is a common cause of intubation and ventilation. A study by the Pediatric Lung Injury and Sepsis Network found that 13.5% of children requiring mechanically ventilator for longer than 24 hours were intubated as a result of airway obstruction. The most frequently diagnosed cause of respiratory failure in children under 1 year old was bronchiolitis; in children older than 1 year, pneumonia was most often the cause.

Recognizing the need for ventilatory support in older pediatric patients involves many of the same criteria used to assess adult patients. Compared with adults, children have a limited capacity for compensation of acute illness and are more likely to develop respiratory distress with apnea and hypoxemia early. In pediatric patients, ventilation that is insufficient to provide adequate gas exchange is determined primarily by ABG results and additional clinical assessments. This clinical condition has many possible causes (Box 22-3).

The Pediatric Ventilator
Ventilators designed for infants and small children have been available since the late 1960s. Even with the availability of neonatal units, many clinicians used adult models for neonates and small pediatric patients through the 1970s and early 1980s. They were more familiar with adult units and often found it difficult to justify financially the purchase of a ventilator that could be used only for a limited number of patients. Currently there is only one commercially available “stand-alone” neonatal-specific ventilator that has higher volume and flow capabilities, which allows it to be used to ventilate larger infants. However, most current-generation, microprocessor-controlled ventilator platforms can provide seamless ventilation for any size patient, from premature newborns to adults. A single ventilator for all patient sizes and age ranges may provide several advantages related to institutional cost, training, and patient safety. Commercially available neonatal and pediatric ventilators should incorporate the essential features described in Box 22-4.

Current-generation ventilators also provide several choices of modes, including volume-controlled mandatory continuous ventilation (VC-CMV), pressure-controlled continuous mandatory

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**BOX 22-3** Indications for Use of Mechanical Ventilation in Pediatric Patients

<table>
<thead>
<tr>
<th>Respiratory Failure</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Partial pressure of arterial carbon dioxide (PaCO₂) over 50 to 60 mm Hg</td>
</tr>
<tr>
<td>• Partial pressure of arterial oxygen (PaO₂) under 70 mm Hg</td>
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<table>
<thead>
<tr>
<th>Neuromuscular or Hypotonic Disorder</th>
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</thead>
<tbody>
<tr>
<td>• Muscular dystrophies</td>
</tr>
<tr>
<td>• Spinal muscular atrophy</td>
</tr>
<tr>
<td>• Guillain-Barré syndrome</td>
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<tr>
<td>• Myasthenia gravis</td>
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<table>
<thead>
<tr>
<th>Intrinsic Pulmonary Disease</th>
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</thead>
<tbody>
<tr>
<td>• Viral/bacterial pneumonia</td>
</tr>
<tr>
<td>• Aspiration pneumonia</td>
</tr>
<tr>
<td>• Asthma</td>
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</tbody>
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<table>
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<tr>
<th>Increased Intracranial Pressure</th>
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</thead>
<tbody>
<tr>
<td>• Direct trauma</td>
</tr>
<tr>
<td>• Diabetic ketoacidosis</td>
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<table>
<thead>
<tr>
<th>Near-drowning</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Seizure disorders</td>
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<table>
<thead>
<tr>
<th>Infection</th>
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</thead>
<tbody>
<tr>
<td>• Surgical procedures involving the head, neck, chest, or abdomen</td>
</tr>
</tbody>
</table>

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**BOX 22-4** Essential Features for Commercially Available Neonatal/Pediatric Ventilators

<table>
<thead>
<tr>
<th>Pressure-control ventilation (PC), volume-control ventilation (VC), pressure support ventilation (PSV) and dual-control ventilation (DC), modes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Continuous mandatory ventilation (CMV), intermittent mandatory ventilation (IMV), and continuous spontaneous ventilation (CSV)</td>
</tr>
<tr>
<td>Flow or pressure triggering</td>
</tr>
<tr>
<td>Visible and audible alarms for high and low pressures and volumes</td>
</tr>
<tr>
<td>High-pressure release to ambient capability</td>
</tr>
<tr>
<td>Visible and audible alarms for low and high oxygen concentrations</td>
</tr>
<tr>
<td>Visible and audible alarms for loss of power and gas source</td>
</tr>
<tr>
<td>Servo-regulated humidifier with low-compressible-volume water chamber and continuous-feed water supply system</td>
</tr>
<tr>
<td>Low compliance ventilator circuit and/or capability for ventilator to measure and subtract compressible volume from delivered and monitored volume displays</td>
</tr>
<tr>
<td>Proximal flow sensor (essential for neonatal patients) or compliance factor (sufficient for larger patients)</td>
</tr>
<tr>
<td>Mechanism to drain water condensate from circuit or heated inspiratory/expiratory circuit limbs</td>
</tr>
</tbody>
</table>
ventilation (PC-CMV), volume-controlled intermittent mandatory ventilation (VC-IMV), pressure-controlled intermittent mandatory ventilation (VC-IMV), CPAP, and pressure support ventilation (PSV). In the last decade, other forms of volume-targeted or “hybrid” modes of ventilation, known as dual-controlled CMV (DC-CMV), dual-controlled IMV (DC-IMV), and dual-controlled PSV (DC-PSV), have been used successfully.

For nearly 30 years, infants were ventilated with infant-specific, time-cycled, pressure-limited, intermittent mandatory ventilation (TCPL/IMV) breaths. Unlike current microprocessor-controlled ventilators, these TCPL/IMV ventilators used a preset continuous flow of an oxygen-air mixture. The patient could breathe from the flow during spontaneous breaths but was unable to trigger mandatory breaths from the ventilator (Fig. 22-6, A). In this design, a machine-triggered positive pressure breath resulted when the machine's exhalation valve closed, permitting the gas mixture to flow to the patient (Fig. 22-6, B). When a preset inspiratory pressure was reached, the pressure was maintained until the ventilator time-cycled into expiration (Fig. 22-6, C). When the exhalation valve opened, the expiratory phase began. As long as the exhalation valve remained open, a constant flow of the gas mixture passed by the patient's airway and was available for spontaneous breathing. The ventilator's inability to permit patient triggering of mandatory breaths during spontaneous respiratory efforts led to asynchrony in spontaneously breathing patients. Nonetheless, the operation of previous TCPL/IMV ventilators still serves as a simple conceptual model for explaining the fundamental operation of neonatal mechanical ventilators and the major advances in pediatric mechanical ventilation.

Neonates that exhibit asynchrony during TCPL/IMV are at an increased risk for developing intraventricular hemorrhage and possibly pneumothorax. Heavy sedation and paralytic drugs (e.g., pancuronium) may reduce these risks, but these drugs also pose potential complications that may prolong ventilator support. In Fig. 22-7, initial patient efforts (breaths A, B, and C) appear to be synchronous; however, when a mandatory breath (breath D) is delivered on top of a spontaneous breath, the ensuing spontaneous breaths (breaths E, F, and G) are asynchronous. A mandatory breath delivered in the middle of a spontaneous breath can result in breath stacking. When flow triggering is used, mandatory breaths can be delivered between spontaneous breaths, resulting in better patient-machine synchrony and a reduction in WOB and caloric and sedation requirements (Fig. 22-8). Although some current pediatric ventilators still allow the option to use a preset continuous flow during PC-CMV and VC-CMV, machine-triggered TCPL/IMV has been replaced with patient-triggered PC-IMV.

Major improvements in ventilator technology now provide the ability for even the smallest patients to be able to control sophisticated demand flow systems to improve triggering and synchrony with mandatory ventilator breaths. Patients can trigger breaths based on a pressure or flow change that is sensed by the ventilator. Flow sensing is more sensitive and allows better synchronization than pressure triggering in neonates. Additionally, patient triggering may reduce the need for heavy sedation and paralytics during mechanical ventilation. Compared with earlier forms of
nontriggered ventilation, patient-triggered ventilation is associated with a shorter duration of ventilation.

Because all ventilators now provide patient-triggered ventilation, the term PC-IMV has replaced the previously used TCPL/IMV constant-flow mode. As such, synchronized intermittent mandatory ventilation, or SIMV, and assist/controlled, or A/C, have been replaced with IMV and CMV, respectively. Additionally, mandatory breaths denote fully supported ventilator breaths (i.e., PC-CMV, VC-CMV) and can be initiated by the patient or by the ventilator and terminated based on time, whereas spontaneous breaths are always initiated and terminated by the spontaneously breathing patient (i.e., CPAP, PSV). During CMV every breath from the ventilator is mandatory, or fully supported, whereas during IMV the patient can breathe using both mandatory and spontaneous breath types. Each manufacturer may use completely different names to differentiate modes and breath types, and as a result, there is great confusion among clinicians, educators, researchers, and manufacturers about mode classification. Efforts are being made to standardize ventilator mode classification based on the approach used in this discussion.

Another important advancement in neonatal and pediatric ventilators has been the development of the accurate measurement of airflow and tidal volumes in very small patients. This allows possible improved methods for measuring dynamic compliance, static compliance, airway resistance, and airway waveform graphics on mechanical ventilators. A complete understanding of lung mechanics and airway graphics may eliminate a lot of conjecture in managing patients on a mechanical ventilator. Tidal volume and airway graphics are usually obtained by using proximal airway sensors that measure airflow at the connection of the ventilators circuit and the patient's artificial airway. It has been shown that ventilators that do not measure tidal volume with a sensor at the proximal airway produce measurements that are not sufficiently accurate to use for managing the ventilator in neonates. Thus, the most useful neonatal-capable ventilators are those that have proximal airway flow sensors for accurate determination of tidal volume, lung mechanics, and airway graphics. The use of a proximal flow sensor also allows more precise flow triggering and graphics monitoring than those provided at the ventilator valve. It is important to mention that a major limitation of proximal flow sensors is that condensation and secretions can form on the flow-sensing elements. Clinicians must remain weary of this limitation and replace or clean flow sensors when changes in the airway graphics are observed.

Additional features or enhancements can make ventilators more useful in a wide range of clinical situations (Box 22-5). Many ventilators have advanced features that allow clinicians to modify the gas flow within the breath to “fine-tune” or improve patient-ventilator synchrony and gas delivery. The slope, or “rise-time,” setting is an advanced feature that can be used during PC, DC, and PS to adjust the aggressiveness of initial gas delivery at the start of the breath. A fast rise time and rapid pressurization may reduce asynchrony in patients with high flow requirements and a slow rise provides slower and hence more laminar gas delivery during inhalation (Fig. 22-9).

Flow cycling is another feature that allows patients to terminate the breath based on flow rather than on time. Flow cycling is described in greater detail later in this chapter. Sophisticated leak-compensation algorithms are available for invasive and noninvasive ventilation. When this option is activated, loss of end-expiratory...
pressure caused by an ET leak triggers the addition of flow to the ventilator circuit to maintain PEEP constant. Some ventilators have also incorporated leak compensation during inhalation to provide stable volume delivery in the presence of large ET leaks. The potential advantages of this feature are discussed in a later section.

As respiratory monitoring has grown more comprehensive and sophisticated, sensing adapters at the proximal airway have become necessary. With some ventilator models, monitoring of end-tidal CO₂ and flow-volume loops require two airway sensors. These sensors add extra weight to the ET, which can lead to excessive tube movement and possibly accidental extubation. These devices can also add dead space to the system, resulting in excessive accumulation in carbon dioxide at the airway and consequent increases in ventilation requirement. Ventilator and monitor manufacturers have addressed this clinical problem and are including integrated end-tidal CO₂/volume monitoring sensors in the mechanical ventilator.

**Pressure-Control Mode**

This section discusses PC-CMV and other applications of ventilator modes commonly used in pediatric settings (see Chapter 5 for a detailed description of ventilator modes.) Disease-specific management strategies, using these modes, will be described in a later section.

The most widely used mode of ventilation in neonates and pediatrics is PC-CMV. The PC-CMV breath can be triggered by pressure or flow and is terminated based on time. Because pressure is constant, the tidal volume delivery can vary widely as a result of changes in lung mechanics and respiratory effort. PC-IMV is very similar to TCPIMV with some subtle but clinically important differences. It usually is not incorporated into a continuous-flow generator, although a small bias flow might be present to allow flow-triggered (or patient-triggered breaths). The major difference is that inspiratory flow is variable and can be much greater in this mode, resulting in an almost immediate rise to peak pressure. Further, if a patient generates spontaneous inspiratory efforts within the breath, the demand valve opens to provide additional flow to maintain inspiratory pressure constant for the duration of the inspiratory time. Additionally, the demand valve will also provide additional flow in the presence of leaky ETs to maintain airway pressure constant. This mode has long been preferred for pediatric and adult patients in clinical situations in which ventilation or oxygenation (or both) is particularly difficult. Because there is a rapid rise to inspiratory pressure, the mean airway pressure tends to be higher than volume control where peak pressure varies and reaches a maximum in the last part of the breath. The theoretical advantage of PC-CMV lies in the characteristics of its inspiratory phase. Lungs with varying time constants may benefit from an early rise to peak pressure by rapid inspiratory flow and a subsequent period of decreased flow, which allows gas to be distributed more evenly to areas of the lung with both long and short time constants. In this way, improved gas distribution to underventilated areas can be achieved with limited distortion of well-ventilated areas.

Most ventilators that provide PC-IMV also provide pressure-supported breaths for spontaneously breathing patients. This combination, sometimes called mixed-mode ventilation, allows patients to assume the breathing load better when lowering the frequency of mandatory breaths during the weaning phase (Fig. 22-10). The results of one study suggest that the addition of pressure support as a supplement to PC-IMV may play a role in reducing the duration of mechanical ventilation and oxygen dependency in VLBW neonates.

In PC-CMV, minimizing Praw is essential in patients with exceptional oxygenation. The ventilator frequency should be only high enough to reach the desired PaCO₂ and inspiratory pressure is adjusted in increments of 1 to 2 cm H₂O to keep the monitored exhaled tidal volume values within an acceptable range. The I:E ratio initially should be 1:3 to 1:2. Changes in blood gas values often take time with PC-CMV; only a few setting changes are made at one time, and sufficient time is allowed before the patient’s response is evaluated.

**Fig. 22-9** A. Shows a child receiving PC-CMV with a slow rise to peak inspiratory pressure because the slope setting on the Carefusion AVEA is set to 9. B. In the same child, there is a rapid rise to peak inspiratory pressure because the slope setting is set on 1. The set inspiratory pressure is the same for both breaths.

**Fig. 22-10** This graphical waveform shows pressure, flow and tidal volume in a neonate receiving patient-triggered PC-IMV (larger inspiratory pressure breaths) with the addition of PS breaths (smaller inspiratory pressure breaths).
**Inspiratory Pressure**

This section discusses the controls, monitoring systems, and alarms used in pressure-control mode. Some of these principles can also be applied to other modes of ventilation.

