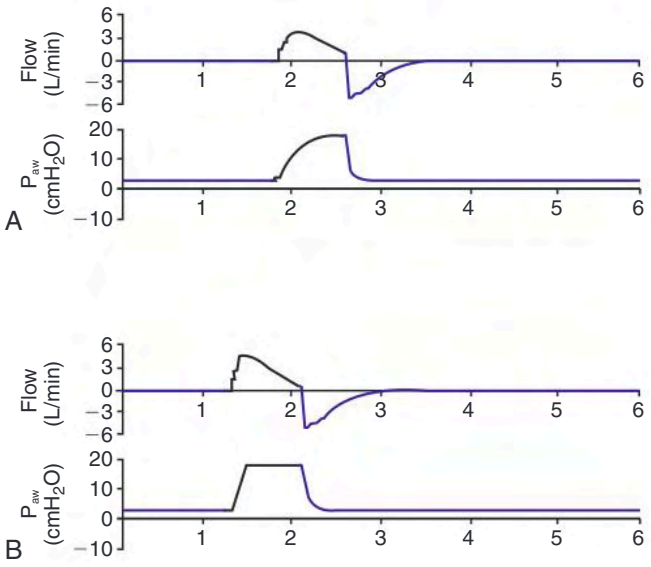

Fig. 22.9 (A) Shows a child receiving pressure-controlled continuous mandatory ventilation (PC-CMV) with a slow rise to peak inspiratory pressure because the slope setting on the CareFusion AVEA is set to 9. (B) In the same child, there is a rapid rise to peak inspiratory pressure because the slope setting is set on 1. The set inspiratory pressure is the same for both breaths.

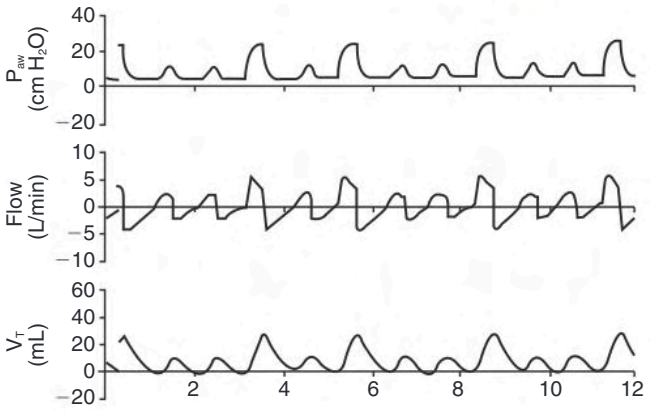

Fig. 22.10 This graphic waveform shows pressure, flow, and tidal volume in a neonate receiving patient-triggered pressure-controlled intermittent mandatory ventilation (PC-IMV) (larger inspiratory pressure breaths) with the addition of pressure-supported breaths (smaller inspiratory pressure breaths).

increases in ventilation requirement. Ventilator and monitor manufacturers have addressed this clinical problem and are including integrated $EtCO_2$/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 are described in a later section.

Traditionally, the most widely used mode of ventilation in neonates and pediatric patients is PC-CMV. The PC-CMV breath can be triggered by pressure or flow and is terminated on the basis of time. Because pressure is constant, the V_T delivery can vary widely as a result of changes in lung mechanics and respiratory effort. PC-IMV is similar to TCPL/IMV with some subtle but clinically important differences. It is usually 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. Furthermore, if a patient generates spontaneous inspiratory efforts within the breath, the demand valve opens to provide additional flow to maintain constant inspiratory pressure for the duration of the T_I. 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 in which 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 distention 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 O_2 dependency in VLBW neonates.[78]

In PC-CMV, minimizing P_{aw} is essential in patients with exceptional oxygenation. The ventilator frequency should be high enough only to reach the desired P_aCO_2, and inspiratory pressure is adjusted in increments of 1 to 2 cm H_2O to keep the monitored exhaled V_T values within an acceptable range. The inspiratory-to-expiratory ratio (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.

A brief discussion of the controls, monitoring systems, and alarms used in pressure control mode is provided in the following sections. Some of these principles can be applied to other modes of ventilation.

Inspiratory Pressure

While ventilating a child with a manual resuscitator or T-piece device with an inline airway pressure manometer, 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 O_2 saturation. Traditionally, the inspiratory pressure has been adjusted on the basis of adequate chest rise and blood gas values. Today, the

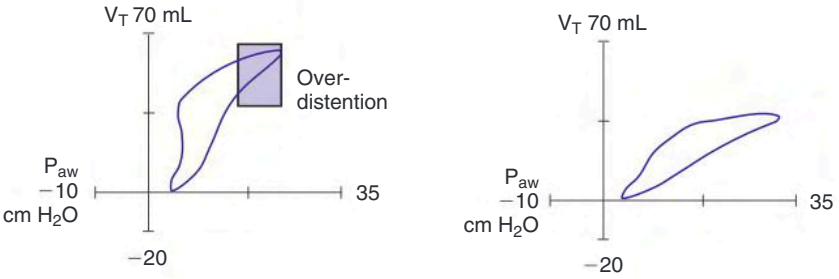

Fig. 22.11 Pressure-volume loop showing overdistention. In the pressure-volume loop on the left, the increase in volume begins to lose its linearity as the inspiratory pressure approaches its set limit. The *shaded area* indicates a small increase in volume even though pressure continues to increase. In the example on the right, a more linear waveform exists without evidence of overdistention. (From Nicks JJ: *Graphics monitoring in the neonatal intensive care unit,* Palm Springs, CA, 1995, Bird Products.)

patient is connected to the ventilator circuit and exhaled V_T 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 CO_2 elimination. Once the inspiratory pressure is set to deliver a preferred V_T, the rate is the primary adjustment for CO_2 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 at which inspiration ends and expiration begins. However, if the volume rise of the loop begins to flatten with a continued increase in the inspiratory pressure, overdistention 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 needed.[75] 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 V_T and the pressure-volume loop relationship. (See Chapter 9 for more information on pressure-volume loops.)

Positive End-Expiratory Pressure

Positive end-expiratory pressure (PEEP) is used to establish the FRC and prevent alveolar collapse. In some conditions, such as asthma, increasing PEEP may reduce R_{aw} and provide better patient triggering. The appropriate level of PEEP can greatly improve oxygenation, reduce $\dot{V}/\dot{Q}$ 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 P_aCO_2 may increase even though the minute ventilation ($\dot{V}_E$) 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 is usually set initially at 4 to 7 cm H_2O. PEEP levels above 7 cm H_2O are occasionally 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 O_2 requirement exceeds an F_IO_2 of 0.6 to maintain a P_aO_2 >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 (e.g., 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. 22.12A). Increasing the PEEP may result in a more immediate rise in volume for the pressure delivered (see Fig. 22.12B).[73] 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 V_T at the same inspiratory pressure, and an increase in PaO_2.[66]

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

Lung mechanics must be considered when the inspiratory time (T_I), expiratory time (T_E), and I/E ratio are set, especially for patients with surfactant deficiency. These patients are likely to have a low C_L with normal R_{aw}, and therefore their T_I must be short. Because R_{aw} is usually 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 constants (see Chapter 1). One (1) time constant is calculated by multiplying the R_{aw} by the C_L (Box 22.6). Historically, neonates and pediatric patients with short time constants (e.g., neonatal RDS, ARDS) have been ventilated using long T_I, and hence higher mean airway pressure, to promote better oxygenation. Although this approach may work well in pharmacologically paralyzed and sedated patients, long T_I may cause an inspiratory breath hold to occur when the patient is breathing spontaneously. In a summary of studies conducted in neonates

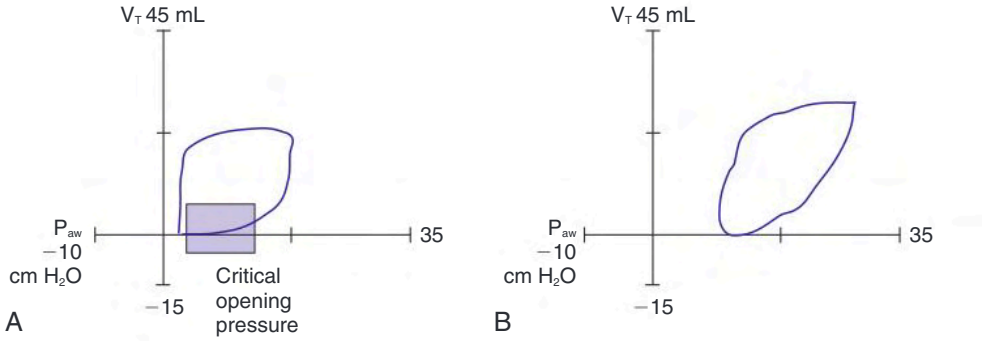

Fig. 22.12 Effects of an increase in positive end-expiratory pressure (PEEP) on critical opening pressure. (A) Volume delivery is delayed until the pressure has increased significantly, indicating a high critical opening pressure. (B) When PEEP is increased, volume delivery begins earlier in the inspiratory phase. P_{aw}, Airway pressure; V_T, tidal volume. (From Nicks JJ: *Graphics monitoring in the neonatal intensive care unit,* Palm Springs, CA, 1995, Bird Products.)

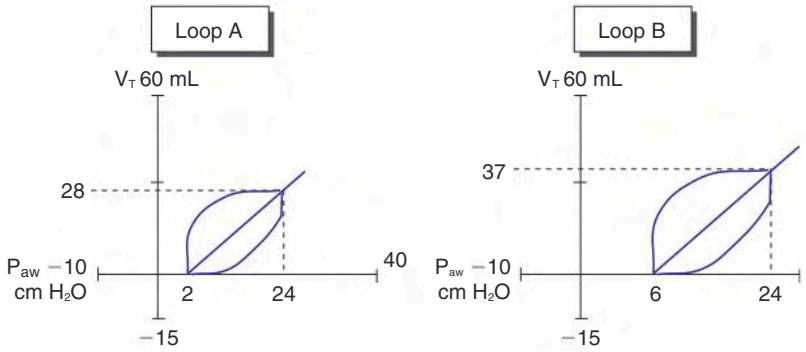

Fig. 22.13 Effect of increasing positive end-expiratory pressure (PEEP) on pressure-volume loop slope and tidal volume (V_T) (see text for more information). P_{aw}, Airway pressure. (From Wilson BG, Cheifetz IM, Meliones JN: *Optimizing mechanical ventilation in infants and children,* Palm Springs, CA, 1995, Bird Products.)