While ventilating a child with a manual resuscitator or T-piece device with an airway pressure manometer inline, it is good practice for the clinician to evaluate bilateral lung aeration and chest movement and to note the average inspiratory pressure required. This average is the starting point for placing the child on the ventilator, especially if manual ventilation at this inspiratory pressure has optimized the child's skin color and oxygen saturation. Traditionally, the inspiratory pressure has been adjusted based on adequate chest rise and blood gas values. Today, the patient is connected to the ventilator circuit and exhaled tidal volume is evaluated as a guide for further inspiratory pressure adjustments. This practice has resulted in less need for blood gases because the rate is the primary adjustment for CO₂ elimination. Once the inspiratory pressure is set to deliver a preferred tidal volume, the rate is the primary adjustment for CO₂ elimination. Pressure-volume loops are helpful in setting the optimum inspiratory pressure. The rising inspiratory pressure produces an almost linear increase in volume; therefore, a peak appears in the loop's configuration at the point where inspiration ends and expiration begins. However, if the volume rise of the loop begins to flatten with a continued increase in the inspiratory pressure, over-distention is likely (Fig. 22-11). If this occurs, the inspiratory pressure should be reduced until little or no flattening of the loop occurs. Because lung mechanics can change rapidly, this graphic display should be rechecked routinely and the inspiratory pressure adjusted as necessary.\(^\text{52}\) When the set inspiratory pressure is lowered, PEEP may need to be increased to maintain acceptable oxygenation. However, when PEEP is increased, it is important to observe the effect of this action on the exhaled tidal volume and the pressure-volume loop relationship. (See Chapter 9 for more information on pressure-volume loops.)

**Positive End-Expiratory Pressure**

Positive end-expiratory pressure, or PEEP, is used to establish the FRC and prevent alveolar collapse. In some conditions, such as asthma, increasing PEEP may reduce airway resistance and provide better patient triggering. The appropriate level of PEEP can greatly improve oxygenation, reduce ventilation/perfusion (V/Q) mismatching and transpulmonary shunting, and increase compliance. Because of the transmission of pressure to the intrapleural space, excessive PEEP can increase pulmonary vascular resistance, which can lead to reduced venous return to the heart, reduced cardiac output, and an increase in dead space (see Chapter 13). The increase in dead space alerts the clinician that the PEEP level might be excessive. The patient's PaCO₂ may increase even though the minute ventilation (Vₐ) remains unchanged. High PEEP levels and consequent hyperinflation may contribute to traumatic lung injury, such as pulmonary interstitial emphysema (PIE) and other air-leak syndromes (see Chapter 17). The effects of PEEP should be closely monitored, especially when lung mechanics improve. PEEP usually is set initially at 4 to 7 cm H₂O. PEEP levels above 7 cm H₂O occasionally are necessary, but they should be used with caution in infants who have diseases with obstructive components, such as bronchiolitis or meconium aspiration syndrome (MAS). Careful inspection of chest radiographs for adequate lung inflation and signs of hyperinflation are vital to monitoring the effects of PEEP.

The clinician should consider increasing the PEEP level when the oxygen requirement exceeds an FIO₂ of 0.6 to maintain a PaO₂ greater than 50 mm Hg. Long before this point, however, the chest radiograph may show decreasing lung volumes. Therefore the need for higher PEEP levels may be recognized before worsening ABG values are seen. Patients who undergo surgical procedures that result in high abdominal girths often require higher than usual levels of PEEP to restore baseline lung volumes.

In patients with surfactant deficiency syndromes (i.e., RDS; ARDS), the pressure-volume loop of a positive pressure breath may show a rapid rise in pressure with a delayed rise in volume (Fig. 22-12, A). Increasing the PEEP may result in a more immediate rise in volume for the pressure delivered (Fig. 22-12, B).\(^\text{52}\) This improvement in volume delivery is associated with the critical opening pressure of various lung units. As more PEEP is applied, progressively more lung units may be opened and recruited.

Ventilator graphics, particularly the pressure-volume loop (Fig. 22-13), show how appropriately applied levels of PEEP can improve compliance. Favorable responses to increasing PEEP levels include a shift of the loop to the left, an increase in Vₐ at the same inspiratory pressure, and an increase in PaO₂.\(^\text{56}\)

**Inspiratory Time, Expiratory Time, and Inspiratory-to-Expiratory Ratio**

Lung mechanics must be considered when the Tₑ, expiratory time (Tₑₑ), and I : E ratio are set, especially for patients with surfactant deficiency. These patients are likely to have a low Cₑ with normal Raw; therefore the Tₑ must be short. Because Raw usually is normal in this scenario, allowing extra time for inspiratory gas to traverse the airways is not necessary. However, because compliance is low,
small volumes must be used to inflate the lungs quickly. Because
elastic forces are high, expiration is also relatively fast. Lungs with
these mechanical characteristics are said to have short time con-
stants (see Chapter 1). One time constant is calculated by multiply-
ing the Raw by the Cₜ (Box 22-6). Historically, neonates and
pediatrics with short time constants (e.g., in RDS, ARDS) have
been ventilated using long Tₜ and, hence, higher mean airway
pressure, to promote better oxygenation. Although this approach
may work well in chemically paralyzed and sedated patients, long Tₜ
may cause an inspiratory breath hold to occur when the patient is
breathing spontaneously. In a summary of studies conducted in
neonates with restrictive lung disease, long Tₜ was associated with
a significant increase in air leak and mortality.¹³

A prolonged Tₜ can be identified, using airway graphics, as a
period where inspiratory flow decays to zero and pressure is held
in the lung during a PC-CMV breath (Fig. 22-14). This subtle
phenomenon is often overlooked and is a major cause of asyn-
chrony during PC-CMV. A breath hold can be avoided by reducing
the inspiratory time so that the breath is terminated just before
zero inspiratory flow. Newer ventilators allow the clinician to set
an adjustable flow-cycle parameter during PC-CMV (Fig. 22-15).
Flow-cycling essentially allows an otherwise time-cycled, PC-CMV
breath to cycle to flow, much like a PSV breath, and Tₜ can be
monitored during spontaneous breathing. At times, a patient-
tiggered exhalation or “flow-cycling” can result in a dramatic
reduction in WOB and PaCO₂. Some clinicians will leave the flow-
cycle parameter “on,” whereas others will disable it and use the
previously measure Tₜ as the “new” Tₜ setting during PC-CMV. It
is important to realize that time constants in the lungs can change
rapidly; thus flow-cycling during PC-CMV may change the inspira-
atory time dramatically as compliance is reduced, resulting in
lower mean airway pressure and lower tidal volume delivery.

Conditions that result in airflow limitation generally have longer
inspiratory and expiratory time constants, which can be a factor in
the inability to deliver the desired Vᵢ and Vₑ (see Fig. 22-15) during
PC-CMV. In severe asthma, for example, gas-flow limitation can
affect inspiratory and expiratory flow as well as Vᵢ volume delivery.
Because as much time as possible must be allowed for the expiratory
phase in such clinical situations, the clinician often must keep $T_i$ at 25% to 33% of the total cycle time (TCT). In doing so, the limitation of inspiratory flow may be so great that the flow does not decelerate to zero as it normally does, which means that the inspiratory phase would “time limit,” delivering a smaller $V_T$ than if the flow had been permitted to taper to zero. Such a phenomenon, called “flow chop” by some clinicians, is unavoidable in some situations, especially if a longer TCT (and thus a lower $V_T$) cannot be tolerated. Many clinicians advocate a permissive hypercapnia ventilation strategy in severe reactive airway disease (discussed later). To accomplish this, a small $V_T$ is selected, as well as a low $V_e$, a low rate, an inspiratory
time sufficient to eliminate flow chop, and an expiratory time sufficient to achieve zero or near-zero expiratory flow. However, this strategy and the recommended settings are controversial and may not be suitable for some patients, leaving no alternative but to accept the presence of some flow chop. Because airway dynamics can change quickly and dramatically and changes in $V_T$ and $V_e$ are directly affected, flow chop must be monitored carefully (Case Study 22-3).

Case Study 22-3

**Patient Case—Acute Status Asthmaticus**

A 7-year-old boy in acute status asthmaticus has not responded to treatment consisting of continuous albuterol aerosol therapy, intravenous (IV) Solu-Medrol, IV terbutaline, and two injections of magnesium sulfate. He has just been intubated with a 5-mm ID endotracheal tube and placed on a Carefusion AVEA ventilator. He has been paralyzed and sedated and is being ventilated in the pressure control mode with a 60/40 helium-oxygen mixture. Ventilator settings are: PIP/PEEP = 24/5 cm H$_2$O, respiratory rate = 16 breaths/min, inspiratory time = 0.9 second.

The patient’s expired $V_i$ is 3 mL/kg. The $P_{ET}$CO$_2$ is 92 mm Hg, and the SpO$_2$ is 88%. The RT has increased the increased the inspiratory pressure in increments of 2 cm H$_2$O to 32 cm H$_2$O, but the $V_T$ has not changed. An ABG sample has been sent to the laboratory.

What additional monitoring should the respiratory therapist consider with this patient? What other setting changes should the RT recommend?

See Appendix A for the answer.
Nearly complete equilibration of alveolar pressures ($P_{aw}$) occurs in 3 to 5 time constants (Fig. 22-16). In infant lungs with normal mechanics, equilibration occurs in at least 0.6 second (time constant $\times 5 = \text{(Raw} \times C_l) \times 5 = (30 \text{ cm H}_2\text{O/L/s} \times 0.004 \text{ L/cm H}_2\text{O}) \times 5 = 0.6$ second). Less time is needed for lung inflation in surfactant-deficient lungs, in which the time constant is shorter. Therefore the $T_1$ can be set for a short interval and the respiratory frequency can be set high with less concern for breath stacking and hyperinflation.64

The concept of time constants can easily be related to the clinical situation by evaluating the spontaneous ventilatory pattern of a premature infant with RDS. The patient's spontaneous WOB is high because of low $C_l$ and high alveolar surface tension. The spontaneous rate may be high and $V_t$ low. $T_1$ and $T_2$ are very short, and inspiratory flow is high. Short time constants are responsible for this familiar ventilatory pattern and are considered when the ventilator's $T_1$ and $T_2$ are set. However, ventilator settings should not simulate a patient's spontaneous breathing pattern, particularly when the TCPL mode is used. It is estimated that infants with RDS can have time constants as short as 0.05 second, which means that the ideal $T_1$ is 0.25 second.64 On the other hand, when acute lung disease makes adequate oxygenation difficult, lengthening the $T_1$ and increasing the $P_{aw}$ can increase the PaO$_2$.65

Bronchopulmonary dysplasia (BPD) is an example of a pulmonary disease of infants in which high Raw is a major component. Time constants for BPD are estimated to be as high as 0.5 second.64 The longer time constants associated with this disorder require careful manipulation of ventilator controls to provide for long inflation and even longer deflation times. Although a patient's compliance and Raw cannot be measured precisely, characteristics of the disease are used to guide the clinician in matching ventilator settings with a patient's inherent ventilatory pattern and in promoting better patient-ventilator synchrony.

With time constants in mind, the I:E ratio usually is set between 1:2 and 1:3 in surfactant deficiency syndromes. If an infant's gas exchange does not improve with these ratios, other techniques may be considered, such as high-frequency ventilation. Inverse ratios are rarely used in PC-CMV because of the risk of hyperinflation and lung trauma. Waveform monitoring is useful for determining the most appropriate $T_1$ and $T_2$ ventilator settings. The expiratory flow waveform does not return to baseline before the next positive pressure breath is delivered in patients with increased expiratory resistance (Fig. 22-17). Although treatment (e.g., bronchodilator therapy) may improve the patient's expiratory flow, manipulation of the I:E ratio to extend the $T_2$ may also permit lung emptying before the next breath. Recognizing this problem and taking the appropriate steps to correct it are important for reducing the potential for hyperinflation and lung injury.65

### Tidal Volume

Tidal volume is not a set parameter in the PC-CMV mode. Mechanical $V_t$ depends on $T_2$ lung mechanics, and patient effort. Changes in compliance after administration of exogenous surfactant are almost immediately reflected in direct $V_t$ measurements. In addition, noting trends in an infant's spontaneous $V_t$ is useful for determining readiness for weaning from the ventilator and extubation.

Cuffless ETs are still used in neonates, especially in premature patients. Leaks around the tube are common. Most clinicians consider small leaks (<20%) acceptable and even desirable as an added safety pressure-release site and as assurance that no significant inflammation is present around the tube. When leaks are present, the $V_t$ monitor can be used to assess the difference between delivered and $V_{tinh}$. This loss of volume often is expressed as the percent leak, which can be calculated with the following formula:

$$\text{Percent leak} = \frac{[V_{tinh} - V_{tinh}] \times 100}{V_{tinh}}$$

where $V_{tinh}$ is the inspired $V_t$ and $V_{tinh}$ is the expired (exhaled) $V_t$.

Some ventilators calculate and display the percent leak. Other monitors display $V_{tinh}$ and $V_{tinh}$. Volume monitoring also provides an important safety measure by alerting clinicians to sudden drops in expired $V_t$. A small leak or an obstructed ET is more easily detected when these monitors are used. Low-pressure alarms, although important, are not as sensitive to all alarm conditions involving a reduction of effective ventilation. High pressure and respiratory rate as well as low tidal volume, and low exhaled $V_e$.
may also alert clinicians to serious conditions that arise during ventilation.

**Frequency**

The initial frequency setting can be gauged while the infant is manually ventilated before being connected to the ventilator, just as an initial inspiratory pressure can be determined by clinical assessment. Trying out different frequencies can help determine the initially setting to achieve the best SpO₂ and vital signs. Airway graphics are a very helpful resource in determining the proper frequency setting. Higher rates can result in gas trapping, thus complete exhalation should be noted on flow scalar when setting the initial frequency and with subsequent adjustments. However, a blood gas or a transcutaneous CO₂ monitor that correlates well with PaCO₂ is the standard method used to adjust frequency. Noting the patient’s Vₑ and relating it to the PaCO₂ is important. Once the initial PaCO₂ is known, it, along with the desired PaCO₂, can be used to calculate an appropriate change in Vₑ or frequency. These calculations may not work well for all clinical purposes but they serve well in larger pediatric patients. (These calculations are discussed in Chapter 12.)

Although pulse oximeters have replaced transcutaneous monitors as noninvasive means of monitoring oxygenation in infants, transcutaneous CO₂ monitors can provide trending information about alveolar ventilation. Even though transcutaneous CO₂ may not correlate all of the time, a rapid change in transcutaneous CO₂ could warn clinicians to a serious condition, whereas a blood gas may take time to get results. For instance, a gradual reduction in transcutaneous CO₂ following surfactant administration may alert the clinician to observe exhaled tidal volumes and compliance and possibly wean inspiratory pressures during PC-CMV monitoring of the transcutaneous partial pressure of carbon dioxide (PtcCO₂) gives the clinician valuable baseline and trending information before a switch is made from a conventional ventilator to a high-frequency ventilator. This information, which is particularly valuable in patients with severe lung disease, can be used to stabilize the patient on high-frequency ventilation (see the section on high-frequency ventilation later in the chapter). Many end-tidal CO₂ monitors add excessive dead space and weight to the ventilator circuit adapter. Often an airway flow sensor is already connected, and attaching another sensor creates an unwieldy apparatus, making CO₂ monitors difficult to use in small patients.