BOX 22.6 Calculation of a Time Constant

If airway resistance (R_{aw}) is 30 cm H_2O/L/s and lung compliance (C_L) is 0.004 L/cm H_2O, the time constant (TC) is calculated as follows:

$TC = R_{aw} \times C_L$

$TC = 30 \text{ cm } H_2O/L/s \times 0.004 \text{ L/cm } H_2O$

$TC = 0.12 \text{ second}$

with restrictive lung disease, long T_I was associated with a significant increase in air leak and mortality.[79]

A prolonged T_I can be identified, using airway graphics, as a period in which 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 asynchrony during PC-CMV. A breath hold can be avoided by reducing the T_I 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 T_I can be monitored during spontaneous breathing. At times a patient-triggered exhalation or flow cycling can result in a dramatic reduction in WOB and P_aCO_2. Some clinicians will leave the flow cycle parameter on, whereas others will disable it and use the previously measured T_I as the new T_I setting during PC-CMV. It is important to realize that time constants in the lungs can change rapidly; as a result, flow cycling during PC-CMV may change the T_I dramatically as compliance is reduced, resulting in lower mean airway pressure and lower V_T 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 V_T and $\dot{V}_E$ (see Fig. 22.15) during PC-CMV. In severe asthma, for example, gas-flow limitation can affect inspiratory and expiratory flow and V_T 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 $\dot{V}_E$) cannot be tolerated. Many clinicians advocate a

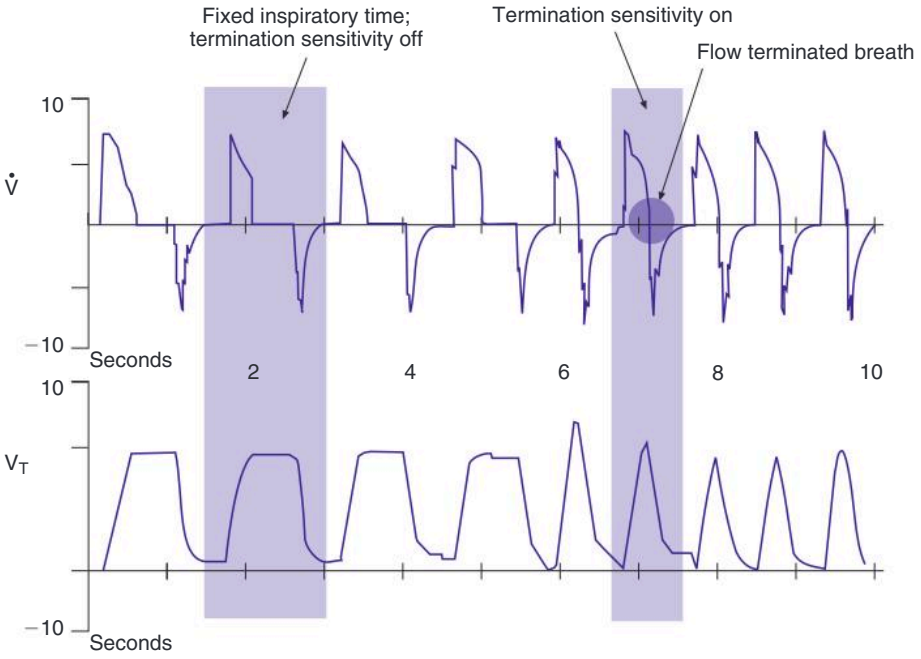

Fig. 22.14 Assist/control (A/C) pressure-controlled ventilation using the flow cycle feature. The flow cycle is off for the first four breaths, and each breath is time cycled. When flow cycle is activated, the breath terminates when a predetermined decrease in flow is sensed at the airway. V_T, Tidal volume. (Modified from Nicks JJ: *Graphics monitoring in the neonatal intensive care unit,* Palm Springs, CA, 1995, Bird Products.)

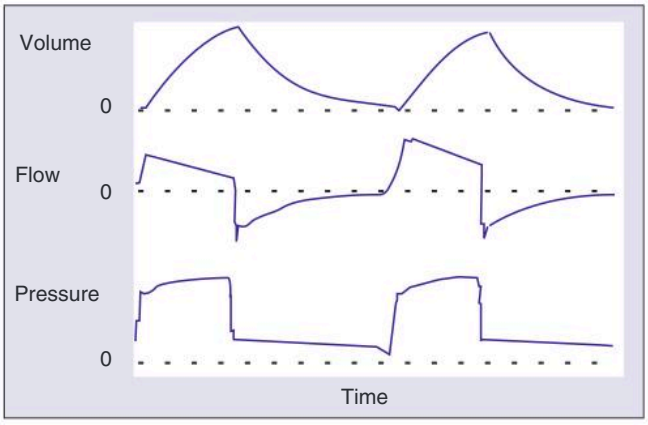

Fig. 22.15 Pressure-controlled ventilation with severe inspiratory airflow limitation. Note that with each breath, the peak pressure is reached and the inspiratory phase time cycles before flow can decelerate to zero.

permissive hypercapnia ventilation strategy in severe reactive airway disease (discussed later). To accomplish this, a small V_T is selected, in addition to a low $\dot{V}_E$, a low rate, a T_I sufficient to eliminate flow chop, and a T_E 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 $\dot{V}_E$ 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 internal-diameter endotracheal tube and placed on a CareFusion AVEA ventilator. He has been paralyzed and sedated and is receiving ventilation with the pressure control mode with a 60/40 helium-O_2 mixture. Ventilator settings are PIP/PEEP = 24/5 cm H_2O, respiratory rate = 16 breaths/min, inspiratory time = 0.9 seconds.

The patient's expired V_T is 3 mL/kg. The end-tidal partial pressure of carbon dioxide ($P_{et}CO_2$) is 92 mm Hg, and the S_pO_2 is 88%. The respiratory therapist (RT) has increased inspiratory pressure in increments of 2 cm H_2O to 32 cm H_2O, but the V_T has not changed. An ABG sample has been sent to the laboratory. What additional monitoring should the RT consider with this patient? What other setting changes should the RT recommend?

Nearly complete equilibration of alveolar pressures (P_{alv}) occurs in three to five time constants (Fig. 22.16). In infant lungs with normal mechanics, equilibration occurs in at least 0.6 seconds (time constant × 5 = (R_{aw} × C_L) × 5 = (30 cm H_2O/L/s × 0.004 L/cm H_2O) × 5 = 0.6 second). Less time is needed for lung inflation in surfactant-deficient lungs, in which the time constant

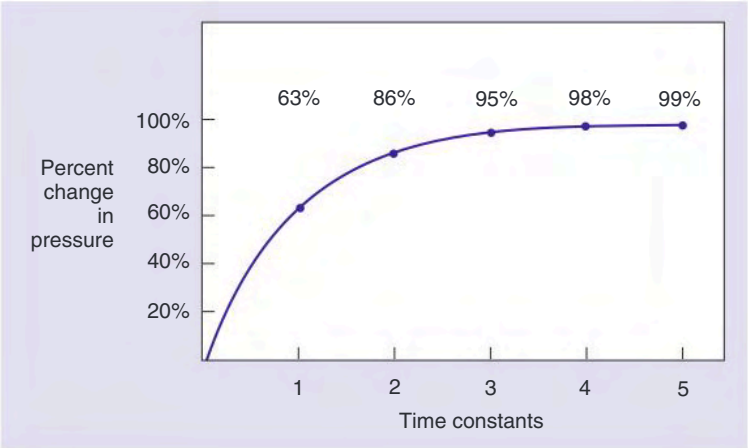

Fig. 22.16 Percentage change in pressure in relation to time (in time constants) allowed for equilibration. As the time allowed for equilibration increases, so does the percentage change in pressure. The same applies to the equilibration for changes in volume.

is shorter. Therefore the T_I can be set for a short interval and the respiratory frequency can be set high with less concern for breath stacking and hyperinflation.[67]

The concept of time constants can be easily 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_I and T_E 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_I and T_E 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_I is 0.25 second.[77] On the other hand, when acute lung disease makes adequate oxygenation difficult, lengthening the T_I and increasing the P_{aw} can increase the P_aO_2.[78] **Bronchopulmonary dysplasia** (BPD) is an example of a pulmonary disease of infants in which high R_{aw} is a major component. Time constants for BPD are estimated to be as high as 0.5 seconds.[77] 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 R_{aw} 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 is usually 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 HFV. Inverse ratios are rarely used in PC-CMV because of the risk for hyperinflation and lung trauma. Waveform monitoring is useful for determining the most appropriate T_I and T_E 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_E 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.[80]

Tidal Volume

V_T is not a set parameter in the PC-CMV mode. Mechanical V_T depends on T_I, 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 expired V_T (V_{Texh}). This loss of volume often is expressed as the *percent leak*, which can be calculated with the following formula:

$$\text{Percentleak} = \left[\left(V_{Tinsp} - V_{Texh}\right) / V_{Tinsp}\right] \times 100$$

where V_{Tinsp} is the inspired V_T and V_{Texh} is the expired (exhaled) V_T.