**Mean Airway Pressure**

Conventional ventilators do not have a Pₐw setting; rather, this is a monitored parameter that must be closely watched. Increases in Pₐw can greatly improve oxygenation but can also reduce venous return and cardiac output. Pₐw levels greater than 12 cm H₂O have been associated with lung injury. The Pₐw is directly affected byPIP and PEEP, inspiratory hold, frequency, Tᵣ, and flow.

**Inspired Oxygen Concentration**

An F₁O₂ higher than 0.6 is avoided as much as possible in pediatric patients to prevent oxygen toxicity. This concern is even greater for premature infants because of oxygen’s role in developing retinopathy of prematurity. Tissue oxygen delivery is as important as F₁O₂
in ventilator management. Maintaining a hematocrit of more than 40%, even in premature infants, maximizes the blood's oxygen-carrying capacity and augments the oxygenating effects of PEEP, Paw, and FIO₂.

PaO₂ should be maintained above 50 mm Hg in infants and above 70 mm Hg in pediatric patients, but clinicians often accept lower limits, especially when a patient's oxygenation fails to improve despite high Paw values and an FIO₂ of 0.6.

One ventilator (AVEA, CareFusion, Vyasys Corp, San Diego, Calif) has implemented a closed-loop FIO₂ algorithm wherein the ventilator titrates automatically the FIO₂ based on a measured oxygen saturation and preset oxygen range (i.e., 88-92%). This may be a useful system for managing oxygenation in patients, but few trials have evaluated the effectiveness of closed-loop FIO₂ in reducing adverse outcomes in patients during mechanical ventilation.

At the time of this writing, closed-loop FIO₂ was not FDA approved.

Volume Control Mode

Older children and adults have been ventilated with VC-CMV mode over the past several decades. Although this mode was not commonly used for neonates in the recent past, with improvements in ventilator performance and V₅ monitoring, clinicians are now using it in the smallest of patients. In the late 1960s and early 1970s, the Bourns LS-104-150 infant ventilator, a linear-driven piston volume ventilator with an IMV option, was commonly used for infants. However, this practice was hampered by technological limitations, which resulted in air leaks and BPD in neonates. Today most ventilators can target a preset tidal volume as low as 2 mL and measure small volumes with great accuracy. These improvements in technology and the improved understanding of the effects of volume overdistention of the lung as a primary cause of VILI have led clinicians to favor VC-IMV/CMV in pediatric patients with ARDS and premature neonates with RDS. A discussion of preferred settings and management is reviewed in greater detail in the section on Lung-Protective Strategies in Conventional Ventilation.

Volume-targeted ventilation permits V₅, rather than inspiratory pressure, to be set. As such, the measured inspiratory pressure will vary based on lung mechanics and patient effort. T₁ is a function of the set V₅ and inspiratory flow. During VC-CMV, some ventilators require the clinician to set the inspiratory time and the calculated flow will be delivered to obtain the preset V₅, whereas other ventilators require the clinician to set the flow and the T₁ is dependent on the preset flow and volume. The constant flow profile provided during VC-CMV is a “square” waveform. It has been speculated that a square flow profile may not be as effective as a decelerating flow profile when considering gas distribution in the lungs. Based on this, manufactures have incorporated the option to change from a traditional square flow waveform to 50% decelerating flow waveform. Because the flow is calculated or preset, constant flow is frequently associated with asynchrony, especially when the flow is insufficient to meet the patient's inspiratory flow requirements. This can be alleviated by increasing the flow or reducing the T₁ setting. Some ventilator systems incorporate an advanced setting that allows patients to transition to a variable flow pattern during VC to meet higher flow requirements by the patient. The volume-targeted mode can be used with CMV or IMV or VC-CMV and VC-IMV, respectively.

Breaths can be triggered by the patient, by flow or pressure or machine triggered if the patient is not assisting the ventilator. Every volume-targeted breath is a positive pressure breath of the same V₅, flow, and T₁. During VC-IMV, PSV breaths can be added to support spontaneous breaths but during VC-CMV, all the breaths are supported with the preset V₅. Patients receiving VC-CMV should be monitored closely for clinical signs of hypocapnea and hyperinflation, especially when the patient is agitated or auto-triggering the ventilator due to large and ET tube leak. In theory, V₅ does not vary with changing lung compliance or airway resistance during VC-CMV; however, V₅ may decrease if the ventilator cannot correct for volume losses resulting from gas compression in the patient circuit. Delivered V₅ volume may also be affected by leaks from cuffless ETs. When ventilating a larger patient, the volume loss may be negligible; however, in a small child or infant it may be a significant portion of the delivered V₅. Failure to consider this volume loss may result in hypoventilation and hypercapnia of the patient.

Infants who undergo cardiothoracic or abdominal surgery often are placed on VC-IMV or VC-SIMV because changes in Cₕ and abdominal distention do not affect V₅ delivery. Unlike pressure-controlled ventilation, there is a slower rise to the peak inspiratory pressure during volume-controlled ventilation and hence, lower mean airway is obtained for the same tidal volume.

Transitioning from pressure-controlled ventilation to volume-controlled ventilation may be preferable in patients that have hemodynamic compromise or do not tolerate higher mean airway pressure.

Pressure-Support Ventilation

Pressure-support ventilation is strictly a spontaneous mode or form of “continuous spontaneous ventilation (CSV) that is used to augment a patient’s V₅ by means of a clinician-set inspiratory pressure. As mentioned, PSV can also be used during IMV to assist with weaning. During PSV, the patient controls frequency and T₁, and patient triggering is based on either pressure or flow. Cycling occurs when flow from the ventilator decays to a preset point. If the cycling flow is not reached because of a leak around the ET, a backup time-cycling mechanism activates. Further, if a patient becomes apneic, based on a preset apnea interval, the ventilator will provide backup ventilation.

Pressure-support ventilation is very useful in pediatric patients who have stable ventilatory drives and acceptable ventilatory mechanics but who must remain intubated for other reasons. These patients may show asynchrony with mandatory breaths and appear more comfortable with PSV. The small diameter of pediatric ETs can significantly contribute to a patient’s WOB. The goal of PSV in this situation is to provide inspiratory pressure sufficient to overcome tube resistance but also to allow the patient's lung and chest wall mechanics to determine V₅. Analysis of a patient’s pressure-volume and flow-volume loops while on PSV provides some indication of the effort required to overcome artificial Raw (Fig. 22-18). Although some patient effort may be desirable to condition ventilatory muscles, other considerations may require minimization of ET resistance by increasing the level of pressure support (Fig. 22-19).

Initially a higher level of pressure often is needed to enable these patients to achieve V₅ in the range of 4 to 7 mL/kg. Over time the PSV level can be reduced if the patient maintains a satisfactory V₅, respiratory rate, and SpO₂. Some clinicians periodically reduce the pressure below the minimum level as a means of reconditioning ventilatory muscles. Once pressure support can be reduced to a minimum level appropriate for the ET's internal diameter, most
patients can breathe spontaneously through the tube until extubation.

Small-diameter ETs, particularly those less than 4.5 mm, may provide excessive resistance during pressure support, and pressurization of the ventilator circuit may occur before sufficient flow enters the patient’s airway (see Fig. 18-9). When that happens, rapid deceleration of flow may prematurely end the inspiratory phase; this is sometimes called premature pressure support termination (PPST). With PPST the desired augmentation of VT does not occur, and patient-ventilator asynchrony may result. When PPST is suspected, a slower rise time can be adjusted, and this may reduce or eliminate it. This problem was frequently seen in the past when adult ventilators were used in pediatrics but is less of a problem when using ventilators that are designed for infants through adults.

Another common problem with PSV in pediatric patients is failure to flow cycle because of ET or tracheostomy tube leaks. The clinician has some control over the length of backup time cycling on most ventilators providing PSV. Establishing and relying on time cycling rather than on flow cycling is sometimes desirable if the tube leak is excessive that the patient cannot trigger the next breath. If both triggering and cycling are problems, the artificial airway may need to be changed to a larger size so that the leak is reduced. Cycling issues are rarely sufficient reason to change to a cuffed airway (Case Study 22-4).

**Case Study 22-4**

**Recommended Changes in Ventilator Settings**

A 1-month-old prematurely born baby boy with a diagnosis of respiratory syncytial virus (RSV) pneumonia is being ventilated with the PC-CMV. The patient’s initial measured VT was about 5 mL/kg with a respiratory rate of 40 to 60 breaths/min, the SpO2 was 95% on an FIO2 of 0.3, and VE was 0.28 L. Over several hours VT diminishes to about 2 to 3 mL/kg, and the respiratory rate increases to over 100 breaths/min. The SpO2 decreases to about 92%, but the VT remains unchanged. What change in ventilator settings is necessary for this patient?

See Appendix A for the answer.

**Dual-Controlled Mode**

The dual-control mode is an adaptive form of pressure-controlled ventilation that can also be used with CMV, IMV, and PSV breaths. It combines the best features of pressure and volume modes to provide a minimum VT during ventilation. Dual-control breaths can be patient triggered based on flow or pressure or machine-triggered if the patient does not have a spontaneous respiratory effort. The dual-control breath can be cycled to exhalation based on time or once the peak flow has decelerated to a preset value. The VT is preset, and the inspiratory pressure level will vary based on...
changes in patient effort, respiratory system mechanics, and measured $V_t$. Dual-control modes provide variable, decelerating inspiratory flow waveforms. The ongoing inspiratory pressure adjustments are servo-controlled based on volume and compliance measurements made at the proximal flow sensor or back at the ventilator. Adaptive algorithms vary based on the different modes provided by manufacturers. Depending on the mode, the inspiratory pressure level will readjust on a breath-to-breath, or “within the breath” basis to target a minimum $V_t$. The ventilator may take time to incrementally adjust the inspiratory pressure level to target the tidal volume, especially when the patient is breathing erratically.

This can result in disparities between the set and delivered $V_t$. Many manufacturers have incorporated a preset volume limit, which limits excessive $V_t$ delivery during dual-control ventilation. An important concept that some clinicians fail to recognize during dual control is that tidal volume may decrease if the ventilator cannot correct for volume losses resulting from gas compression in the patient circuit. Delivered $V_t$ may also be affected by leaks from cuffless ETs. It may be very difficult for dual-control modes to provide a precise tidal volume with ET leaks of more than 30%. In these cases, clinicians may change the mode or reintubate with a larger ET tube. The latest ventilator manufacturers have incorporated new algorithms to target a “theoretic” delivered $V_t$, after the leak has been calculated, and adjust inspiratory pressure based on this value, rather than the measured inspiratory or expiratory $V_t$.

The following sections provide only a brief explanation of commercially available dual-control modes. Further descriptions of these modes can be found in Chapters 5 and 23.

**Pressure-regulated volume control.** The most widely used form of dual control ventilation in neonates and pediatric patients is pressure-regulated volume control (PRVC). PRVC is commonly used in patients with CMV or IMV breath types. $V_t$, frequency, PEEP, and $T_i$ are preset by the operator. The ventilator initially performs a test breath sequence, which measures dynamic or static system compliance. Subsequent adjustments in pressure or $V_t$ are made on the basis of the previous breath or a historical average of breaths. Some ventilators initiate a “test breath” sequence during PRVC by implementing a brief inspiratory pause during a volume-controlled breath. The static pressure measured during the pause will be the pressure control level for the next breath. The following breaths will increase or decrease the pressure control level by a maximal value of 3 cm H₂O to try to achieve the set $V_t$ with the lowest possible inspiratory pressure (Fig. 22-20). Within a few sequential breaths, the $V_t$ goal may be reached. Certain conditions can restart the test breath sequence for optimal accuracy, including high-pressure limitation, $V_t$ in excess of 150% of the set $V_t$, and after-settings changes. It should be noted that during PRVC, the inspiratory pressure is usually adjusted based on the monitored inspiratory tidal volume. In the presence of substantial ET leaks and patient effort, PRVC may reduce the level of support provided, which may result in underinflation and consequent hypercapnia. Alarms should be adjusted properly, and patients should be monitored frequently for signs of respiratory distress. Additionally, in some ventilators, inspiratory tidal volumes are measured at the airway, using a proximal flow sensor, but inspiratory pressure is being regulated based on a volume measurement at the ventilator. In this case, the $V_t$ may need to be readjusted in neonates with reduced compliance to eliminate underventilation from compressible volume loss in the circuit.

**Machine volume with volume bracketing.** Another form of dual-control ventilation that is used primarily in neonatal patients, with the CareFusion AVEA, is machine volume (MV) with volume bracketing. Machine volume uses an intrabreath pressure adjustment to target volume. The breath can start out as pressure control, using a variable decelerating flow waveform, and transition to a volume-targeted, constant square waveform, within the same breath to guarantee a minimum $V_t$. The ventilator flow control valve measures compliance every 2 msec within the breath and can increase or decrease the pressure adjustment within this time by manipulating the flow if the delivered volume is not being met or if the patient requires more flow. The ventilator calculates a target flow rate on the basis of the minimal volume set at the ventilator and the inspiratory time. The breath begins as a pressure-controlled breath with a variable-decelerating flow signal. Once the minimal $V_t$ has been met, the breath is terminated at the preset inspiratory time and ends as a pressure-controlled breath. If the minimal $V_t$ goal has not been delivered, then the ventilator transitions from a decelerating flow to a continuous flow signal to reach the $V_t$ goal within the preset inspiratory time. The high-pressure limit must be set appropriately to protect against high pressure. The operator can set a volume-limit feature, which will terminate the breath once this preset volume is exceeded either at the proximal flow sensor or at the ventilator flow-control valve. This mode is similar to volume- assured pressure support (VAPS) and pressure augmentation but different in that the inspiratory time will not by increased to deliver the set $V_t$. The clinician must set an uncorrected minimal $V_t$ when using this mode in neonates. This includes the volume of gas delivered to the patient as well as the volume of gas delivered to the ventilator circuit.

**Volume-assured pressure support.** Volume-assured pressure support (VAPS) is similar to MV, PRVC, or volume support, depending on how it is set. Available on the Bird V.I.P. Gold models (CareFusion, Viasys Corp, San Diego, Calif), VAPS is a dual-control mode that guarantees that a pressure-control breath, a pressure-supported breath, or a time-cycled, pressure-limited breath will target a preset volume. During a VAPS breath, $V_t$ may be augmented by extending the inspiratory phase at the set flow.
for a period beyond the point that flow otherwise would terminate. When the ventilator determines that the delivered volume has not reached the set target, it allows flow to decelerate to its minimum set point; then, rather than terminating, the set flow continues over a slightly extended T,
causing inspiratory pressure to rise. The breath therefore is augmented to the desired V,
An augmented breath essentially transitions from a PC-CMV mode to VC-CMV within a single breath. With VAPS, electronic extension of the inspiratory phase occurs only if the microprocessor determines that the pressure settings alone cannot deliver the preset V,

**Volume guarantee.** Volume guarantee is yet another variation of PRVC or volume support is used only in neonates. Available on the Dräger Babylog 8000plus (Dräger Medical, Inc. Telford, Pa.), the volume guarantee setting allows a set V,
target while maintaining either the pressure-control mode or PSV mode and its characteristic waveforms. Volume guarantee can be used in all the patient-triggered modes, and the clinician can control both the pressure and volume limits. Similar to time-cycle, pressure-limited/IMV ventilators, the operator must set a continuous flow to maintain pressure and V,

delivery. This setting may need to be readjusted throughout the ventilator course with changes in lung mechanics and ET leaks.

The microprocessor assesses an eight-breath historical average of expired V,
and will increase pressure on the basis of these measurements up to the pressure limit to deliver the target volume. If lung mechanics improve dramatically, then the ventilator will terminate breath delivery if the delivered V,
ceeds 130% of the set V,
Pressure will also warn as the result of improving compliance, based V,
on the breath average. Because the ventilator makes manipulations on the basis of expiratory V,
this mode can correct for compressible volume loss of inspired gases and small ET leaks and is useful in the neonatal population. The practitioner should exercise some caution when using this mode with excessive ET leaks because there are concerns that this system will falsely underestimate the actual V,
delivered to the lung and overcompensate the subsequent breaths with excessive V, 

When volume guarantee is used according to accepted guidelines, the inspiratory pressures required to provide effective ventilation have been statistically lower than those used without volume guarantee (Case Study 22-6).  

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**Volume-Support Ventilation**

Volume-support ventilation (VSV) is well suited to infants and pediatric patients. Its use in infants is similar to PSV in that both ventilator triggering and cycling are patient controlled. However, VSV has additional features that may make it preferable to PSV. In most ventilator models, VSV targets a preset V,
or V, or both, whereas PSV does not. If apnea occurs, many ventilators switch automatically to a mode with a mandatory rate (e.g., PRVC, PC-CMV, or VC-CMV). The ventilator measures changes in compliance, such as might occur after administration of surfactant, and automatically adjusts the required PIP. This is essentially a self-weaning mode. However, as with other spontaneous modes, sizeable ET and tracheostomy tube leaks make VSV difficult to use.

In pediatric patients, VSV can be used instead of PSV. The advantages are essentially the same as for infants: the target V, is maintained, and self-weaning is possible. Muscle reconditioning can be promoted by reducing the target volume; this allows pressures to decrease and requires the patient to participate actively if a higher V, is to be achieved by patient effort. Some practitioners prefer switching to CPAP or PS modes for reconditioning periods.

**Airway Pressure Release Ventilation**

Improvements in exhalation valve performance have made possible new forms of ventilation. Airway pressure release ventilation (APRV) mode is similar to inverse I:E ratio ventilation, a mode previously used in patients to promote higher mean airway pressures and improve oxygenation. APRV differs from this approach by allowing spontaneous breathing throughout the entire respiratory cycle; hence, less sedation or paralytics is required. The mode has been referred to as “CPAP with releases” (Fig. 22-21). The clinician sets a high pressure (P,high) slightly greater than the measured mean airway pressure, inspiratory pressure, or plateau pressure during conventional ventilation and the low pressure (P,low) is set between 0 and 5 cm H2O. The frequency controls the rate of rapid pressure releases from the P,high and P,low, which, in combination with spontaneous ventilation, aids in alveolar ventilation and breathing at P,high allows recruitment of air spaces. The P,high is held in the lung for up to 2 seconds for neonates and 4 seconds for pediatrics.  

Spontaneous breathing at a higher pressure not only aids in alveolar recruitment but, through the application of pleural pressure change, improvements in the distribution of lung volume to diseased lung
units may improve FRC and pulmonary compliance. APRV has been used in neonatal, pediatric, and adult forms of respiratory failure, but few studies have been performed in neonates and pediatrics to ascertain specific management protocols.

**Neurally Adjusted Ventilatory Assist**

Neurally adjusted ventilator assist (NAVA) allows the patient full neurologic control of the triggering, magnitude, and timing of the mechanical support provided, regardless of changes in respiratory drive, mechanics, and muscle function. NAVA uses a nasogastric tube with specialized sensors that obtain signals from the electrical activity of the diaphragm to control the timing and pressure of the ventilation delivered. In theory, this form of triggering and support is particularly useful in patients with severe gas trapping and auto-PEEP because it bypasses the effort required in these patients to trigger a ventilator breath. This form of triggering is not affected by leaks and secretions; therefore, autocycling and hypocapnia in newborns can be avoided. However, this modality is invasive and placement of the nasogastric tube must be evaluated to ensure proper function of this modality. Noninvasive NAVA is now FDA approved and is being evaluated in neonates and pediatrics. More information about NAVA can be found in Chapter 23.

**Lung-Protective Strategies in Conventional Ventilation**

As mentioned in previous sections, avoiding or limiting the amount of time a patient is exposed to invasive mechanical ventilation is the primary means of avoiding VILI. Premature neonates are particularly susceptible to developing VILI because the lungs are fluid-filled, critically underdeveloped, and lack mature surfactant. Additionally, the pliable chest wall of neonates is less able than ossified chest walls in larger pediatric patients to limit lung over-inflation. Thus, the goal of any lung-protective strategy is to:

1. Avoid repetitive opening and closing of small airways (atelectrauma)
2. Limit overinflation during inhalation (volutrauma)
3. Reduce gas trapping during exhalation (auto-PEEP)
4. Alleviate pulmonary inflammation (biotrauma)

The standard approach to providing the best lung-protective strategy in neonates embraces low \( V_T \) or pressures and higher PEEP settings, or an “open lung approach.” However, it is also important to realize that different neonatal lung diseases warrant different approaches. Table 22-1 provides some evidence-based lung-protective strategies for initiating and managing infants with different lung disorders during neonatal mechanical ventilation.

As previously discussed, compelling evidence now suggests that “volutrauma” created by excessive volumes, and not necessarily “barotraumas,” is chiefly responsible for instigating VILI. Even short-term exposure to volutrauma during mechanical ventilation initiates lung inflammation in premature infants, even after only a few minutes of manual resuscitation. Ventilation for 15 minutes with a \( V_T \) of 15 mL/kg has been shown to cause an injurious process in the preterm lung. As few as three overdistending breaths at birth have been shown to compromise the therapeutic effect of subsequent surfactant replacement in an animal model of prematurity. Critical underinflation, using small \( V_T \) (atelectrauma) can also contribute to VILI. Further, VILI can put premature neonates with RDS at a greater risk for arrest lung growth and development.

Over the last decade, volume-targeted strategies have been at the forefront of clinical investigation. Volume-targeted ventilation strategies, using a preset \( V_T \), are usually implemented using dual-control or volume-control modes, whereas some clinicians still prefer to use pressure control and guide the inspiratory pressure based on measured \( V_T \).

In a recent review of all clinical trials comparing pressure-targeted to volume-targeted modes, neonates supported with volume-targeted modes had significantly lower duration of ventilation, pneumothorax, hypocapnia, severe intraventricular hemorrhage, periventricular leukomalacia, and the combined outcome of death or BPD than infants supported with pressure-control modes.

In the acute phase of lung disease, it has been suggested that the initial strategy should use CMV mode rather than IMV mode to deliver volume-targeted breath types, so that every breath that the infant receives is volume-targeted and without PSV. With use of IMV, infants were shown to be more tachypneic and to have faster heart rates and consistently lower oxygen saturations, suggesting substantially higher WOB compared with VC-CMV. During weaning and when applicable, it has been suggested that volume-targeted strategies be implemented using PS, so that infants can determine their own inspiratory time.

At present, it is unclear what the absolute target \( V_T \) or target “range” should be used in infants and whether this \( V_T \) needs to be adjusted according to varying levels of disease severity. Generally, the consensus among clinicians is to use \( V_T \) target around 4 to 6 mL/kg in low-birth-weight (LBW) neonates. One study evaluated lung injury response in 30 preterm infants with RDS using \( V_T \) of 3 mL/kg or 5 mL/kg. The 3 mL/kg group showed significantly higher levels of lung inflammation and longer duration of ventilation than the 5 mL/kg. A \( V_T \) target of 3 mL/kg has also been associated with increased alveolar dead-space, tachypnea and higher transcutaneous carbon-dioxide in pre-term infants compared to higher \( V_T \) targets (5 mL/kg).

Larger infants and pediatric patients with acute lung injury and ARDS are susceptible to lung injury and hyperinflation when placed on mechanical ventilatory support. The most common causes for respiratory distress in these patients are pneumonia, bronchiolitis, trauma, seizures, sepsis, and pulmonary edema. Studies have reported reduced mortality in adults when
### Lung-Protective Ventilation Strategies for Neonatal Lung Disorders

<table>
<thead>
<tr>
<th>Lung Disease</th>
<th>Ventilator Settings</th>
<th>Blood gas or SpO₂ targets</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Respiratory distress syndrome (RDS)</strong></td>
<td>1. PC, VC, or DC ventilation to target tidal volume 4-6 mL/kg</td>
<td>1. pH 7.25-7.35</td>
</tr>
<tr>
<td></td>
<td>2. Rapid rates ≥60 breaths/min</td>
<td>2. PaCO₂ 45-55 mm Hg</td>
</tr>
<tr>
<td></td>
<td>3. Moderate PEEP (4-5 cm H₂O)</td>
<td>3. PaO₂ 50-70 mm Hg</td>
</tr>
<tr>
<td></td>
<td>4. Short inspiratory time 0.25-0.4 s</td>
<td>4. SpO₂ 88%-94%</td>
</tr>
<tr>
<td><strong>Meconium aspiration syndrome (MAS)</strong></td>
<td>1. PC, VC, or DC ventilation with lowest PIP to maintain adequate chest excursion</td>
<td>Without PPHN</td>
</tr>
<tr>
<td></td>
<td>2. Relatively rapid rates (40-60 breaths/min)</td>
<td>1. pH 7.3-7.4</td>
</tr>
<tr>
<td></td>
<td>3. Moderate PEEP (4-5 cm H₂O)</td>
<td>2. PaCO₂ 40-50 mm Hg</td>
</tr>
<tr>
<td></td>
<td>4. Short inspiratory time to allow exhalation time (0.5-1 s)</td>
<td>3. PaO₂ 70-80 mm Hg</td>
</tr>
<tr>
<td></td>
<td>5. Sedation, neuromuscular paralysis, and inhaled NO (20 ppm)</td>
<td>4. SpO₂ &gt;90%</td>
</tr>
<tr>
<td><strong>Congenital diaphragmatic hernia (CDH)</strong></td>
<td>1. PC, VC, or DC ventilation with lowest PIP to maintain adequate chest excursion</td>
<td>With PPHN</td>
</tr>
<tr>
<td></td>
<td>2. Rapid rates (40-80 breaths/min)</td>
<td>1. pH 7.30-7.4</td>
</tr>
<tr>
<td></td>
<td>3. Moderate PEEP (4-5 cm H₂O)</td>
<td>2. PaCO₂ 35-45 mm Hg</td>
</tr>
<tr>
<td></td>
<td>4. Short inspiratory time (0.3-0.5 s)</td>
<td>3. PaO₂ 80-100 mm Hg</td>
</tr>
<tr>
<td></td>
<td></td>
<td>4. SpO₂ &gt;95%</td>
</tr>
<tr>
<td><strong>Persistent pulmonary hypertension of the newborn (PPHN)</strong></td>
<td>1. PC, VC, or DC ventilation with lowest PIP to maintain adequate chest excursion</td>
<td>1. pH 7.35-7.4</td>
</tr>
<tr>
<td></td>
<td>2. Higher rates (50-70 breaths/min)</td>
<td>2. PaCO₂ 30-40 mm Hg</td>
</tr>
<tr>
<td></td>
<td>3. Low PEEP (3-4 cm H₂O)</td>
<td>3. PaO₂ 70-100 mm Hg</td>
</tr>
<tr>
<td></td>
<td>4. Inspiratory time (0.3-0.5 s)</td>
<td>4. SpO₂ &gt;95%</td>
</tr>
<tr>
<td></td>
<td>5. Inhaled NO (20 ppm)</td>
<td></td>
</tr>
<tr>
<td><strong>Bronchopulmonary dysplasia</strong></td>
<td>1. PC, VC, or DC ventilation to maintain tidal volume (5-8 mL/kg)</td>
<td>1. pH 7.25-7.35</td>
</tr>
<tr>
<td></td>
<td>2. Slow rates (20-40 breaths/min)</td>
<td>2. PaCO₂ 45-55 mm Hg</td>
</tr>
<tr>
<td></td>
<td>3. Moderate PEEP (4-6 cm H₂O)</td>
<td>3. PaO₂ 50-70 mm Hg</td>
</tr>
<tr>
<td></td>
<td>4. Inspiratory time (0.4-0.7 s)</td>
<td>4. SpO₂ range</td>
</tr>
</tbody>
</table>

PC, Pressure-control ventilation; VC, volume-controlled ventilation; DC, dual-controlled ventilation; PIP, peak inspiratory pressure; PEEP, positive end-expiratory pressure.


Lung-protective strategies are used.84,85 Reduction of barotrauma, volutrauma, and atelectrauma are thought to be the reasons (see Chapter 17).86 Repeated collapse and inflation result in stress injury to alveolar and pulmonary vascular tissue and loss or alteration of surfactant.87

Adult studies have had a dramatic impact on the management of pediatric patients with ARDS. Central to these strategies are the use of a V̇₂ less than 6 mL/kg, plateau pressures (Pplat) less than 30 cm H₂O, and appropriately levels of PEEP in patients with ARDS. PEEP itself has been shown to have lung-protective effects during mechanical ventilation (Case Study 22-7).72

Mechanical ventilation has the potential to create dynamic hyperinflation (auto-PEEP) in patients affected by diseases of airflow limitation (e.g., asthma, bronchiolitis, or ARDS). These patients often have a prolonged expiratory time because of early collapse or obstruction of smaller airways. As auto-PEEP dynamic hyperinflation occurs, trapped air increases in the lung and peak pressures gradually increase during VC-CMV in a spontaneously

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**Interpretation and Response to Monitored Data**

A 7-month-old girl with a diagnosis of bronchiolitis is being ventilated. The machine’s pressure control settings are PIP = 26 cm H₂O, PEEP = 6 cm H₂O, inspiratory time = 0.8 second, and respiratory rate = 16 breaths/min. The Fio₂ is 1.0. ABG values are: PaO₂ = 55 mm Hg, PaCO₂ = 73 mm Hg, and pH = 7.19. An inline Cosmo Plus monitor shows a V̇CO₂ of 83 mL/min. (See Chapter 11 to review volumetric CO₂ monitoring.)

Guided by chest radiographic findings, the attending physician and the respiratory therapist decide to increase the PEEP to 8 cm H₂O. Soon after making the change, they note that the V̇CO₂ has risen and is now at 85 mL/min. What action should the physician and respiratory therapist take?