Some ventilators calculate and display the percent leak. Other monitors display V_{Tinsp} and V_{Texh}. Volume monitoring also provides an important safety measure by alerting clinicians to sudden drops in expired $\dot{V}_E$. 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 in addition to low V_T and low exhaled $\dot{V}_E$ 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

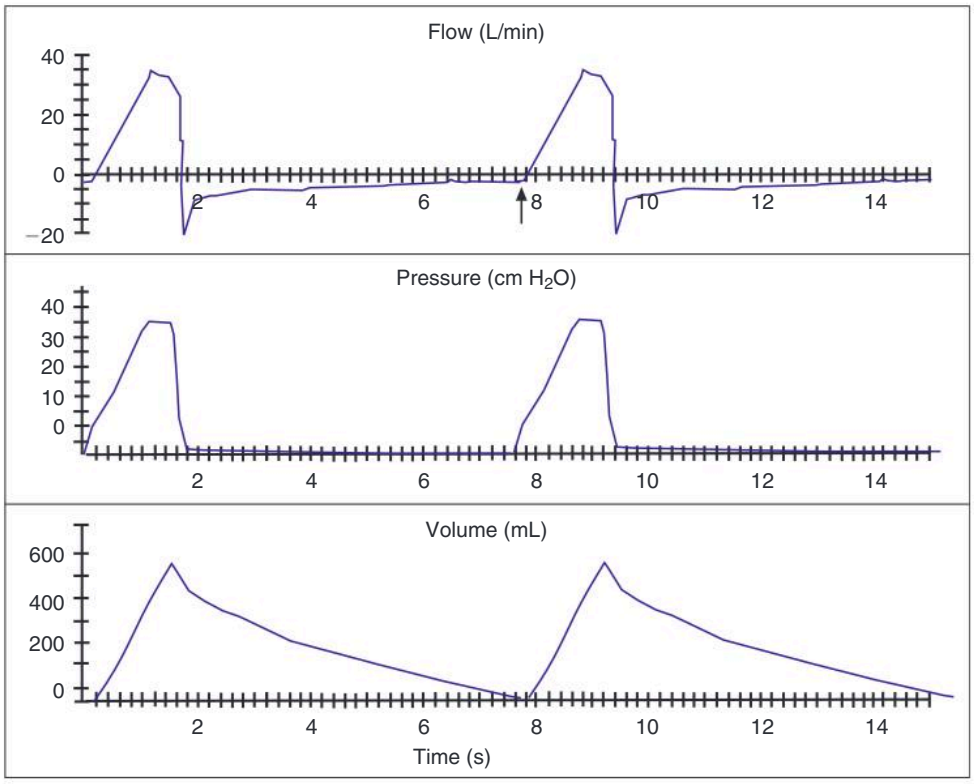

Fig. 22.17 Prolonged expiratory flow pattern in a patient with autopositive end-expiratory pressure (auto-PEEP). Prolonged expiratory flow can lead to breath stacking and hyperinflation. Expiratory gas flow shows an initial spike downward and then changes to a low level of flow, which continues throughout the expiratory phase. Expiratory flow does not return to baseline before the next breath. (From Kacmarek RM, Stoller JK, Heuer AJ: *Egan's fundamentals of respiratory care,* ed 12, St. Louis, MO, 2021, Elsevier.)

as an initial inspiratory pressure can be determined by clinical assessment. Trying out different frequencies can help determine the initial setting to achieve the best S_pO_2 and vital signs. Airway graphics are a helpful resource in determining the proper frequency setting. Higher rates can result in gas trapping, so complete exhalation should be noted on flow scalars when setting the initial frequency and with subsequent adjustments. However, a blood gas or a transcutaneous CO_2 monitor that correlates well with P_aCO_2 is the standard method used to adjust frequency. Noting the patient's $\dot{V}_E$ and relating it to the P_aCO_2 is important. Once the initial P_aCO_2 is known, it, along with the desired P_aCO_2, can be used to calculate an appropriate change in V_T 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 CO_2 monitors can provide trending information about alveolar ventilation. Even though transcutaneous CO_2 may not correlate all of the time, a rapid change in transcutaneous CO_2 could warn clinicians of a serious condition, whereas a blood gas sample may take time to get results. For instance, a gradual reduction in transcutaneous CO_2 after surfactant administration may alert the clinician to observe exhaled V_Ts and compliance and possibly wean inspiratory pressures during PC-CMV. Monitoring of the transcutaneous partial pressure of CO_2 ($P_{tc}CO_2$) 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 HFV (see the section on HFV later in the chapter).

Mean Airway Pressure

Conventional ventilators do not have a P_{aw} setting; rather this is a monitored parameter that must be closely watched. Increases in P_{aw} can greatly improve oxygenation but also reduce venous return and cardiac output. P_{aw} levels >12 cm H_2O have been associated with lung injury. The P_{aw} is directly affected by PIP and PEEP, inspiratory hold, frequency, T_I, and flow.

Inspired Oxygen Concentration

An F_IO_2 higher than 0.6 is avoided as much as possible in pediatric patients to prevent O_2 toxicity. This concern is even greater for premature infants because of the role of O_2 in developing retinopathy of prematurity. Tissue O_2 delivery is as important as F_IO_2 in ventilator management. Maintaining a hematocrit of more than 40%, even in premature infants, maximizes the blood's O_2-carrying capacity and augments the oxygenating effects of PEEP, P_{aw}, and F_IO_2.

P_aO_2 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 P_{aw} values and an F_IO_2 of 0.6.

One ventilator (AVEA, CareFusion) has implemented a closed-loop F_IO_2 algorithm wherein the ventilator automatically titrates the F_IO_2 based on a measured O_2 saturation and preset O_2 range (i.e., 88%–92%). This may be a useful system for managing oxygenation, but few trials have evaluated the effectiveness of closed-loop F_IO_2 in reducing adverse outcomes in patients during mechanical ventilation. At the time of writing, closed-loop F_IO_2 has not been approved by the U.S. Food and Drug Administration (FDA).

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 V_T 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 V_T 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 V_T, rather than inspiratory pressure, to be set. Thus the measured inspiratory pressure will vary on the basis of lung mechanics and patient effort. T_I is a function of the set V_T 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 V_T, whereas other ventilators require the clinician to set the flow and the T_I depends 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. Thus manufacturers 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. Increasing the flow or reducing the T_I setting can alleviate asynchrony. 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 and IMV or VC-CMV and VC-IMV, respectively.

Breaths can be patient triggered 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 V_T, flow, and T_I. During VC-IMV, PSV breaths can be added to support spontaneous breaths, but during VC-CMV all the breaths are supported with the preset V_T. Patients receiving VC-CMV should be monitored closely for clinical signs of hypocapnia and hyperinflation, especially when the patient is agitated or autotriggering the ventilator as a result of a large ET tube leak. In theory, V_T does not vary with changing C_L or R_{aw} during VC-CMV; however, V_T may decrease if the ventilator cannot correct for volume losses resulting from gas compression in the patient circuit. Delivered V_T 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 V_T. Failure to consider this volume loss may result in hypoventilation and hypercapnia.[81]

Infants who undergo cardiothoracic or abdominal surgery are often placed on VC-IMV because changes in C_L and abdominal distention do not affect V_T delivery. Unlike pressure-controlled ventilation, there is a slower rise to the PIP during volume-controlled ventilation, and hence lower mean airway is obtained for the same V_T. Transitioning from pressure-controlled ventilation to volume-controlled ventilation may be preferable in patients who have hemodynamic compromise or do not tolerate higher mean airway pressure.

Pressure Support Ventilation

Pressure support ventilation (PSV) is strictly a spontaneous mode or form of continuous spontaneous ventilation (CSV) that is used to augment a patient's V_T 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 T_I, 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. Furthermore, if a patient becomes apneic on the basis of a preset apnea interval, the ventilator will provide backup ventilation.

PSV is 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 not only provide inspiratory pressure sufficient to overcome tube resistance but also allow the patient's lung and chest wall mechanics to determine V_T. Analysis of a patient's pressure-volume and flow-volume loops while on PSV provides some indication of the effort required to overcome artificial R_{aw} (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).[69]

Initially a higher level of pressure is often needed to enable these patients to achieve V_T in the range of 4 to 7 mL/kg. Over time, the PSV level can be reduced if the patient maintains a satisfactory V_T, respiratory rate, and S_pO_2. 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 <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. 18.10). 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 V_T does not occur and patient-ventilator asynchrony may result. When PPST is suspected, a slower rise time can be adjusted and this may reduce

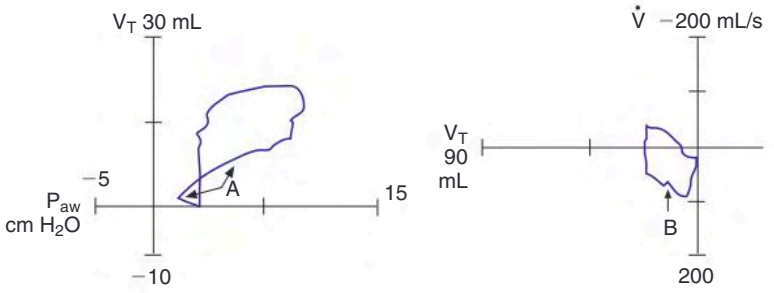

Fig. 22.18 Flow asynchrony arising from inadequate pressure support. Note the "figure 8" pattern *(double arrow)* in the pressure—volume loop (A) and the notching on the inspiratory limb *(arrowhead)* in the flow—volume loop (B). These signs indicate that gas flow is inadequate to overcome artificial airway pressure (R_{aw}). The pressure support level is 6 cm H_2O. P_{aw}, Airway pressure; $\dot{V}$, flow; V_T, tidal volume. (From Wilson BG, Cheifetz IM, Meliones JN: *Optimizing mechanical ventilation in infants and children,* Palm Springs, CA, 1995, Bird Products.)

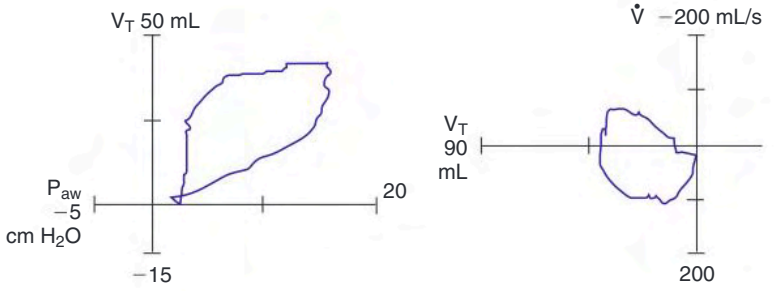

Fig. 22.19 Same patient as in Fig. 22.18; flow synchrony has improved with the appropriate level of pressure support. Pressure support has been increased to 13 cm H_2O. The figure 8 pattern in the pressure-volume loop *(left)* and the notching on the inspiratory limb in the flow-volume loop *(right)* have been eliminated. P_{aw}, Airway pressure; $\dot{V}$, flow; V_T, tidal volume. (From Wilson BG, Cheifetz IM, Meliones JN: *Optimizing mechanical ventilation in infants and children,* Palm Springs, CA, 1995, Bird Products.)

or eliminate it. This problem was frequently seen in the past when adult ventilators were used in pediatric patients 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 TT 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 flow cycling is sometimes desirable if the tube leak is so 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

Recommending Changes in Ventilator Settings

A 1-month-old prematurely born baby boy with a diagnosis of respiratory syncytial virus (RSV) pneumonia is receiving PC-CMV. The patient's initial measured V_T was about 5 mL/kg with a respiratory rate of 40 to 60 breaths/min, the S_pO_2 was 95% on an F_IO_2 of 0.3, and $\dot{V}_E$ was 0.28 L. Over several hours, V_T diminishes to about 2 to 3 mL/kg and the respiratory rate increases to over 100 breaths/min. The S_pO_2 decreases to about 92%, but the $\dot{V}_E$ remains unchanged. What change in ventilator settings is necessary for this patient?