See Appendix A for the answer.
breathing patient. In assisted ventilation, WOB usually increases. The lung-protective strategy for minimizing the effects of both VILI and dynamic hyperinflation is to use a lower \( \dot{V}_{\text{r}} \), appropriate PEEP levels, and low \( P_{\text{plateau}} \) and to allow permissive hypercapnia (i.e., an increased \( \text{PaCO}_2 \)). Maintaining adequate PEEP, low inspiratory pressures (\( P_{\text{plateau}} < 30 \text{ cm H}_2\text{O} \)), and low volumes also reduces alveolar shear injury and overdistention. Additionally, using a short \( \text{T}_1 \) and prolonging the expiratory phase of each mechanical breath allow more time for exhalation but result in a low respiratory rate. Incorporation of all these strategies usually makes an increase in \( \text{PaCO}_2 \) unavoidable.

Extensive experience has shown that ventilated patients usually tolerate moderate hypercapnia and often some degree of hypoxemia if the patient does not experience shock, hemodynamic complications, or anemia during the clinical course. Patients with severe cardiac disease or elevated intracranial pressure are not good candidates for permissive hypercapnia. Experience has shown that permitting the \( \text{PaCO}_2 \) to rise has little deleterious effect as long as the pH is maintained above 7.2. As mentioned earlier, maintaining a higher than normal \( \text{CO}_2 \) may actually have an anti-inflammatory effect. (NOTE: Inflammation is the biochemical complication associated with overstretching of the lung [see Chapter 17].)⁸⁸-⁹¹

### HIGH FREQUENCY VENTILATION

Emerson⁹² introduced the first high-frequency ventilator in 1959, and many attempts have been made since to apply various forms of this type of ventilation to a wide range of patients. With technological advances, sophisticated devices have been introduced and continue to improve. Interest in high-frequency techniques for neonates was sparked primarily by two complications of conventional mechanical ventilation: pulmonary air leaks and the development of BPD. Before high-frequency ventilators became widely accepted, about 24% of infants with RDS who required ventilatory support developed air leaks.⁹³ Among LBW infants who survived RDS, 25% to 33% eventually developed BPD.⁹⁴

Minton et al⁹⁵ used the term pulmonary injury sequence (PIS) to describe the issue of prematurity and pulmonary disease, or the “continuum of disease.” The continuum of PIS includes RDS, PIE, pulmonary air leak syndrome, oxygen toxicity, and BPD. The extent to which high-frequency ventilation can reduce the incidence of PIS remains unclear. However, the consensus is that the high pressures sometimes used with conventional ventilation are contributory factors. With high-frequency techniques, lung-volume recruitment can be accomplished with a higher \( \text{Paw} \) than with conventional ventilation. Even at a higher \( \text{Paw} \), lung injury is less likely because high peak pressures can be avoided.

High-frequency ventilation can be used in conjunction with surfactant therapy. Studies have demonstrated that lung injury can be reduced with high-frequency ventilation if early recruitment of optimal lung volumes is achieved and maintained after surfactant administration.⁹⁵ This has prompted some clinicians to apply an early intervention strategy to the management of premature infants: high-frequency ventilation is initiated and the first dose of exogenous surfactant is given within the first few hours of life.

Another problem with conventional ventilation, not only in LBW infants but also in older pediatric patients, is ineffective gas exchange despite extremely high settings. This most often occurs in acute lung injury (ALI) and is clearly attributable to the limitations of conventional devices. Experience with high-frequency techniques has shown that improved gas exchange is possible without excessive \( \text{Paw} \), not only in newborns but also in older children and adults (Box 22-7; also see Chapter 23).

### Indications for High-Frequency Ventilation

High-frequency ventilation (HFV) should be considered for patients with heterogeneous lung disease (e.g., ALI/ARDS) if the \( \text{Paw} \) on conventional ventilation exceeds 15 cm H₂O. A change from conventional ventilation to HFV may be seriously considered at a lower \( \text{Paw} \) if the patient’s clinical picture is worsening and the settings on the conventional ventilator are rising. HFV should also be used as an early intervention (i.e., before high conventional settings are used) for premature infants or patients who have air-leak syndromes. Patients with an oxygen index of 40 *(which in some centers meets the criterion for extracorporeal life support) should have a trial of HFV if possible. A trial of HFV also may be considered for patients with sepsis, persistent pulmonary hypertension of the newborn, or congenital diaphragmatic hernia when a high \( \text{Paw} \) is required for effective alveolar ventilation on a conventional ventilator.

### Contraindications and Complications of High-Frequency Ventilation

No absolute contraindications to HFV have been reported, but patients with obstructive airway disease (e.g., asthma) may not be good candidates for some high-frequency techniques because of the risk of overinflation. Overinflation of one or both lungs is a possible complication with any patient receiving HFV. Overinflation can occur as a consequence of inadequate lung unit emptying or remarkably fast reductions in alveolar surface tension that dramatically reduce compliance.

A chest radiograph should be taken within 2 hours of initiation of HFV and at least daily thereafter to check for lung hyperinflation. Frequent radiographs may be necessary for patients at greater risk for overinflation. Placement of the ET tip is checked on every chest radiograph because the position of the tube can affect lung volumes when high-frequency techniques are used.

### Box 22-7 Conditions for which High-Frequency Ventilation Is Used in Infants and Children

- Homogenous lung disease requiring conventional \( \text{Paw} \) over 15 cm H₂O
- Respiratory distress syndrome (RDS)
- Pneumonia
- Aspiration syndromes
- Pulmonary hemorrhage
- Acute respiratory distress syndrome (ARDS)
- Persistent pulmonary hypertension of the newborn (PPHN)
- Air leak syndromes
- Pulmonary interstitial emphysema
- Pneumothorax/bronchopleural fistula
- Pneumomediastinum
- Pneumoperitoneum
- Pulmonary hypoplasia
- Impaired cardiac function
- Bronchoscopy and airway-thoracic surgery
Focal obstruction of the lungs caused by mucus plugging is a potential complication of HFV. Plugging is not necessarily caused by the high-frequency technique; rather, the small \( V_t \) cannot traverse plugging obstructions as well as the higher \( V_t \) delivered in conventional ventilation. Loss of chest movement sometimes is seen when an obstruction develops. Infants with meconium aspiration and other aspiration syndromes may require frequent and aggressive suctioning with ET lavage and chest vibration. However, mucus plugging rarely responds to suctioning, and a brief period on conventional ventilation may be necessary. Increased mucus production in the airways is associated with overdistention and volutrauma and could be problematic if conventional ventilation was used before HFV. Mucus plugging can be caused by inadequate humidification, especially if prolonged manual ventilation took place with a heat and moisture exchanger humidifier.

Impaired cardiac output has been observed as a complication of HFV, particularly in HFOV and with techniques that require high lung volumes and \( P_{aw} \). In addition, crystallloids and colloids are more often necessary over the first 24 hours in these situations than they are with conventional ventilation. Infants, in particular, depend on sufficient intravascular volume for adequate pulmonary perfusion and left atrial filling. Monitoring of the blood pressure (BP), heart rate (HR), and central venous pressure for adverse hemodynamic effects is very important. Echocardiograms are useful for evaluating and maximizing myocardial function and blood volume status.

Intraventricular hemorrhage (IVH) has been reported to be higher in premature infants receiving HFOV than in those receiving conventional ventilation. Presumably this is due to elevated intrapleural pressure and fluctuations in cerebral vascular pressures. Fewer cases of IVH are seen with the combination of HFOV and surfactant therapy than with conventional treatment. Some have suggested that a low \( PaCO_2 \) is a primary cause of IVH in premature infants. A possible explanation is that HFV can dramatically reduce the \( PaCO_2 \) before the clinician is aware of this development.

High-Frequency Ventilation Techniques

As its name implies, HFV is a form of mechanical ventilation that uses high respiratory rates, or frequencies. Frequencies usually are specified in hertz (Hz) or cycles per second; 1 hertz equals 60 cycles/min or 60 “breaths”/min. Under guidelines established by the U.S. FDA, HFV is any form of mechanical ventilation in which the breath frequency exceeds 150 breaths/min. HFV has evolved into five basic types: high-frequency positive-pressure ventilation, high-frequency flow interruption, high-frequency percussive ventilation, high-frequency oscillatory ventilation, and high-frequency jet ventilation. Each type has been somewhat successful in improving outcomes in the management of severe lung disease.

High-Frequency Positive-Pressure Ventilation

High-frequency positive-pressure ventilation (HFPPV) is a modified form of conventional ventilation that uses high frequencies and low \( V_t \) values. HFPPV usually is delivered by conventional ventilators with low-compliance circuits. Until jet ventilators and oscillating devices became readily available, HFPPV was a reasonable alternative for LBW infants with RDS when the combined problems of severe hypoxemia and respiratory acidosis did not respond to more conventional methods. HFPPV also was effective for pediatric patients with surfactant deficiency syndromes. This type of ventilation, which was developed by Sjöstrand et al. in the late 1970s, originally was intended to minimize the cardiovascular effects of PPV. It was discovered that HFPPV sometimes could also improve gas exchange while keeping airway pressures \( (P_{aw}) \) lower than would be with the low frequency/high \( V_t \) technique. HFPPV does not technically fit the FDA definition of HFV because it uses frequencies up to 150 breaths/min. Rates up to this limit are attainable on most of the conventional ventilators currently in use.

Two potential problems are associated with HFPPV: (1) the high rate and short \( T_i \), may prevent adequate \( V_t \) delivery; and (2) because expiration is entirely passive, breath stacking can occur, causing pulmonary hyperinflation as a consequence of insufficient time for emptying of all lung units. Both problems can be managed by careful application of HFPPV and close monitoring of ventilatory waveforms and chest radiographs. The use of HFPPV has diminished with the availability of other types of high-frequency devices and with clinicians’ tentative acceptance of permissive hypercapnia in pediatrics.

High-Frequency Flow Interruption

Flow interruption is similar to jet ventilation (discussed later in the chapter) in that the \( V_t \) is created by a device that interrupts a gas flow or a high-pressure source at frequencies as high as 15 Hz. High-frequency flow interruption (HFFI) can be used either with a jet catheter in the airway or as a bulk flow device connected directly to the artificial airway. The most often discussed flow interrupter device is the one invented by Emerson, which consists of a conduit through which gas flow is directed. The conduit contains a ball that has a flow port in its center. An electric motor moves the ball back and forth in the conduit at a frequency of up to 200 cycles/min, interrupting the outflow of gas. As with a high-frequency jet ventilation system, the high-pressure streams of gas can entrain more static gas supplied by an added bias flow to augment the delivered \( V_t \). This type of HFV was among the early models developed, and it currently is used mostly for investigational purposes.

High-Frequency Percussive Ventilation

High-frequency percussive ventilation (HPFV) was developed by Forrest M. Bird, one of the pioneers in mechanical ventilation technology. Bird’s intent was to incorporate the most effective characteristics of jet and conventional ventilation into one device. HPFV can be used with a mask or mouthpiece as a therapeutic device to percuss the chest internally to remove secretions, or it can be used intermittently or before extubation by intubated patients to help mobilize secretions. It also can be used as a continuous mode of ventilation. HPFV has been used successfully as a continuous mode in children and has served as a prophylactic measure to prevent pneumonia and atelectasis in patients with thermal injury.

The VDR-4 (Bird Space Technology, Sandpoint, Idaho) is a high-frequency percussive generator that is used to superimpose high-frequency breaths onto conventional breaths. The device can be compared with time-cycled, pressure-limited ventilation, except that high-frequency pulsations (as high as 600 cycles/min [10 Hz]) are injected throughout the inspiratory phase. At the heart of the device is a sliding venturi (Fig. 22-22) with a jet orifice at its mouth. The jet is surrounded by a continuous bias flow of warmed, humidified gas. On inspiration, a diaphragm connected to the venturi fills with gas and slides it forward, toward the patient’s airway, blocking
the expiration ports. At the same time, the jet begins delivering short, percussive pulsations. A large amount of air is entrained so that flow to the patient is very high; this is the result of the large pressure gradient between the patient connection and the jet. When the gradient begins to decrease during inspiration, air entrainment and total flow also begin to slow, but the jet pulsations continue. When the time limit for inspiration is reached, the jet cycles off. The diaphragm, no longer pressurized, collapses, and the venturi slides back, opening the expiratory ports. A counter flow of gas sufficient to maintain a set PEEP is directed toward the airway during the expiratory phase. A schematic of the HFPV system is shown in Fig. 22-23.

**High-Frequency Oscillatory Ventilation**

High-frequency oscillatory ventilation (HFOV) has become the most widely used high-frequency technique for infants and pediatric patients. It differs from other high-frequency techniques in several important ways:

- Both inspiration and expiration are active.
- Gas flow is sinusoidal rather than triangular.
- Bulk flow, rather than jet pulsations, is delivered.
- \( V_t \) is less than dead space.

An HFOV device can be powered by a reciprocating pump, diaphragm, or piston. Although they are not true oscillators, flow interrupters can be assimilated into ventilators called *pseudooscillators* that provide the effect of an oscillator.99

Oscillators and at least one pseudooscillator have been in use since the late 1980s. A modern example of an oscillator is the SensorMedics 3100A (SensorMedics, a division of Viasys, San Diego, Calif). This oscillator uses a diaphragm-shaped piston that is driven magnetically, similar to the action of a stereo speaker (Fig. 22-24).

The 3100A has a rigid plastic circuit into which a warmed, humidified bias flow of gas is introduced just in front of the piston (Fig. 22-25). The bias gas flows through the circuit and exits from a restricted orifice and mushroom valve assembly that maintain the set \( P_{aw} \). The \( P_{aw} \) control is used to set the tension on the diaphragm, which oscillates the flow. The \( V_t \), or amplitude, is set by the power control and is determined by the forward and backward excursion distance of the piston. The frequency is determined by the number of piston excursions. Two other mushroom valves function as safety releases on the circuit. See the Clinical Scenario involving HFOV below.

**Clinical Scenario: Pulmonary Interstitial Emphysema (PIE)**

A premature infant with severe pulmonary interstitial emphysema (PIE) is being ventilated on a conventional infant ventilator at a \( P_{aw} \) of 18 cm \( H_2O \). The clinical team caring for this patient has decided to place him on high-frequency oscillatory ventilation (HFOV). What strategy would you use, and what initial \( P_{aw} \) and fraction of inspired oxygen \( (FIO_2) \) would you choose?

A low-volume HFOV strategy often is used for patients with an air leak syndrome (e.g., PIE). The \( P_{aw} \) used for the conventional ventilator also should be set on the oscillator initially. The \( FIO_2 \) should be set as high as 1.0 for the first 12
Neonatal and Pediatric Respiration Support

Compressed air

Entrained room air

Pressure regulator

Air amplifier

Flexible diaphragm “bellows”

Plastic “bellows” housing

Coil

Patient circuit port

Diaphragm

Square wave driver

Water trap

Condensate drain tube

Fig. 22.24 Drive mechanism for the SensorMedics 3100A oscillator. A timer (square wave driver) signals the motor to drive the piston toward and away from the patient circuit port, making both inspiration and expiration active. Because the movement of the piston generates extreme heat, compressed air and entrained room air are introduced around the motor’s coil to provide cooling. (Courtesy SensorMedics, Corp., Yorba Linda, Calif.)

to 24 hours because the goal is to keep the arterial partial pressure of oxygen (PaO₂) above 55 mm Hg. This strategy maintains adequate oxygenation and ventilation while preventing extension of the air-leak syndrome. It also promotes resolution of the PIE by eliminating the potential volutrauma-producing factors. Achievement of optimal volumes (as indicated by the chest radiograph) should be avoided until the PIE has resolved. Serial chest radiographs are obtained to evaluate resolution of the condition. Once the air-leak syndrome has resolved, an optimal lung-volume strategy can be pursued (Case Study 22-8). (Chapter 23 presents information on HFOV for adults.)

Case Study 22-8

Patient Case—Acute Respiratory Distress Syndrome Managed with HFO

A 5-year-old girl with a diagnosis of ARDS secondary to sepsis and aspiration pneumonia has been on the SensorMedics 3100A oscillator for about 4 hours. The mean airway pressure is set at 28 cm H₂O, the frequency at 8 Hz, and the amplitude at 38 cm H₂O. The F₁O₂ is 0.7. Initially the ABG values and vital signs improved. However, over the past 30 minutes, the heart rate has increased and the SpO₂ has dropped from 97% to 87%. What action should the respiratory therapist consider?

See Appendix A for the answer.

High-Frequency Jet Ventilation

Largely pioneered in the late 1970s, high-frequency jet ventilation (HFJV) remains a widely used high-frequency technique, particularly in infants. It was the first high-frequency technique to attempt delivery of a V₁ smaller than dead space. HFJV originally was used to provide short-term ventilatory support during adult upper airway surgery and instrumentation, but animal studies showed

Fig. 22.25 Basic breathing circuit of the SensorMedics Model 3100A high-frequency oscillator. ET, Endotracheal tube; ATM, atmosphere. (Courtesy ViaSys Respiratory Care, Inc. Yorba Linda, Calif.)
that it also provided effective alveolar ventilation and gas exchange in acute lung disease.

Previously HFJV was delivered through a specially made, triple-lumen ET. Adapters are now available for converting a conventional ET to a jet tube. A ventilator designed to deliver HFJV is the Bunnell Life Pulse (Bunnell, Salt Lake City, Utah). The principle of HFJV involves the delivery of short jet breaths, or pulsations, of an air-oxygen gas mixture under considerable pressure through an ET. The Bunnell Life Pulse can deliver frequencies in the range of 240 to 660 cycles/min. \(^{(15)}\) Jets are delivered by electronic solenoids or fluidic valves. Most infants are well ventilated at an HFJV rate of 420 cycles/min. Small changes in the rate usually have little effect on PaCO\(_2\) because of patients’ broad range of resonant frequency. Larger patients and those prone to gas trapping may benefit from lower HFJV rates (240-360 cycles/min).

Tidal volume in HFJV depends on the length of the pulsation; the amplitude, or driving pressure, of the jet; the size of the jet orifice; and the patient’s Raw and C\(_T\). For infants the typical delivered V\(_j\) is 1 to 3 mL. However, a V\(_j\) that is larger or smaller than the patient’s dead space volume can be delivered. Under certain conditions, gas entrainment can occur around the jet, slightly increasing V\(_j\) by a physical process called jet mixing, which is caused by the viscous shearing of the jet-gas layer with stagnant gas in the airway. The stagnant gas is dragged downstream in an entrainment-like effect. The volume of entrained gas varies with the patient’s lung mechanics.

Often the PEEP is set much higher in HFJV than in conventional ventilation. Because jet devices deliver significantly less V\(_j\) and P\(_aw\) than other forms of mechanical ventilation, a higher PEEP may be used without elevating P\(_aw\) to potentially harmful levels. \(^{(45\text{-}65)}\)

In most patients the conventional ventilator is operated in the continuous mandatory ventilation (CMV) mode at a rate of 10 breaths/min or less. In HFJV the jet accomplishes much of the alveolar ventilation. Experience has shown that when an appropriate level of PEEP is used to achieve optimum recruitment of lung units, the jet device can effectively ventilate without the need for conventional breaths. \(^{(100)}\) Once the patient is ready to be weaned from the ventilator, transitioning to conventional ventilation and discontinuation of the jet are relatively easy, and from that point conventional weaning can take place.

**Physiology of High-Frequency Ventilation**

In the high-frequency techniques in which V\(_j\) is less than dead space, the predominant means of gas transport by bulk convection is superseded by other mechanisms. However, alveoli close to the airways are still ventilated by convection, as in conventional ventilation. Many other mechanisms of gas transport in HFV are theoretical and are not completely understood. Such mechanisms include pendelluft, streaming, Taylor-type dispersion, and simple molecular diffusion (Fig. 22-26). The degree to which these mechanisms play a role depends on the HFV technique used, the characteristics of the high-frequency generator, the ventilator settings, and the patient’s lung characteristics. \(^{(101)}\)

**Pendelluft**, which is the exchange of gas between lung units with different time constants, is observed through photographic studies or by the measurement of different pressure values in the airways. \(^{(102\text{-}103)}\) Although other gas transport mechanisms may bring fresh gas to the small airways, the movement of gas across lung units before it leaves the lung may enhance gas exchange between alveoli and pulmonary capillaries. Over time, more gas may enter lung units with longer time constants so that these units may be recruited.

**Streaming**, or asymmetric velocity profiles, is thought to occur because the velocity of gas flow is higher in the center of the airway (Fig. 22-27). Pulsations from the jet push the gas forward in the center, causing gas along the airway walls to be pushed backward. This outer layer of gas moves at a slower velocity. Because much of the gas occupying the space along the walls is dead space gas, the forward-moving alveolar gas may be used more efficiently. \(^{(104)}\)

**Taylor-type dispersion** is the enhanced diffusion of gases caused by the turbulence of high gas flows reaching small airways. This is thought to be a principal mechanism of gas transport in high-frequency oscillation. \(^{(105\text{-}106)}\) With the rapid injection of small gas volumes at high flows, the erratic formation of streams and eddies (particularly at airway bifurcations) shortens the diffusion times of gases over the distances they normally travel. With this type of enhanced gas transport, **simple molecular diffusion** likely is enhanced as well because more mixing of inspired and expired gas occurs at more distal points in the tracheobronchial tree.

The mechanism of augmented transport is further affected by the active expiration produced by the oscillator. With other ventilators, the formula for the V\(_j\) produced is V\(_j\) = f × V\(_j\); with HFOV, the formula is V\(_j\) = f × V\(_j\). Although V\(_j\) values are in the range of only 0.8 to 2 mL/kg, the interplay of gas transport mechanisms provides highly effective CO\(_2\) elimination. V/Q matching is improved with HFV because P\(_aw\) is used to achieve optimal lung volume and to maintain that volume throughout the respiratory cycle. **Reaching optimal lung volume** means that lung units that otherwise would be closed are open, providing more area for gas exchange. Moreover, the duration of gas exchange is greatly extended because no inspiration or expiration takes place.

**Oxygenation** is one of the factors that increase pulmonary blood flow. If a higher lung volume is achieved, pulmonary vasodilation can result because of improved oxygenation. The diameter of pulmonary vessels is yet another factor that greatly affects pulmonary blood flow. With higher lung volumes, radial traction to the walls of larger pulmonary vessels increases, enhancing blood flow.

**Management Strategies for High-Frequency Ventilation**

Assessment of breath sounds, heart sounds, pulmonary compliance, and other such parameters is difficult in patients receiving HFV; therefore a thorough assessment should be performed before the patient is connected to the high-frequency device. The baseline V\(_j\) should be noted if the patient initially received conventional ventilation. If possible, a chest radiograph should be taken shortly before HFV is initiated to document baseline lung inflation and to check the position of the ET.

If indicated, an initial dose of artificial surfactant is given while the patient is receiving conventional ventilation. Subsequent doses may be given after HFV has been started. Note that some clinicians prefer to keep the capability of conventional ventilation at the bedside to use during surfactant dosing; others prefer to give surfactant while manually ventilating the patient and forgo any use of conventional ventilation.

Preparations for placing a patient on HFV include repositioning the patient and completing any procedures that could cause agitation. Endotracheal suctioning is performed so that interruptions do not occur during the initial period. A pulse oximeter is

Airway

Expiratory gas flow

“Spike” of inspiratory gas

Flow from ventilator

Atelectatic compartments exposed to increased oscillatory pressures

Cardiogenic mixing

Convection

Oscillatory pressure applied at airway opening is damped by flow-dependent resistance and inertance of tracheal tube and central airways

Convection and diffusion

Turbulent flow and radial mixing

High peripheral resistance increases pressure transmission to more proximal airways and nearby alveoli

Alveoli distal to a zone of increased peripheral resistance see low pressures due to decreased flow

Diffusion

Laminar flow and radial mixing

Expanded and aerated alveoli protected from high oscillatory pressures

Collateral ventilation

Put in place, and an electrocardiogram and BP are monitored continuously. Transcutaneous CO₂ monitors, used for trending purposes, work well on most patients regardless of age. If a transcutaneous monitor is used, the sensor is placed on the patient and the baseline comparison to PaCO₂ is made before HFV is initiated.

Cardiovascular assessment focuses on intravascular volume and cardiac output. A high, sustained Paw can greatly reduce cardiac output if circulatory volume is not adequate. Once a patient has been placed on HFV, some practitioners prefer to administer crystalloids and colloids only if the mean arterial pressure drops.

If this strategy is chosen, the patient may need to be removed from the high-frequency ventilator several times for manual ventilation until additional fluid volume can be given. Adequate sedation is provided before HFV is initiated. Some spontaneous breathing may be acceptable. However, depending on the severity of cardiopulmonary disease, agitation and excessive movement can interfere with high-frequency breaths and gas exchange. Paralysis is not always necessary, but some suppression of respiratory drive is desirable. Management strategies differ according to the specific type of HFV used. Generally the goal in all types is to provide effective gas exchange at the lowest possible F₂O₂ and Paw. Also inherent in all types of HFV is the need to escalate support frequently until a certain threshold is reached and the patient is said to be “captured” on the ventilator. Often a dramatic improvement in oxygenation or ventilation, or both, is seen when this occurs. If the patient's condition is stable, some weaning can begin almost at once.

**Management of High-Frequency Oscillatory Ventilation in Infants**

A recent review compared outcomes in preterm neonates using HFOV versus gentle conventional ventilation. HFOV was associated with an increase in air leaks and a reduction in surgical...
ligation of patent ductus arteriosus or retinopathy of prematurity. There were no differences in BPD, mortality, or neurologic insult. However, in neonates where randomization occurred earlier (1-4 hours), HFOV showed a significant benefit for reducing death or BPD over conventional ventilation.107

Unlike with the jet ventilator, which incorporates a conventional ventilator as part of its gas delivery system, the patient cannot be gradually transitioned from conventional ventilation to HFOV. Typically, manual ventilation may be the only means of optimizing alveolar recruitment and oxygenation before a patient is placed on an oscillator. Sustaining manual inflations with increasing levels of PEEP immediately before connection may enhance initial recruitment and make the transition to HFOV more successful.

Two basic treatment strategies are suggested for HFOV, depending on the patient's condition. One is the optimum lung volume strategy (Fig. 22-28). The goal of this strategy is to increase $P_{aw}$ on the oscillator until oxygenation stabilizes. The PaCO$_2$ is maintained within a range established by the management team. A chest radiograph is obtained within the first 2 hours and every 12 to 24 hours thereafter. Optimum lung inflation is indicated by radiographic findings of decreased opacification and lung expansion to the eighth or ninth posterior rib level on the right hemidiaphragm. Once this level of expansion has been established, subsequent chest radiographs should be used primarily to check for overinflation rather than to guide adjustments in $P_{aw}$. Reducing the F>O$_2$ to 0.45 to 0.5 may be possible, depending on cardiovascular status. This is followed by weaning $Paw$ (Fig. 22-29). Patients with air leak syndromes (e.g., PIE, pneumothorax, and bronchopleural fistula) are placed on HFOV using a low-volume strategy; the goals of this strategy are to prevent extension of the air-leak syndrome and to promote its resolution by eliminating factors that can potentially produce volutrauma. This strategy differs from the optimum lung volume strategy in that the lowest acceptable lung volumes are maintained using the lowest possible $Paw$. The initial $Paw$ usually is set at the same level or 2 to 3 cm H$_2$O higher than the $Paw$ required for conventional ventilation. The amplitude and frequency are set and adjusted according to the optimum lung volume strategy algorithm (see Fig. 22-28). An F>O$_2$ as high as 1 is considered acceptable during the first 12 to 24 hours.44 The F>O$_2$ usually is reduced to 0.8 before weaning the $Paw$. This is done to provide some margin in case oxygenation drops after the $Paw$ is lowered; the F>O$_2$ can be increased to help restore oxygenation. The decision to wean from the $Paw$ rather than the F>O$_2$ is made according to the progress seen in correcting air leaks. If they are resolving, the need to reduce $Paw$ is not as important as the need to reduce F>O$_2$. Conversely, extension of air leaks may require a lower $Paw$, if possible, with a higher F>O$_2$. (see Case Study 22-8). Patients with a pulmonary air leak should not be removed from the oscillator, and manual ventilation should be avoided. The ET should be suctioned with an inline suction catheter if possible. Pediatric patients with ARDS and other forms of hypoxic respiratory failure can be supported using HFOV. HFOV is typically initiated after conventional modes of ventilation have been unsuccessful. Many of the same principles that guide neonatal HFOV management can be applied to pediatric HFOV management. The major difference is that HFOV is applied using higher mean airway pressure, greater amplitude, and lower-frequency settings in pediatrics. Compared with CV, HFOV initiated earlier in the disease process may improve gas exchange and reduce VILI in pediatric patients with ARDS.108

WEANING AND EXTUBATION

The length of time a patient receives mechanical ventilation is an independent risk factor for morbidity. For this reason, many institutions have established weaning protocols in an effort to remove unnecessary obstacles to weaning and extubation. A multicenter study of the weaning of pediatric patients from the ventilator showed little difference between weaning according to clinical guidelines and weaning without following guidelines.52 The time from start of weaning to extubation, as well as the rate of extubation failure, seemed to be unaffected by the use of written weaning protocols.52 Routinely evaluating a patient for weaning readiness has been shown to be far more useful in facilitating timely ventilator discontinuation and extubation.109 Careful attention should be paid to the balance of sedative drugs and the patient’s ventilatory status because excessive sedation is the most significant factor contributing to weaning failure. Patient preparation and use of the extubation readiness test (see Box 22-8) can help achieve the earliest possible extubation.

Some institutions have established a testing procedure for determining a patient’s readiness for weaning. This so-called weaning readiness test (Box 22-8) usually is conducted in patients whose sedation score would permit extubation. The patient’s enteral feedings are stopped for the test, the F>O$_2$ is reduced to 0.5, and PEEP is reduced to 5 cm H$_2$O. If the SpO$_2$ is greater than 95% on these settings or with a lower F>O$_2$, the pressure support level is reduced to the minimum amount for the ET size (see Box 22-8).