Dual-Control Mode

The dual-control mode is an adaptive form of pressure-controlled ventilation that can be used with CMV, IMV, and PSV breaths. It combines the best features of pressure and volume modes to provide a minimum V_T during ventilation. Dual-control breaths can be patient triggered on the basis of 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 on the basis of time or once the peak flow has decelerated to a preset value. The V_T is preset, and the inspiratory pressure level will vary on the basis of 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 on the basis of 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 V_T, 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 V_T 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 difficult for dual-control modes to provide a precise V_T with an ET leak 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

A widely used form of dual-control ventilation in neonates and pediatric patients is pressure-regulated volume control (PRVC), used commonly 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 H_2O 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.[73] It should be noted that during PRVC, the inspiratory pressure is usually adjusted on the basis of the monitored inspiratory V_T. 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 (Fig. 22.21).[81-83]

Alarms should be adjusted properly, and patients should be monitored frequently for signs of respiratory distress. Additionally, in some ventilators, inspiratory V_Ts 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. Generally speaking, ventilator-displayed V_T, without circuit compensation, generally overestimates true-delivered V_T, and with circuit compensation, it generally underestimates true-delivered V_T.[84,85] If a proximal flow sensor is not available in neonates, then a tubing compliance factor should be used to improve V_T delivery.

Volume Guarantee

Volume guarantee is yet another variation of PRVC that is used primarily in neonates. Available on the Dräger Babylog models 8000plus and VN500 (Dräger Medical, Luebeck, Germany), the volume guarantee setting allows a set V_T target while maintaining either the pressure control mode or PSV mode and its characteristic waveforms. Volume guarantee with the Dräger Babylog model 8000plus adjusts inspiratory pressure on a breath-by-breath average based on an expiratory V_T measurement obtained from a hot-wire flow sensor at the patient. Similar to time cycle, pressure-limited/IMV ventilators, the operator must set a continuous flow to maintain pressure and V_T 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_T and will increase pressure on the basis of these measurements up to the pressure limit to deliver the target volume (Fig. 22.22).[86]

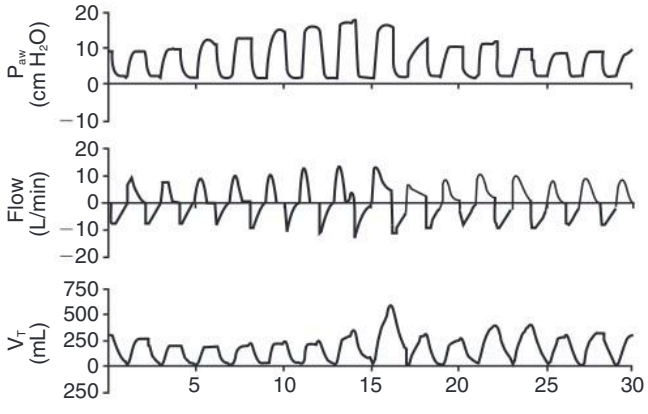

Fig. 22.20 Pressure-regulated volume control (PRVC) mode being used in a pediatric patient with acute respiratory distress syndrome (ARDS). The flow and pressure vary from breath to breath, with changes in respiratory system compliance as the ventilator attempts to maintain a minimum tidal volume.

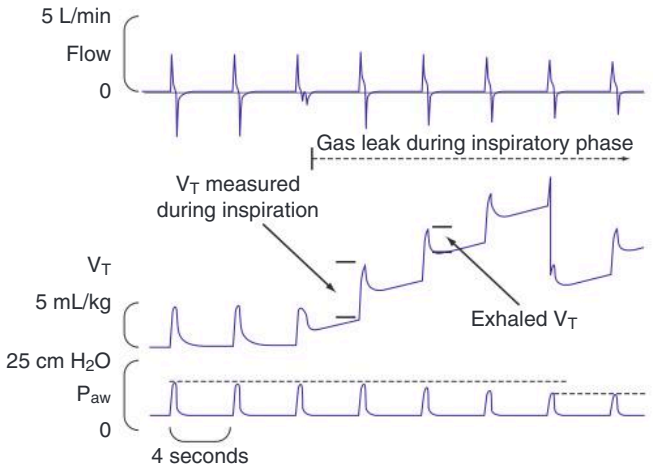

Fig. 22.21 Effects of leaks during the inspiratory phase of pressure-regulated volume-controlled (PRVC) ventilation. (From Claure N, Bancalari E: Methods and evidence on volume-targeted ventilation in preterm infants, *Curr Opin Pediatr* 2008;20:125–131.)

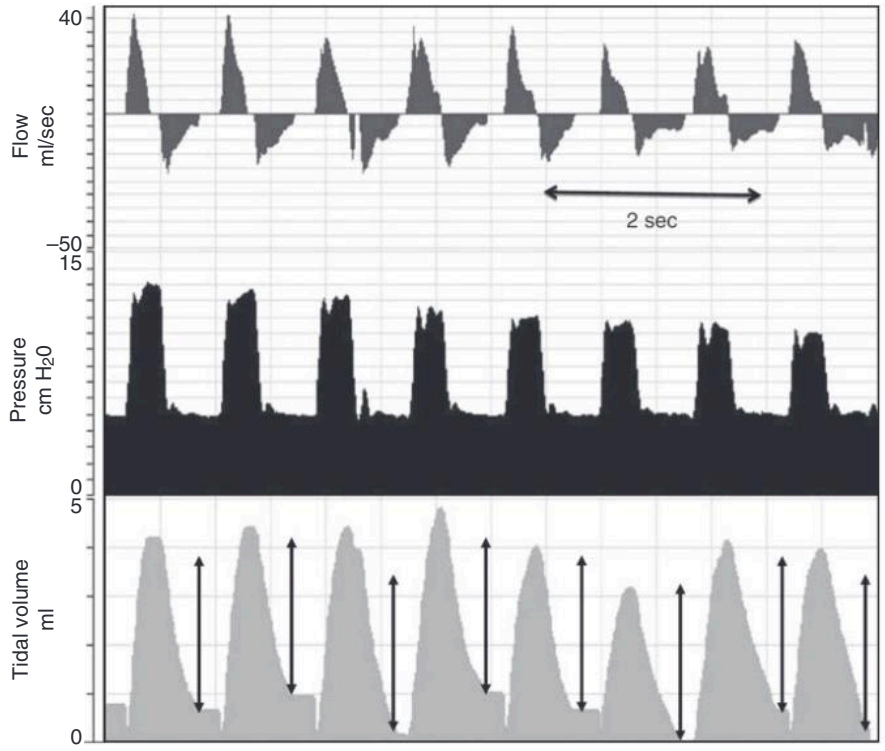

Fig. 22.22 Volume guarantee breaths illustrating flow, pressure, and tidal volume (V_T) waveforms for triggered inflations during volume-controlled continuous mandatory ventilation (VC-CMV). The *vertical arrows* show the set V_T. Notice that the expired V_T is larger than the set V_T for all breaths and the peak inspiratory pressure (PIP) is reduced for each subsequent breath. (From Klingenberg C, Wheeler KI, Davis PG, Morley CJ: A practical guide to neonatal volume guarantee ventilation, *J Perinatol* 2011;31:575–585.)

If lung mechanics improve dramatically, the ventilator will terminate breath delivery if the delivered V_T exceeds 130% of the set V_T. Pressure will also wean as the result of improving compliance-based V_T on the breath average. Because the ventilator makes manipulations on the basis of expiratory V_T, 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_T delivered to the lung and overcompensate the subsequent breaths with excessive V_T.

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.[83] (See Case Study 22.5.)

A new form of volume guarantee provided by the Dräger Babylog VN500 (Dräger Medical) uses an algorithm that adjusts inspiratory pressure based on a calculated leak during inhalation. Therefore infants with ET tube leaks >50% could be supported by volume guarantee on the VN500 more effectively than a ventilator that uses inspiratory or expiratory V_Ts to guide pressure adjustments. Also, when an infant contributes to volume delivery during a triggered inflation, the inspiratory pressure is lower than the untriggered breaths. This may prevent overdistention of the lungs (Fig. 22.23).[85,87]

This mode requires the clinician to set a maximum inflation pressure limit (P_{max}) that alarms once a pressure of 25 to 30 is being met. If the patient volume exceeds the inspiratory preset V_T (because of agitation or improvement in the lung condition), inspiratory pressures will wean (Fig. 22.24).[85] In some cases, the inspiratory pressure could be equal to the PEEP level. If the pressure is consistently low and the patient appears stable, clinicians may assess for weaning; otherwise, if the patient has high WOB with low inspiratory pressures and is not ready for extubation, increasing the V_T gradually in small increments may reduce the WOB and stabilize the patient.[87] This phenomenon is

Case Study 22.5

Evaluation of Pressure-Regulated Volume Control (PRVC) Dual-Control Mode

A respiratory therapist is setting PRVC on a premature neonate patient who is recovering from RDS breathing spontaneously. The neonate is stable while receiving PC-CMV on the Servo-i ventilator. The peak inspiratory pressure is 20 cm H_2O. The patient has a large endotracheal leak (60%). The ventilator is switched to PRVC with a V_T of 5 mL/kg. Within 2 hours, the patient's respiratory rate increases to 80 beats/min and the F_IO_2 is increased by 40% to maintain adequate O_2 saturations. What are some reasons why the patient is doing poorly on PRVC mode?