## BOX 22-8 Extubation Readiness Test

**Procedure**

1. Temporarily stop enteral feedings.
2. Reduce the fractional inspired oxygen (F>O$_2$) to 0.5.
3. Reduce the positive end expiratory pressure (PEEP) to 5 cm H$_2$O.
4. Evaluate the oxygen saturation by pulse oximetry (SpO$_2$):
   a. If the SpO$_2$ is below 95% and the F>O$_2$ is less than 0.5, increase the F>O$_2$ to 0.5.
   b. If the SpO$_2$ is above 95%, change to pressure support ventilation (PSV) at the minimal amount for the endotracheal (ET) tube size used:
      - 3-3.5 mm: 10 cm H$_2$O
      - 4-4.5 mm: 8 cm H$_2$O
      - 5 mm or larger: 6 cm H$_2$O
   c. Monitor the SpO$_2$, effective tidal volume ($V_t$), and respiratory rate (f).

**Assessment**

The patient potentially is ready for extubation if:

- The SpO$_2$ is over 95%.
- The effective $V_t$ is over 5 mL/kg.
- The respiratory rate is within the goal range for the patient’s age (see chart) for up to 2 hours:

<table>
<thead>
<tr>
<th>Age</th>
<th>Goal Range (breaths/min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Under 6 mo</td>
<td>20-60</td>
</tr>
<tr>
<td>6 mo to 2 yr</td>
<td>15-45</td>
</tr>
<tr>
<td>2-5 yr</td>
<td>15-40</td>
</tr>
<tr>
<td>Over 5 yr</td>
<td>10-35</td>
</tr>
</tbody>
</table>
HFOV “Optimum” Lung Volume Strategy
Early Intervention for Pulmonary Injury Sequence*

Infant intubated for pulmonary injury sequence without air leak

A

HFOV
MAP 10 cm H2O
Hertz 10 for >750 gm
Hertz 15 for <750 gm
Ti = 33%
LiP = adequate for chest wall movement
PCO2 - high normal

B

Conventional mechanical ventilation

Switch to HFOV
MAP 1-2 cm H2O higher than on CMV
Hertz 10 for >750 gm
Hertz 15 for <750 gm
Ti = 33%
LiP = adequate for chest wall movement
PCO2 - high normal

Immediately increase MAP in 1 cm H2O increments until arterial oxygenation improves
Adjust LiP to achieve PCO2 45

C

O2 sat begins to fall
O2 sat stable
O2 sat rises
O2 stable

D

Evaluate FiO2 need

FjO2 <30%

CXR after 1 hr

FjO2 >30%

On CXR diaphragm below T9 or intercostal bulging
Wean MAP 1-2 cm

E

Evaluate cardiovascular status

Evaluate lung volume CXR

Consider Non-RDS diagnosis

F

On CXR diaphragm below T9 or intercostal bulging

Evaluate blood volume
CVP
ECHO
BP/CRT

Evaluate Myocardial function
ECHO

R hemidiaphragm <T9 go to F
consider hypoplasia

R hemidiaphragm
T8-9 go to F

R hemidiaphragm
>T9 wean MAP and go to D as indicated

G

Follow CXR for lung volume
Q6H X 24 hr then Q12H

Begin turning infant at 24 hr

Suctioning
1) Prior to 24-48 hr, PRN
2) Routine suctioning at 24 hr Q4H, PRN unless nonindicated

Go to weaning protocol

*Follow arrows as protocol. Letters do not necessarily follow in sequence.

Fig. 22-28 Strategy flow chart for optimum lung volume HFOV. HFOV, High-frequency oscillatory ventilation; MAP, mean airway pressure; ΔP, pressure gradient; Pco2, partial pressure of carbon dioxide; FIo2, fraction of inspired oxygen; CPR, chest radiograph; RDS, respiratory distress syndrome; CVP, central venous pressure; ECHO, echocardiogram; T8-T9, thoracic vertebrae; PV, pressure/ volume; qH, every hour; CRT, hematocrit. (From Minton S, Gerstmann D, Stodard R: Cardiopul Rev, PN 770118-001, Yorba Linda, Calif, 1995, SensorMedics.)
Once the patient has been placed on the minimum-pressure support level for the ET size, the respiratory rate and SpO$_2$ are monitored. Increases in the respiratory rate above guideline parameters or a drop in SpO$_2$ signals a failed test and suggests that additional support is needed. Although the test is called an extubation readiness test, it is only one criterion in the decision on extubation. It also is a useful test for determining whether PSV might be an appropriate mode for the patient and the pressure support level that should be set on the ventilator.

Extubation failure often is attributed to glottic or subglottic injury or edema. Patients who have had airway manipulations, multiple intubations, or unplanned extubations tend to have unsuccessful extubation more often. Yet in various studies a large number of patients who had an unventilated ventilator course and planned extubation nevertheless had a failed extubation.

An air-leak test has been recommended before extubation is scheduled, but there is controversy over whether this test can predict successful extubation or not. For this simple test, the clinician deflates the cuff of the ET, places a stethoscope directly over the larynx, and gives a manual breath; the rush of gas around the ET should be heard. The pressure at which the air leak is heard should be noted. If an air leak is present at 20 mm Hg pressure or less, the patient is unlikely to have postextubation stridor. In one clinical trial the drug was given 6 to 12 hours before extubation and every 6 hours afterward, for a total of six doses. This protocol sometimes is followed for patients at especially high risk for stridor and extubation failure. Steroid administration appears to be somewhat beneficial.

**ADJUNCTIVE FORMS OF RESPIRATORY SUPPORT**

**Surfactant Replacement Therapy**
Pulmonary surfactant has the remarkable ability to distribute itself in a thin layer between the alveolar surface and the alveolar gas. About 90% of human surfactant is made up of phospholipids, about 60% of which is dipalmitoyl phosphatidylcholine (DPPC). DPPC, other phospholipids, and neutral lipids produce the surface-active effects in the lungs. The distribution of surfactant is thought to be due to the low pH of the phospholipids, which makes them easily absorbed. Surfactant also contains at least three types of proteins that help distribute and regulate its life cycle and absorption. The challenge to manufacturers of artificial surfactant has been to produce a material that has the same type of surface action, can be instilled into the lung, and can immediately distribute itself to the periphery. A major problem with instilling any material into the lung is that it can obstruct gas flow and impede gas exchange.
Survanta and Infasurf (calfactant) are currently the most frequently used surfactant replacements. These preparations, which are extracted from calf-lung washings, contain some proteins plus the major phospholipids. Both have proven highly effective at reducing mortality in very premature infants.\textsuperscript{111}

Two strategies are suggested for surfactant replacement therapy: prophylactic therapy and rescue therapy. \textit{Prophylactic therapy} consists of surfactant administration immediately at birth or soon after for infants who are at risk of developing RDS. \textit{Rescue therapy} involves surfactant administration in infants who have RDS or another surfactant deficiency syndrome. Although no indications for surfactant replacement other than RDS have been established, surfactant replacement therapy is used in infants with meconium aspiration, pneumonia, and pulmonary hemorrhage. Older children and adults with ARDS also have been treated successfully with surfactant.

The procedure for administering surfactant depends on the type used and the manufacturer’s recommendations. Usually the patient is placed on a conventional ventilator set at a frequency of at least 30 breaths/min and an F\textsubscript{O\textsubscript{2}} of 1.0. Each partial dose usually is followed by 30 seconds on the ventilator. Once the full dose has been given, the ventilator is adjusted back to baseline settings (or HFV can be resumed at baseline settings). Regardless of the mode of ventilation used, signs of improving pulmonary compliance are monitored. For conventional ventilation, changes in V\textsubscript{T} and waveforms are noted and settings adjusted appropriately. If the patient is placed on a HFV, the F\textsubscript{O\textsubscript{2}}, P\textsubscript{aw}, Sp\textsubscript{O\textsubscript{2}}, and ABG values are evaluated (see section of HFV previously discussed). Some clinicians prefer to obtain a chest radiograph shortly after surfactant replacement regardless of the type of ventilation used.

During and after surfactant administration, the clinician watches for signs of ET or large airway obstruction, including poor chest excursion, oxygen desaturation, and bradycardia. If the patient has preexisting obstructions, the liquid can be preferentially administered into one lung. Another problem that might be encountered during surfactant administration is reflux of the surfactant up the ET because of patient agitation and coughing. In these cases the dose may be inadvertently deposited in pharynx because of a leak around the ET. Even without tube obstructions, hypoxemia may worsen.\textsuperscript{112} Some patients develop prolonged periods of apnea after surfactant dosing.\textsuperscript{113}

Other complications of surfactant replacement therapy have been reported. Pulmonary hemorrhage is a serious complication and is most often seen in very premature infants. The incidence of pulmonary hemorrhage varies inversely with birth weight.\textsuperscript{114} Mucus plugging, especially of smaller ETs, has been reported in the hours after surfactant dosing. A long-term complication is an increase in retinopathy of prematurity in infants who have received surfactants; the cause of this is not entirely understood.\textsuperscript{115}

Volutrauma and overdistention of the lungs have been reported in infants who have responded favorably to surfactant replacement; these conditions may be a result of failure to address increasing compliance by promptly reducing the P\textsubscript{aw} delivered by the ventilator.\textsuperscript{116} This situation underscores the importance of monitoring V\textsubscript{T} and using VSV after the immediate postdosing period. Monitoring for changes in shunt is crucial after surfactant replacement in patients with patent ductus arteriosus (PDA), particularly newborns with BPD.\textsuperscript{117} Theoretically, the reduced pulmonary vascular resistance produced by improved oxygenation can increase the left-to-right shunting caused by the PDA. This may prevent spontaneous closure of the ductus. A common belief is that oxygenation will worsen after the initial improvement in lung mechanics because of the effects of a PDA. The immense success of surfactant therapy in infants with RDS has prompted clinicians to use it in the treatment of ARDS in older patients. Studies have been conducted using both aerosolized administration and intratracheal instillation of surfactant.\textsuperscript{118} The studies showed an initial improvement in the P\textsubscript{a}O\textsubscript{2}/F\textsubscript{O\textsubscript{2}} ratio in most subjects, but sustained improvement beyond 48 hours of treatment has not been achieved.\textsuperscript{119} Future studies are needed to demonstrate a long-term benefit of surfactant replacement therapy in adult ARDS patients. Until that time, its use is not recommended in adults.

### Prone Positioning

Pediatric patients treated for acute respiratory failure are sometimes positioned prone in an attempt to improve oxygenation. The overall beneficial effect of the prone position is to improve V/Q matching and reduce physiological shunt (see Chapter 12). Assuming that the dorsal regions of the lung are atelectatic because the patient has been supine, repositioning into the prone position may help recruit collapsed areas. However, results from one clinical trial were unable to show any benefit in outcomes related to the use of prone positioning in pediatric patients with acute lung injury.\textsuperscript{119}

### Inhaled Nitric Oxide Therapy

Inhaled nitric oxide (INO) is a colorless, odorless gas that is also a potent pulmonary vasodilator. When given via inhalation, NO rapidly diffuses across the alveolar capillary membrane and is bound to hemoglobin and thus has little effect on the systemic circulation. The therapeutic goal of most NO regimens is to improve pulmonary blood flow and enhance arterial oxygenation. Medically its effectiveness can spare patients the need for more invasive procedures, such as extracorporeal membrane oxygenation (ECMO).

Several systems have been designed to administer the most common system for providing INO through circuits for spontaneously breathing patients or through ventilator and anesthesia circuits. In one widely used system, the INOVent Max or DS (Ikaria, Clinton, NJ), a pneumotachometer incorporated into an injector module is placed inline with the delivered gas. The module measures the actual flow and simultaneously injects NO to achieve the set concentration. Changes in flow or the use of a variable flow pattern do not affect the delivered NO concentration. This system also monitors the delivered NO and nitrogen dioxide (NO\textsubscript{2}) and the F\textsubscript{O\textsubscript{2}}.\textsuperscript{120} Safe administration of iNO largely depends on monitoring of the inhaled gas mixture. Two types of toxicity have been reported with iNO in both animal and human subjects: pulmonary tissue toxicity and methemoglobinemia.\textsuperscript{121} Pulmonary tissue toxicity is a well-known side effect and results when NO combines with oxygen and forms the reddish brown gas NO\textsubscript{2}. When NO\textsubscript{2} is exposed to NO, dinitrogen trioxide (N\textsubscript{2}O\textsubscript{3}) is produced and reacts with water, forming either nitrous or nitric acid, both of which are very toxic to the alveolar epithelium.\textsuperscript{122} The higher the concentration of oxygen, the greater the potential for the development of toxic levels of NO\textsubscript{2}.

Many clinical situations that require administration of iNO also require very high concentrations of oxygen in the gas mixture. In such cases even low-dose NO can produce toxic levels of NO\textsubscript{2}, thus underscoring the importance of NO/NO\textsubscript{2} monitoring in the inhalation circuit. However, when NO is administered at low doses,
Determining Appropriateness of Nitric Oxide Therapy

A 33-hour-old infant with respiratory distress has just been transferred to the newborn ICU. Mild cyanosis is developing, and the SpO2 percentage is in the low 60s on supplemental oxygen. The chest radiograph is unremarkable. There is no evidence of meconium aspiration and no maternal history of infection. The peripheral pulses are weak, particularly in the lower extremities. The blood pressure is 32/12 mm Hg, and the heart rate is 190 beats/min. No murmur is noted. The respiratory rate is 80 to 100 breaths/min with moderate retractions and nasal flaring.

The patient eventually is intubated, sedated and paralyzed. An umbilical artery catheter (UAC) is placed, and administration of dopamine and fluids is initiated. ABG values show refractory hypoxemia, a low PaCO2, and metabolic acidosis. The patient is placed on mechanical ventilation with 100% oxygen.

The ICU team is leaning toward a diagnosis of persistent pulmonary hypertension (PPHN) but is not ruling out congenital cyanotic heart disease. The respiratory therapist is asked her opinion about starting nitric oxide therapy. How should she respond?

See Appendix A for the answer.

it usually reacts slowly with oxygen, and the formation of toxic products is small. Nonetheless, close monitoring is essential to control the therapeutic level of NO while avoiding excessive levels of NO2.

Methemoglobinemia develops primarily through the oxidation of NO when it comes in contact with oxyhemoglobin. Methemoglobin occurs naturally, and its level normally is maintained partly by the enzyme methemoglobin reductase. This enzyme, which is found largely in red blood cells, converts methemoglobin to hemoglobin. The rate of methemoglobin formation rarely exceeds the ability of the reductase to convert methemoglobin to hemoglobin; therefore the methemoglobin level is usually less than 2%.

Studies of iNOs effectiveness at reducing intrapulmonary shunt and improving V/Q matching suggest that the drug is most effective when used with high-frequency ventilation.124,125 These investigators maintain that effective recruiting of lung units enhances the effect of NO. A comprehensive review of evidence for the labeled use of iNO in hypoxemic infants, devices, clinical monitoring, and management has been provided in an AARC clinical practice guideline (Case Study 22-9). (See the Evolve website for this text for additional information on NO therapy.)