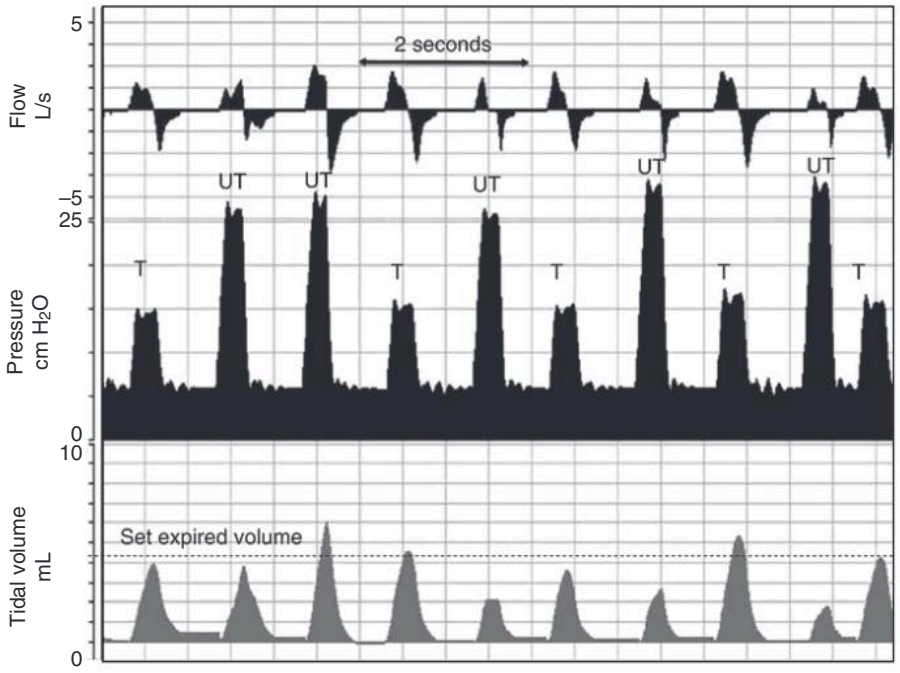

Fig. 22.23 Flow pressure and volume waveforms from an 850-g neonate showing the effects of triggered and spontaneous inflations. Notice that the breaths occur close to each other because the ventilator backup rate is set too close to the neonate's spontaneous rate. The untriggered breaths are indicated as *UT*. The inflating pressure for each breath depends on the expired tidal volume of the preceding inflation. Notice that a large difference exists between inflation pressures although there is a relatively small difference in tidal volume delivery. (From Klingenberg C, Wheeler KI, Davis PG, Morley CJ: A practical guide to neonatal volume guarantee ventilation, *J Perinatol*. 2011;31:575—585.)

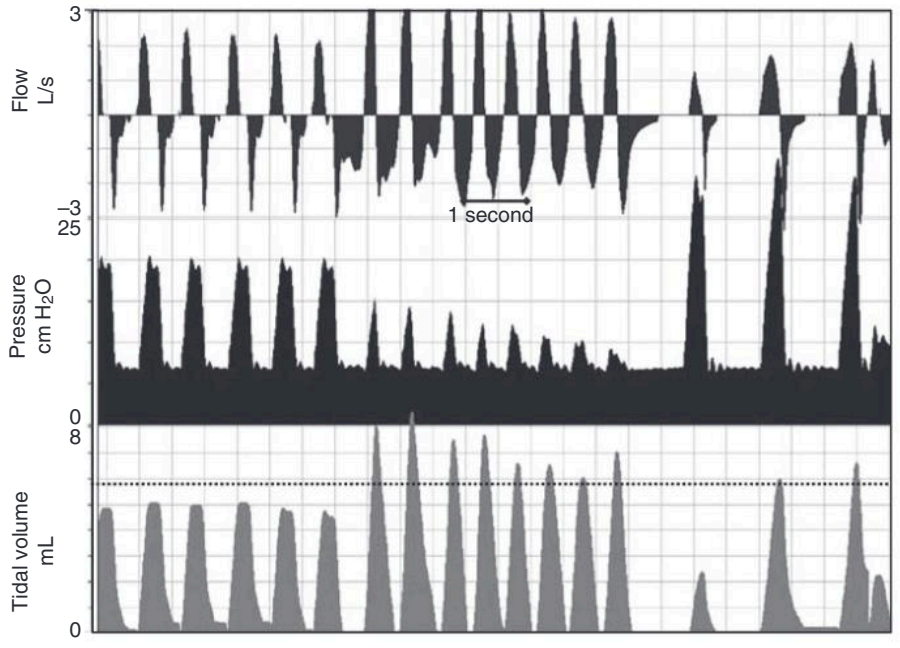

Fig. 22.24 Pressure-controlled continuous mandatory ventilation (PC-CMV) with volume guarantee of a 1000-g neonate illustrating the effect of the large spontaneous breaths. During the first six breaths, the baby is breathing quietly and triggering each breath. During the next eight breaths, the baby demonstrates increased inspiratory efforts and the inspired tidal volume (V_T) exceeds the set V_T shown by the *horizontal dotted line*. During the remaining three breaths, the baby stops making inspiratory efforts and three backup ventilator (timed) breaths are delivered. (From Klingenberg C, Wheeler KI, Davis PG, Morley CJ: A practical guide to neonatal volume guarantee ventilation, *J Perinatol*. 2011;31:575—585.)

prevalent when the V_T has not been adjusted as the patient grows or when the patient develops chronic lung disease. It is important to update the patient weight at least on a weekly basis so that measured V_Ts reflect the appropriate V_Ts in milliliters per kilogram. If the WOB still continues to be high, placing the patient onto pressure control mode may be a better option. However, some ventilators allow a minimum pressure setting during DC-CMV or DC-PSV.

Volume Support Ventilation

Volume support ventilation (VSV) is well suited for infants and pediatric patients. Its use in infants is similar to that of 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_T or $\dot{V}_E$, 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, 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 TT 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_T 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_T is to be achieved by patient effort. Some practitioners prefer switching to CPAP or pressure support 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 are required. The mode has been referred to as *CPAP with releases* (Fig. 22.25). 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 H_2O. 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} and allows recruitment of air spaces. The P_{high} is held in the lung for up to 2 seconds for neonates and 4 seconds for pediatric patients.[88] Spontaneous breathing at a higher pressure aids in not only alveolar recruitment but also through the application of pleural pressure change and makes improvements in the distribution of lung volume to diseased lung units that improve FRC and pulmonary compliance.[70,83] APRV has been used in neonatal, pediatric, and adult forms of respiratory failure, but few studies have been performed in neonates and pediatric patients to ascertain specific management protocols.

Neurally Adjusted Ventilatory Assist

Neurally adjusted ventilator assist (NAVA) allows the patient full neurological control of the triggering, magnitude, and timing of the mechanical support provided, regardless of changes in respiratory drive, mechanics, and muscle function.[89] 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.[90] 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 potentially avoided. In neonates this mode has been shown to provide better comfort and less need for sedation than PRVC.[91] However, this modality is invasive, and placement of the nasogastric tube must be evaluated to ensure proper function of this modality. Additional information about the application of NAVA can be found in Chapter 23.

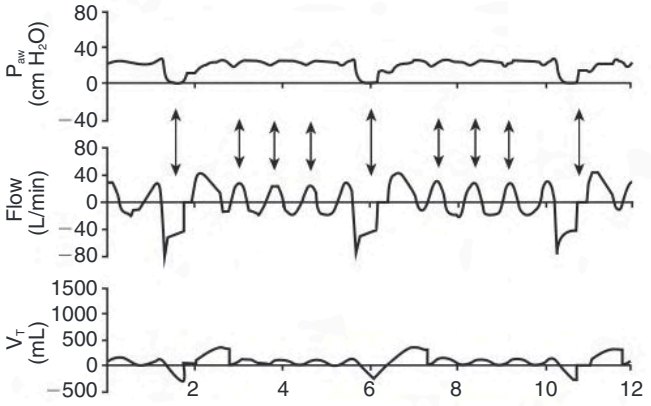

Fig. 22.25 A 50-kg patient with severe acute respiratory distress syndrome breathing spontaneously during airway pressure release ventilation. *Large arrows* indicate pressure releases. *Small arrows* indicate spontaneous breathing at P_{high}.

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, are 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 overinflation. Therefore 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.

TABLE 22.1 Lung-Protective Ventilation Strategies for Neonatal Lung Disorders

Lung Disease	Ventilator Settings	Blood Gas or S_pO_2 Targets
Respiratory distress syndrome (RDS)	1. PC, VC, or DC ventilation to target V_T 4–6 mL/kg 2. Rapid rates ≥60 breaths/min 3. Moderate PEEP (4–5 cm H_2O) 4. Short inspiratory time 0.25–0.4 second	1. pH 7.25–7.35 2. P_aCO_2 45–55 mm Hg 3. P_aO_2 50–70 mm Hg 4. S_pO_2 88%–94%
Meconium aspiration syndrome (MAS)	1. PC, VC, or DC ventilation with lowest PIP to maintain adequate chest excursion 2. Relatively rapid rates (40–60 breaths/min) 3. Moderate PEEP (4–6 cm H_2O) 4. Short inspiratory time to allow exhalation time (0.5–1 second) 5. Sedation, neuromuscular paralysis, and inhaled NO (20 ppm)	*Without PPHN* pH 7.3–7.4 P_aCO_2 40–50 mm Hg P_aO_2 70–80 mm Hg S_pO_2 >90% *With PPHN* pH 7.30–7.4 P_aCO_2 35–45 mm Hg P_aO_2 80–100 mm Hg S_pO_2 >95%
Congenital diaphragmatic hernia (CDH)	1. PC, VC, or DC ventilation with lowest PIP to maintain adequate chest excursion 2. Rapid rates (40–80 breaths/min) 3. Moderate PEEP (4–5 cm H_2O) 4. Short inspiratory time (0.3–0.5 second)	1. pH >7.25 2. P_aCO_2 45–55 mm Hg 3. P_aO_2 50–70 mm Hg 4. S_pO_2 >95%
Persistent pulmonary hypertension of the newborn (PPHN)	1. PC, VC, or DC ventilation with lowest PIP to maintain adequate chest excursion 2. Higher rates (50–70 breaths/min) 3. Low PEEP (3–4 cm H_2O) 4. Inspiratory time (0.3–0.5 second) 5. Inhaled NO (20 ppm)	1. pH 7.35–7.45 2. P_aCO_2 30–40 mm Hg 3. P_aO_2 70–100 mm Hg 4. S_pO_2 >95%
Bronchopulmonary dysplasia	1. PC, VC, or DC ventilation to maintain V_T (5–8 mL/kg) 2. Slow rates (20–40 breaths/min) 3. Moderate PEEP (4–6 cm H_2O) 4. Inspiratory time (0.4–0.7 second)	1. pH 7.25–7.35 2. P_aCO_2 45–55 mm Hg 3. P_aO_2 50–70 mm Hg 4. S_pO_2 range

DC, Dual-controlled ventilation; *PC,* pressure-controlled ventilation; *PEEP,* positive end-expiratory pressure; *PIP,* peak inspiratory pressure; *VC,* volume-controlled ventilation; V_T, tidal volume.