- Neonatal and pediatric patients have smaller lungs, higher airway resistance, lower lung compliance, less surface area for gas exchange, and lower cardiovascular reserve than do adults, making them more vulnerable to rapid onset of respiratory distress.
- Neonates experiencing respiratory distress present with tachypnea, nasal flaring, and intercostal, substernal, and retrasteral retractions.
- The Silverman Anderson respiratory scoring system is a useful clinical tool to assess the degree of respiratory distress in neonates.
- Pediatric patients experiencing respiratory distress can present with some of the same clinical manifestations as neonates. However, larger pediatric patients have ossified or “stiffer” chest walls and are able to sustain longer periods of WOB than neonates.
- Determining oxygenation and ventilation in neonate and pediatric patients is evaluated by ABG analysis and noninvasive techniques, such as SpO2 and transcutaneous CO2 measurements. Chest radiograph evaluation is another important tool.
- The goals of mechanical ventilatory support in newborn and pediatric patients are to (1) provide adequate ventilation and oxygenation, (2) achieve adequate lung volume, (3) improve lung compliance, (4) reduce WOB, and (5) limit lung injury.
- Noninvasive respiratory support can include nasal CPAP, nasal IPPV, nasal IMV for neonates and CPAP, and BiPAP in pediatric patients.
- Used appropriately, CPAP is a less invasive and less aggressive form of therapy than other forms of ventilatory support.
- Complications of CPAP include pulmonary overdistention and can lead to ventilation/perfusion mismatching, decreased pulmonary blood flow, increased pulmonary vascular resistance, and decreased cardiac output.
- NIV, aka “CPAP with a rate,” is an established form of ventilatory support in adults and pediatric patients wherein superimposed positive pressure inflations are combined with CPAP to reexpand atelectatic areas, improve gas exchange, reduce respiratory distress, avoid apnea, and potentially obviate invasive mechanical ventilation.
- Nasal synchronized and nasal IMV are the most commonly used breath types and pressure control is the most common mode for providing NIV in neonates.
- Nasal “sigh” positive airway pressure (SiPAP) is being used more frequently to assist spontaneously breathing infants in the NICU because it allows the neonate to breathe at a high and a low CPAP setting.
- Nasal HFV is becoming more common in clinical practice as a form of NIV as it uses smaller pressures, higher frequencies, and may be more lung protective than other NIV devices that apply higher pressure to the lungs.
- CPAP is used less often for pediatric patients than for adults; however, it is useful in children to restore FRC, reduce WOB with acute hypoxemia, neuromuscular disorders, and conditions that cause abdominal distention. It also is used to relieve the airway obstruction associated with obstructive sleep apnea or airway lesions like laryngotracheal malacia.
- Conventional mechanical ventilation or “invasive” mechanical ventilation involves the use of positive-pressure inflations in intubated patients that are breathing spontaneously or whom
are heavily sedated or paralyzed, but management of such patients should always be to avoid invasive ventilation whenever possible to minimize ventilator induced lung injury.

- Ventilator care of the newborn is often an integral part of the broader management of premature infants.
- Most newborns who require full ventilatory support are placed on infant ventilators or infant through adult ventilators specifically designed to respond to even the smallest of patients.
- The most frequently diagnosed cause of respiratory failure in pediatric patients under the age of 1 year was bronchiolitis; in children older than 1 year, pneumonia was most often the cause.
- Pressure-control ventilation is the most widely used mode of ventilation in neonates and pediatrics. The pressure-controlled breath can be triggered by pressure or flow and is terminated based on time. It can be used during IMV and CMV breath types.
- Monitoring inspiratory pressure, PEEP, Tt, expiratory time, I:E ratio, tidal volume, frequency, mean airway pressure and inspired oxygen concentration are all necessary with pressure-controlled ventilation.
- Older children and adults have been ventilated with volume-control ventilation in patient with ARDS and premature neonates with RDS.
- The most widely used form of dual-control mode used in neonates and pediatric patients is PRVC and is commonly used in patients with CMV or IMV breath types.
- VSV is well suited to infants and pediatric patients as target Vt is maintained, and self-weaning is possible.
- The goal of any lung-protective strategy is to (1) avoid repetitive opening and closing of small airways (atelectotrauma); (2) limit overinflation during inhalation (volutrauma); (3) reduce gas trapping during exhalation; (4) and alleviate pulmonary inflammation (biotrauma).
- HFV can be complicated by PIS, which includes RDS, PIE, pulmonary air-leak syndrome, oxygen toxicity, and development of BPD.
- Two potential problems are associated with HFPPV: (1) the high rate and short Tt may prevent adequate Vt delivery; and (2) because expiration is entirely passive, breath stacking can occur, causing pulmonary hyperinflation as a consequence of insufficient time for emptying of all lung units.
- HFOV has become the most widely used high-frequency technique for infants and pediatric patients because both inspiratory and expiration are active, gas flow is sinusoidal rather than triangular, blf flow, rather than jet pulsations, is delivered, and Vt is less than dead space.
- Assessment of breath sounds, heart sounds, pulmonary compliance, and other such parameters is difficult in patients receiving HFV; therefore a thorough assessment should be performed before the patient is connected to the high-frequency device.
- Adjunctive forms of respiratory support include surfactant replacement therapy, prone positioning and inhaled NO2 therapy.
- Prophylactic therapy consists of surfactant administration immediately at birth or soon after for infants who are at risk of developing RDS.
- Rescue therapy involves surfactant administration in infants who have RDS or another surfactant deficiency syndrome.
- The overall beneficial effect of the prone position is to improve V/Q matching and reduce physiological shunt.
- The therapeutic goal of most NO (a pulmonary vasodilator) regimens is to improve pulmonary blood flow and enhance arterial oxygenation.

**REVIEW QUESTIONS**

(See Appendix A for answers.)

1. A 6-hour-old term infant has intercostal retractions, nasal flaring, and grunting. HR is 180 beats/min, f is 70 breaths/min and regular, and SpO2 is 90% on room air. ABG values reveal a pH of 7.34, a PaCO2 of 28 mm Hg. and a PaO2 of 58 mm Hg. Which of the following would be most appropriate?
   A. Intubation and mechanical ventilation  
   B. Intubation and CPAP  
   C. Nasal CPAP  
   D. No intervention necessary at this time

2. Which of the following is (are) potential complications of CPAP in newborns?
   A. Pulmonary overdistention  
   B. Air-leak syndromes  
   C. Increased WOB  
   D. All of the above

3. A 1.4-kg neonate has been receiving nasal CPAP with the same NP tube for 2 days. The NP tube is connected to a ventilator set to deliver CPAP at 6 cm H2O with a flow rate of 8 L/min. Over the past 2 hours, the infant’s f increased from about 40 breaths/min to about 60 breaths/min. FIO2 had to be increased from 0.25 to 0.45 because of decreasing SpO2 values. Which of the following actions should be taken?
   A. Increase the flow rate  
   B. Increase the CPAP level

4. Which of the following is (are) considered essential for all infant mechanical ventilators?
   A. Pressure support capability  
   B. Patient-triggering  
   C. Leak compensation  
   D. All of the above

5. When an infant ventilator is operating in the pressure-control mode, the expiratory phase of the breath cycle begins when what preset cycle is reached?
   A. Pressure  
   B. Time  
   C. Volume  
   D. Flow

6. What is the difference between a demand flow IMV and a continuous flow IMV system in an infant ventilator delivering pressure control?
   A. A demand flow IMV system has a baseline bias flow; when the patient’s inspiratory flow exceeds the bias flow, a demand valve opens to provide whatever additional flow is needed.
B. A demand flow IMV system has a bias flow that is set by the manufacturer; it is activated only if the patient takes a spontaneous breath.

C. A demand flow IMV system does not have a bias flow that is set by the manufacturer; patient flow triggering opens a demand valve that immediately meets the patient’s inspiratory flow needs.

D. A demand flow IMV system has a baseline flow rate that is set by the clinician; if the patient’s inspiratory flow rate exceeds the set value, no additional flow is provided.

7. A previously healthy 3-year-old child is admitted to the ICU with an unconfirmed diagnosis of Varicella pneumonia. The child is lethargic, the breathing is labored, and the skin is cool and mottled. The respiratory rate is 15 breaths/min, HR is 190 beats/min, temperature is 38.8°C, BP is 70/44 mm Hg, and SpO2 is 83% on a nonbreathing oxygen mask. Breath sounds are distant, but coarse rales can be heard bilaterally.

ABG values reveal a pH of 7.26, PaCO2 at 64 mm Hg, and PaO2 at 55 mm Hg on the nonbreathing mask. Which of the following interventions would be appropriate based on the above information?

A. Intubate the patient and begin CPAP
B. Place the patient on a BiPAP system with supplemental oxygen
C. Maintain the patient on the nonbreathing mask, begin fluid replacement therapy to treat the low blood pressure, and obtain appropriate cultures
D. Intubate the patient and initiate mechanical ventilation

8. For an infant about to receive mechanical ventilation, the initial PIP and Tt are best determined by:

A. Placing the infant on the ventilator and adjusting PIP and Tt to obtain the desired VT
B. Manually ventilating the infant while noting the PIP and Tt that achieve the best SpO2 and lung aeration
C. Placing the infant on the ventilator and adjusting PIP and Tt to obtain the desired SpO2
D. Manually ventilating the infant while monitoring the waveform, noting the PIP and Tt that produce the best waveform

9. Theoretical advantages of the A/C mode on the Bird V.I.P. with both adjustable flow triggering and inspiratory flow termination for newborns are:

A. Less potential for lung injury
B. Reduced sedation requirements
C. Easier weaning
D. All of the above

10. A 3.5-kg newborn with a diagnosis of group B Streptococcus pneumonia is intubated with a 3 mm ID ET and is receiving mechanical ventilation with the CareFusion AVEA in the CMV mode. A monitoring device is in-line. The initial settings are as follows:

- Inspiratory Pressure = 24 cm H2O, PEEP = 4 cm H2O, FIO2 = 1.0
- set frequency = 20 breaths/min
- Actual frequency = 50 to 55 breaths/min
- Vmean = 45 to 50 mL, Vpeak = 12 to 15 mL, set Tt = 0.6 second, actual Tt = 0.6 second
- Flow cycle = 10%

The infant has received little sedation and is awake and breathing but appears to be fighting the ventilator. Patient triggering seems to be occurring with every breath, but the ventilator does not flow cycle regardless of the termination sensitivity setting. Which of the following interventions would be appropriate based on the preceding information?

A. Reintubate with a larger ET
B. Increase the PIP
C. Administer muscle relaxants and switch to a control mode
D. Increase the Tt

11. A 3.1-kg term infant has just returned from the operating room after removal of a small bowel obstruction. The infant will remain paralyzed and sedated over the next 12 hours and is receiving pressure control with the following settings:

- PIP = 20 cm H2O, PEEP = 6 cm H2O, frequency = 22 breaths/min, Tt = 0.6 second
- FIO2 = 0.3, flow rate = 8 L/min
- ABG values are pH = 7.26, PaCO2 = 66 mm Hg, PaO2 = 78 mm Hg

Additional data are as follows:

- VT = 7 to 10 mL, Vd = 1.38 L/min
- SpO2 = 95%, BP = 68/42 mm Hg

Based on these data, which of the following ventilator control manipulations would be most appropriate?

A. Increase the Tt
B. Increase the flow
C. Increase the frequency
D. Increase the PIP

12. A 2-year-old patient intubated with a 4 mm ID nasal ET is recovering from surgical repair of a ventricular septal defect and has been weaned from volume ventilation on SIMV to PSV (Servo ventilator). Since the changeover to this mode, the ventilator at times seems to trigger on and cycle off very rapidly, making the patient uncomfortable and very agitated. Which of the following should correct this problem?

A. Reintubate the patient with a larger tube
B. Switch to ventilator tubing with a larger diameter
C. Check sensitivity, rise time to set pressure, and flow-cycling criteria
D. Select a more appropriate mode

13. A newborn patient of 29 weeks’ gestational age has RDS. She weighs 950 g. She is receiving conventional mechanical ventilation at a Paw of 16 cm H2O. The patient is to be changed to HFOV. Which of the following settings would you initially select?

A. Paw = 18 cm H2O; frequency = 15 Hz
B. Paw = 16 cm H2O; frequency = 15 Hz
C. Paw = 18 cm H2O; frequency = 10 Hz
D. Paw = 16 cm H2O; frequency = 10 Hz

14. An 18-month-old, 15-kg child with a diagnosis of ARDS has been mechanically ventilated for 5 days. The patient initially received volume-cycled SIMV but now is receiving PCV at the following settings:

- PIP = 37 cm H2O, PEEP = 8 cm H2O, Paw = 16.4 cm H2O
- Frequency = 40 breaths/min, Tt = 0.9 second, FIO2 = 1
- ABG values are: pH = 7.29, PaCO2 = 53 mm Hg, PaO2 = 46 mm Hg, SaO2 = 79%

Additional data are as follows:

- VT = 75 to 85 mL
- Vd = 2.92 L/min

Based on the above data, which of the following would be most appropriate?
A. Increase the PEEP, maintain the PIP, and give sodium bicarbonate (NaHCO₃) to normalize the pH.
B. Change to high-frequency ventilation.
C. Maintain the present settings but give NaHCO₃ to normalize the pH.
D. Change to a high V̇/flow strategy

15. A patient with RDS who developed diffuse PIE on the right side has been on HFOV for 8 hours. Vital signs are stable, and ABG values on an FIO₂ of 0.7 are within acceptable limits. A chest radiograph shows that the PIE is worsening and expanding to the ninth posterior rib level on the right. Which of the following ventilator management strategies should be applied to this situation?
A. Maintain the current strategy and try to wean the FIO₂ as soon as possible.
B. Reduce the Paw even if the FIO₂ must be increased.
C. Increase the Paw and wean the FIO₂ as much as possible.
D. Switch to conventional ventilation.

16. A 640-g newborn is receiving HFOV at the following settings:
- P₁₅ = 19 cm H₂O, FIO₂ = 0.28, frequency = 15 Hz, amplitude (P) = 34 cm H₂O.
- ABG values: pH = 7.56, PaCO₂ = 23 mm Hg, PaO₂ = 85 mm Hg.
Based on the above data, which of the following would be most appropriate?
A. Reduce the amplitude.
B. Reduce the frequency.
C. Reduce the Paw.
D. Maintain the current settings.

17. A full-term, 3 kg infant is on HFJV at the following settings:
- PIP = 22 cm H₂O, PEEP = 11 cm H₂O, Paw = 12 cm H₂O.
- Frequency = 420 cycles/min, jet T₁ = 0.02 second, FIO₂ = 0.4.
- ABG values: pH = 7.3, PaCO₂ = 55 mm Hg, PaO₂ = 90 mm Hg.
Based on the above data, which of the following control changes should be made first?
A. Reduce the PEEP.
B. Reduce the frequency.
C. Increase the jet T₁.
D. Maintain the current settings.

18. An infant is receiving pressure-control ventilation. Which of the following parameters most likely will need to be adjusted first after surfactant replacement therapy?
A. T₁
B. Frequency
C. PIP
D. PEEP

19. The most important advantage of nitric oxide in the treatment of pulmonary hypertension is
A. It does not have to be analyzed.
B. It is selective in its effects.
C. It is inexpensive and easy to use.
D. It has no toxic effects.

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