From Logan JW, Cotten CM, Goldberg RN, Clark RH: Mechanical ventilation strategies in the management of congenital diaphragmatic hernia, *Semin Pediatr Surg* 16:115–125, 2007; Goldsmith JP: Continuous positive airway pressure and conventional mechanical ventilation in the treatment of meconium aspiration syndrome [review], *J Perinatol* 28(suppl 3):S49–S5, 2007; Vitali SH, Arnold JH: Bench-to-bedside review: ventilator strategies to reduce lung injury lessons from pediatric and neonatal intensive care, *Crit Care* 9:177–183, 2005; Ambalavanan N, Schelonka RL, Carlo W: Ventilatory strategies. In Goldsmith JP, Karotkin EH, editors: *Assisted ventilation of the neonate,* ed 4, Philadelphia, PA, 2004, WB Saunders, pp 249–259.

As previously discussed, compelling evidence now suggests that volutrauma created by excessive volumes, and not necessarily barotrauma, is chiefly responsible for instigating VILI.[92,93] Even short-term exposure to volutrauma during mechanical ventilation initiates lung inflammation in premature infants, which can occur after only a few minutes of manual resuscitation.[94] Ventilation for 15 minutes with a V_T of 15 mL/kg has been shown to cause an injurious process in the preterm lung.[95] 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.[96] Critical underinflation, using small V_T (atelectrauma), can also contribute to VILI.[97] Furthermore, VILI can put premature neonates with RDS at a greater risk for arrested lung growth and development.[98]

Over the past 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 with volume-targeted modes, neonates supported with volume-targeted modes had significantly lower duration of ventilation, pneumothorax, hypocarbia, severe intraventricular hemorrhage, periventricular leukomalacia, and the combined outcome of death or BPD than infants supported with pressure control modes.[99,100]

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 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 O_2 saturations, suggesting substantially higher WOB compared with VC-CMV.[101] During weaning and when applicable, it has been suggested that volume-targeted strategies be implemented using pressure support so that infants can determine their own inspiratory time.[7]

At present, it is unclear what the absolute target V_T or target range should be 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 LBW neonates.[99] 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 group.[102] A V_T target of 3 mL/kg has been associated with increased alveolar dead space, tachypnea, and higher transcutaneous CO_2 in preterm infants compared with higher V_T targets (5 mL/kg).[103] In larger infants with established chronic lung disease, larger V_T targets (above 7 mL/kg) may be needed to reduce high WOB associated with increased anatomical deadspace.[104]

Larger infants and pediatric patients with acute lung injury (ALI) 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.[67] Studies have reported reduced mortality in adults when lung-protective strategies are used.[105,106] Reduction of barotrauma, volutrauma, and atelectrauma are thought to be the reasons (see Chapter 17).[107,108] Repeated collapse and inflation result in stress injury to alveolar and pulmonary vascular tissue and loss or alteration of surfactant.[107]

Adult studies have had a dramatic effect on the management of pediatric patients with ARDS. Central to these strategies is the use of a V_T <6 mL/kg, plateau pressures (P_{plat}) <30 cm H_2O, and appropriate levels of PEEP in patients with ARDS. PEEP itself has been shown to have lung-protective effects during mechanical ventilation (Case Study 22.6).[106]

Mechanical ventilation has the potential to create dynamic hyperinflation (auto-PEEP) in patients affected by diseases of airflow limitation (e.g., asthma, bronchiolitis, 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 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 V_T, appropriate PEEP levels, and low P_{plat} and to allow permissive hypercapnia (i.e., an increased P_aCO_2). Maintaining adequate PEEP, low inspiratory pressures (P_{plat} <30 cm H_2O), and low volumes also reduces alveolar shear injury and overdistention. Additionally, using a short T_I and prolonging the expiratory phase of each mechanical breath allows more time for exhalation but results in a low respiratory rate. Incorporation of all these strategies usually makes an increase in P_aCO_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 P_aCO_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 CO_2 may actually have an antiinflammatory effect. (NOTE: Inflammation is the biochemical complication associated with overstretching of the lung [see Chapter 17].)[108-112]

Case Study 22.6

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 peak inspiratory pressure = 26 cm H_2O, positive end-expiratory pressure (PEEP) = 6 cm H_2O, inspiratory time = 0.8 second, and respiratory rate = 16 breaths/min. The F_IO_2 is 1.0. Arterial blood gas values are: P_aO_2 = 55 mm Hg, P_aCO_2 = 73 mm Hg, and pH = 7.19. An inline Cosmo Plus monitor shows a $\dot{V}CO_2$ of 83 mL/min. (See Chapter 10 to review volumetric CO_2 monitoring.)

Guided by chest radiographic findings, the attending physician and the respiratory therapist decide to increase the PEEP to 8 cm H_2O. Soon after making the change, they note that the $\dot{V}CO_2$ has risen and is now at 85 mL/min. What action should the physician and respiratory therapist take?

HIGH-FREQUENCY VENTILATION

Emerson introduced the first high-frequency ventilator in 1959, and many attempts have been made since then to apply various forms of this type of ventilation to a wide range of patients.[113] 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.[114] Among LBW infants who survived RDS, 25% to 33% eventually developed BPD.[71]

Minton and colleagues used the term *pulmonary injury sequence (PIS)* to describe the issue of prematurity and pulmonary disease, or the "continuum of disease."[115] The continuum of PIS includes RDS, PIE, pulmonary air-leak syndrome, O_2 toxicity, and BPD. The extent to which HFV 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 P_{aw} than with conventional ventilation. Even at a higher P_{aw}, lung injury is less likely because high peak pressures can be avoided.

HFV can be used in conjunction with surfactant therapy. Studies have demonstrated that lung injury can be reduced with HFV if early recruitment of optimal lung volumes is achieved and maintained after surfactant administration.[116] This has prompted some clinicians to apply an early intervention strategy to the management of premature infants: HFV is initiated and the first dose of exogenous surfactant is given within the first few hours of life.

Another problem with conventional ventilation in both LBW infants and older pediatric patients is ineffective gas exchange despite extremely high settings. This most often occurs in 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 P_{aw} in not

BOX 22.7 Conditions for Which High-Frequency Ventilation Is Used in Infants and Children

- Homogenous lung disease requiring conventional P_{aw} over 15 cm H_2O
- 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

only newborns but also 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., RDS/ARDS) if the P_{aw} on conventional ventilation exceeds 15 cm H_2O. A change from conventional ventilation to HFV may be seriously considered at a lower P_{aw} 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 O_2 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 may also be considered for patients with sepsis, persistent pulmonary hypertension of the newborn, or congenital diaphragmatic hernia when a high P_{aw} 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) have historically been considered poor candidates for HFV because of the risk for overinflation. However, some recent data suggest that high-frequency oscillatory ventilation (HFOV), a form of HFV, may be safe and effective in patients with small airways disease (e.g., bronchiolitis), hyperinflation, and hypercapnia. 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.

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 in addition to 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-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, crystalloids 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, heart rate, and central venous pressure for adverse hemodynamic effects is 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.[71] Presumably this is caused by 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.[116] Recent data have indicated that when HFOV is used as an initial ventilation strategy, neurodevelopmental outcomes were actually improved.[117] Some have suggested that a low P_aCO_2 is a primary cause of IVH in premature infants.[116] A possible explanation is that HFV can dramatically reduce the P_aCO_2 before the clinician is aware of this development.[113] This is why trending P_aCO_2 levels, discussed later in this section, are essential during HFV.

High-Frequency Ventilation Techniques

As its name implies, HFV is a form of mechanical ventilation that uses high respiratory rates, or *frequencies.* Frequencies are usually specified in hertz (Hz) or cycles per second; 1 Hz equals 60 cycles/min or 60 breaths/min. Under guidelines established by the 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 is usually 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 was also effective for pediatric patients with surfactant deficiency syndromes. This type of ventilation, which was developed by Sjöstrand and colleagues in the late 1970s, was originally intended to minimize the cardiovascular effects of PPV.[113] It was discovered that HFPPV sometimes could also improve gas exchange while keeping airway pressures (P_{aw}) lower than they 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.[114] 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 pediatric patients.

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 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 is currently used mostly for investigational purposes.

High-Frequency Percussive Ventilation

Forrest M. Bird, a pioneer in mechanical ventilation technology, developed high-frequency percussive ventilation (HFPV). Bird's intent was to incorporate the most effective characteristics of jet and conventional ventilation into one device. HFPV 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 of intubated patients to help mobilize secretions. It can also be used as a continuous mode of ventilation. HFPV 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.[115]

The VDR-4 (Bird Space Technology, Sandpoint, Idaho) is a high-frequency percussive generator 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. 22.26) 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 high; this is the result of the large pressure gradient between the patient connection and 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. 22.27.

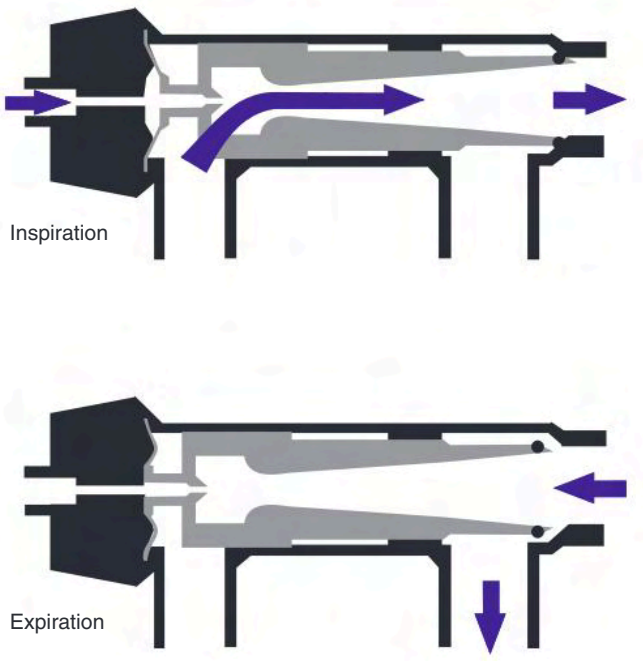

Fig. 22.26 Design of the sliding Venturi for the high-frequency percussive generator. (See text for additional information.)

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.[118] Another type of HFOV device has been implemented by Dräger (Babylog VN500) and combines HFOV with volume guarantee. The volume-targeted HFOV feature has been shown to result in better gas exchange HFOV without volume-targeted option in preterm newborns.[119] This device shows promise in its ability to measure and target V_T. However, it is currently unavailable in the United States.

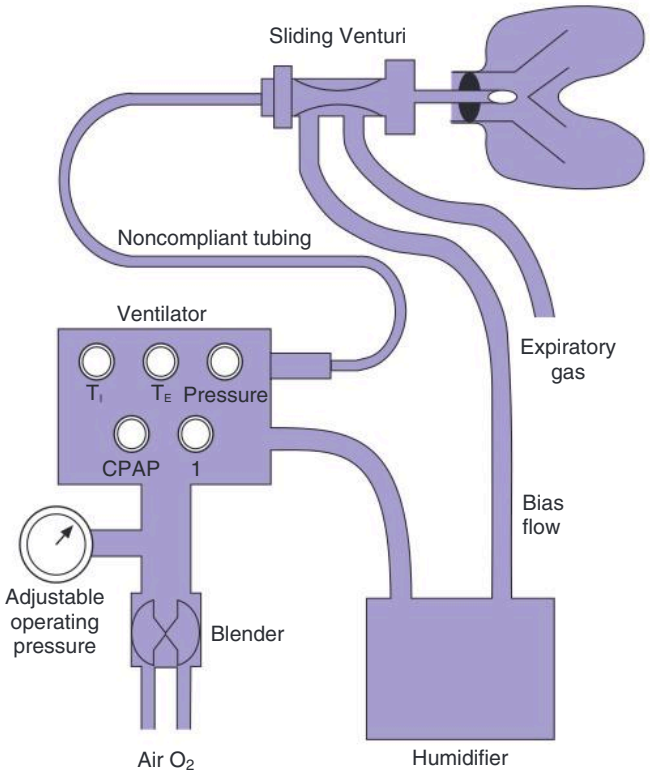

Fig. 22.27 Schematic of a high-frequency percussive ventilation system. *CPAP*, Continuous positive airway pressure. (Modified from Branson RD, Hess DR, Chatburn RI: *Respiratory care equipment*, Philadelphia, PA, 1995, JB Lippincott.)

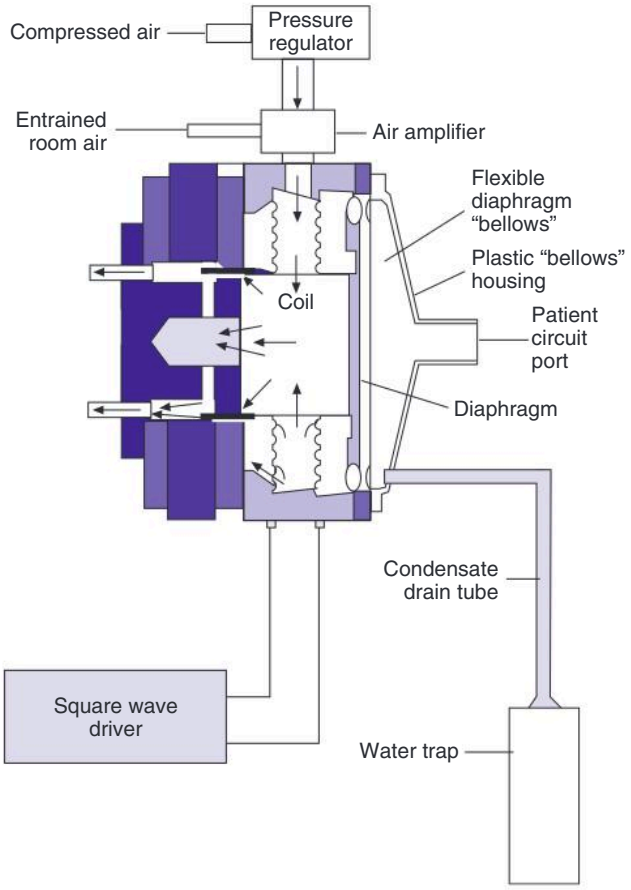

Fig. 22.28 Drive mechanism for the 3100A oscillator. A timer *(square wave driver)* signals the motor to drive the piston toward and away from the patient circuit port, making both inspiration and expiration active. Because the movement of the piston generates extreme heat, compressed air and entrained room air are introduced around the motor's coil to provide cooling. (Courtesy Vyaire Medical, Mettawa, IL.)

Oscillators and at least one pseudooscillator have been in use since the late 1980s. An example of a commonly used oscillator is the Sensor Medics 3100A (Vyaire Medical, Mettawa, IL). This oscillator uses a diaphragm-shaped piston that is driven magnetically, similar to the action of a stereo speaker (Fig. 22.28). 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. 22.29). 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 number of piston excursions determines the frequency. Two other mushroom valves function as safety releases on the circuit. See the Clinical Scenario involving HFOV.

Clinical Scenario: Pulmonary Interstitial Emphysema

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 HFOV. Which strategy would you use, and what initial P_{aw} and fraction of inspired O_2 (F_IO_2) would you choose?

A low-volume HFOV strategy is often used for patients with an air-leak syndrome (e.g., PIE). The P_{aw} used for the conventional ventilator should be set on the oscillator initially. The F_IO_2 should be set as high as 1.0 for the first 12 to 24 hours because the goal is to keep the arterial partial pressure of O_2 (P_aO_2) 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 22.7). (Chapter 23 presents information on HFOV for adults.)

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 V_T smaller than dead space. HFJV was originally used to provide short-term ventilatory support during adult upper airway surgery and instrumentation, but animal

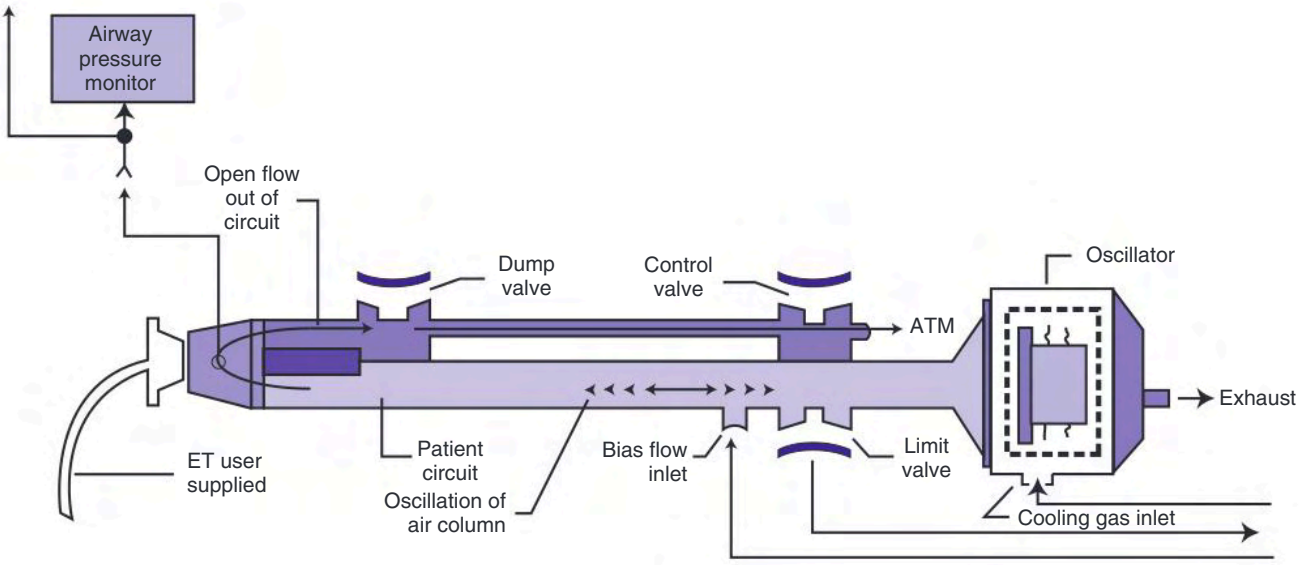

Fig. 22.29 Basic breathing circuit of the Model 3100A high-frequency oscillator. *ATM,* Atmosphere; *ET,* endotracheal tube. (Courtesy Vyaire Medical, Mettawa, IL.)

Case Study 22.7

Patient Case—Acute Respiratory Distress Syndrome (ARDS) Managed With High-Frequency Oscillation

A 5-year-old girl with a diagnosis of ARDS secondary to sepsis and aspiration pneumonia has been on the 3100A oscillator for about 4 hours. The mean airway pressure is set at 28 cm H_2O, the frequency at 8 Hz, and the amplitude at 38 cm H_2O. The F_IO_2 is 0.7. Initially the ABG values and vital signs improved. However, over the past 30 minutes, the heart rate has increased and the S_pO_2 has dropped from 97% to 87%. What action should the respiratory therapist consider?

studies showed 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-O_2 gas mixture under considerable pressure through an ET. The Bunnell Life Pulse can deliver frequencies in the range of 240 to 660 cycles/min. 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 P_aCO_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).

V_T 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 R_{aw} and C_L. For infants the typical delivered V_T is 1 to 3 mL. However, a V_T 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_T 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_T and P_{aw} than other forms of mechanical ventilation, a higher PEEP may be used without elevating P_{aw} to potentially harmful levels.[119]

In most patients the conventional ventilator is operated in the 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.[118] 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_T 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. 22.30). 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.[120-123]

Pendelluft, which is the exchange of gas between lung units with different time constants, is observed through photographic

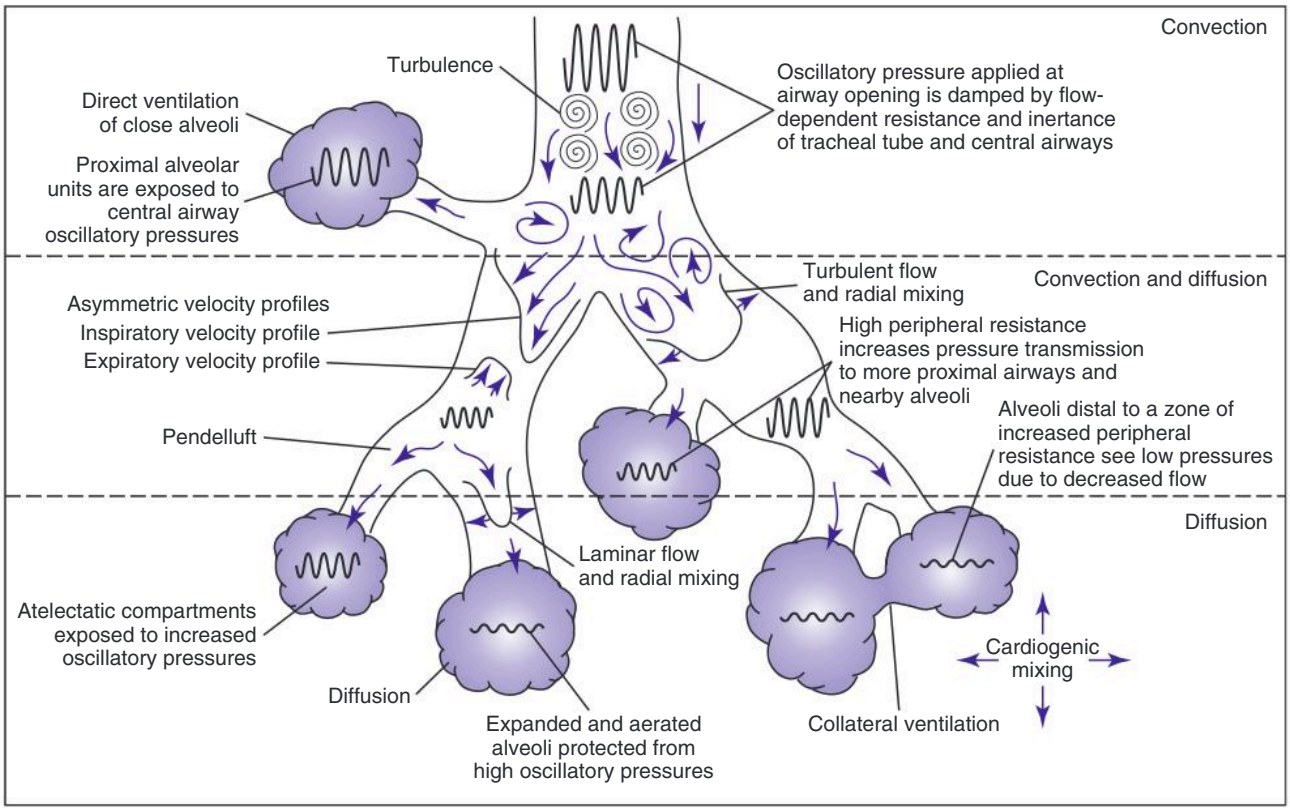

Fig. 22.30 Gas transport mechanisms and pressure damping during high-frequency oscillatory ventilation (HFOV). The major gas-transport mechanisms operating during HFOV in convection, convection-diffusion, and diffusion zones include turbulence, bulk, convection (direct ventilation of close alveoli), asymmetrical inspiratory and expiratory collateral ventilation between neighboring alveoli, and molecular diffusion (see text for details). (Redrawn from Pillow JJ: High frequency oscillatory ventilation: mechanisms of gas exchange and lung mechanics, *Crit Care Med* 33:S135-S141, 2005.)

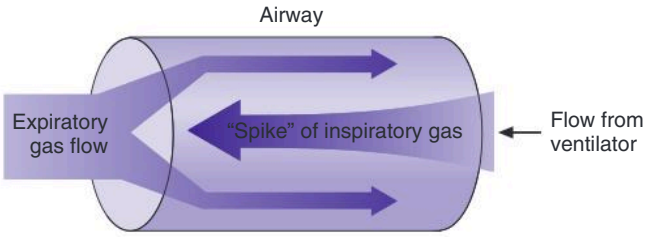

Fig. 22.31 Effect of streaming in high-frequency jet ventilation. Pulsations from the jet push the gas forward in the center; this causes gas along the airway walls to be pushed backward.

studies or by the measurement of different pressure values in the airways.[121-126] 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 these units may be recruited.

Streaming, or asymmetrical velocity profiles, is thought to occur because the velocity of gas flow is higher in the center of the airway (Fig. 22.31). 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.[127-129]

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.[125,126] 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* is likely 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 $\dot{V}_E$ produced is $\dot{V}_E = f \times V_T$; with HFOV, the formula is $\dot{V}_E = f \times V_T^2$. Although V_T 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. $\dot{V}/\dot{Q}$ 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 increases 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_T 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 providing manual ventilation to 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 put in place, and an electrocardiogram and BP are monitored continuously. Transcutaneous CO_2 monitors work well on most patients regardless of age. Monitoring of transcutaneous CO_2 is useful for noninvasively trending P_aCO_2 between blood gas draws. If a transcutaneous monitor is used, the sensor is placed on the patient and the baseline comparison to P_aCO_2 is made before HFV is initiated.

Cardiovascular assessment focuses on intravascular volume and cardiac output. A high, sustained P_{aw} 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, 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 F_IO_2 and P_{aw}. 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 neurological insult. However, in neonates in whom randomization occurred earlier (1–4 hours), HFOV showed a significant benefit for reducing death or BPD over conventional ventilation.[117] 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. 22.32). The goal of this strategy is to increase P_{aw} on the oscillator until oxygenation stabilizes. The P_aCO_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_IO_2 to 0.45 to 0.5 may be possible, depending on cardiovascular status. This is followed by weaning P_{aw} (Fig. 22.33).[130] Patients with air-leak syndromes (e.g., PIE, pneumothorax, 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 P_{aw}. In infants with PIE, this strategy can also feature a lower frequency than is typical to allow a longer expiratory time, thus encouraging interstitial gas resorption.

The initial P_{aw} is usually set at the same level or 2 to 3 cm H_2O higher than the P_{aw} 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_IO_2 as high as 1 is considered acceptable during the first 12 to 24 hours.[130] The F_IO_2 is usually reduced to 0.8 before weaning the P_{aw}. This is done to provide some margin in case oxygenation drops after the P_{aw} is lowered; the F_IO_2 can be increased to help restore oxygenation. The decision to wean from the P_{aw} rather than the F_IO_2 is made according to the progress seen in correcting air leaks. If they are resolving, the need to reduce P_{aw} is not as important as the need to reduce F_IO_2. Conversely, extension of air leaks may require a lower P_{aw}, if possible, with a higher F_IO_2 (see Case Study 22.7). 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

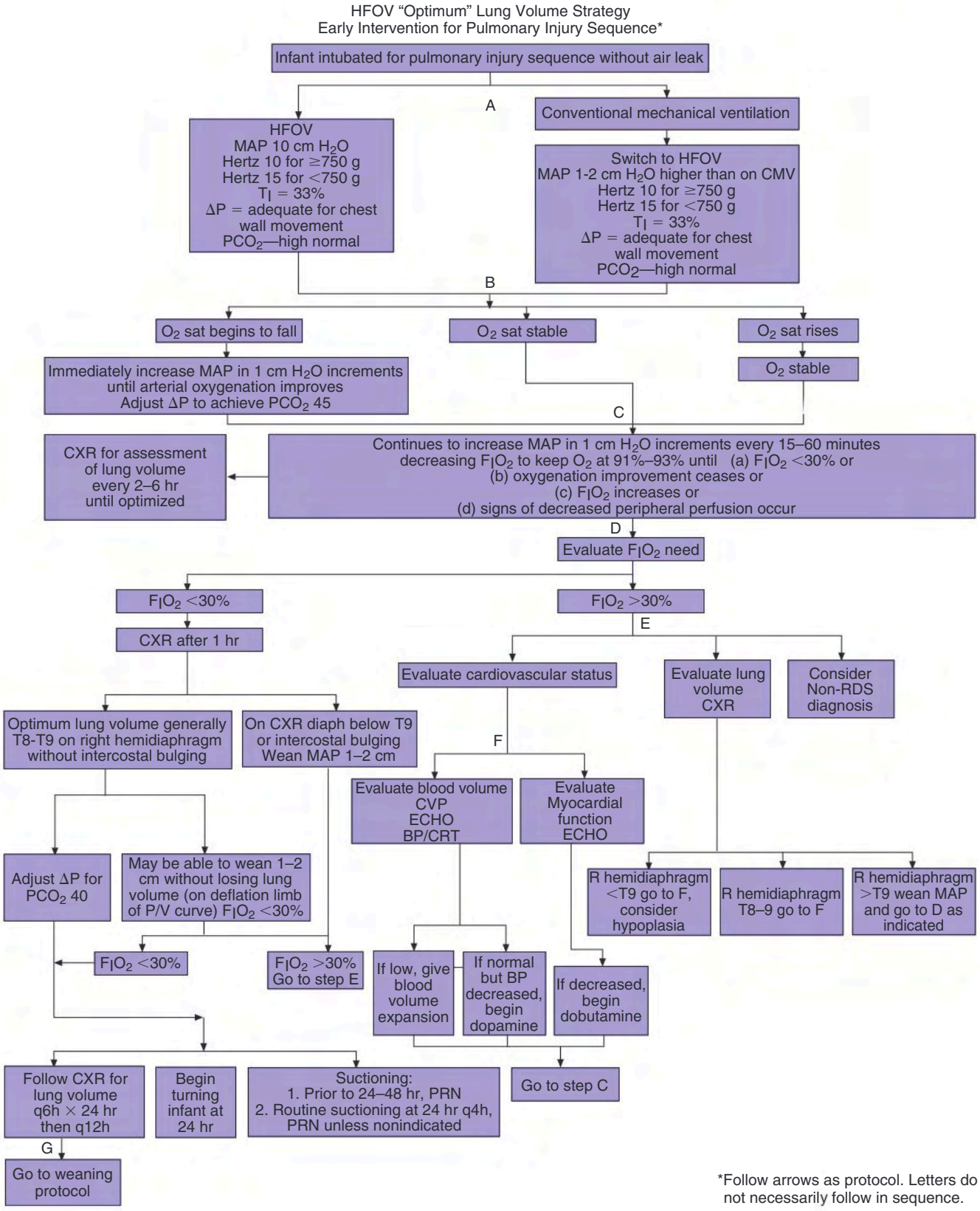

Fig. 22.32 Strategy flow chart for optimum lung volume high-frequency oscillatory ventilation (HFOV). *CRT,* Hematocrit; *CVP,* central venous pressure; *CXR,* chest radiograph; *ECHO,* echocardiogram; F_IO_2, fraction of inspired oxygen; *MAP,* mean airway pressure; ΔP, pressure gradient; PCO_2, partial pressure of carbon dioxide; *P/V,* pressure/volume; *qH,* every hour; *RDS,* respiratory distress syndrome; *T8 to T9,* thoracic vertebra. (From Minton S, Gerstmann D, Stoddard R: Cardiopulmonary review PN 770118—001. Yorba Linda, Calif. Mettawa, IL, 1995, Vyaire Medical.)

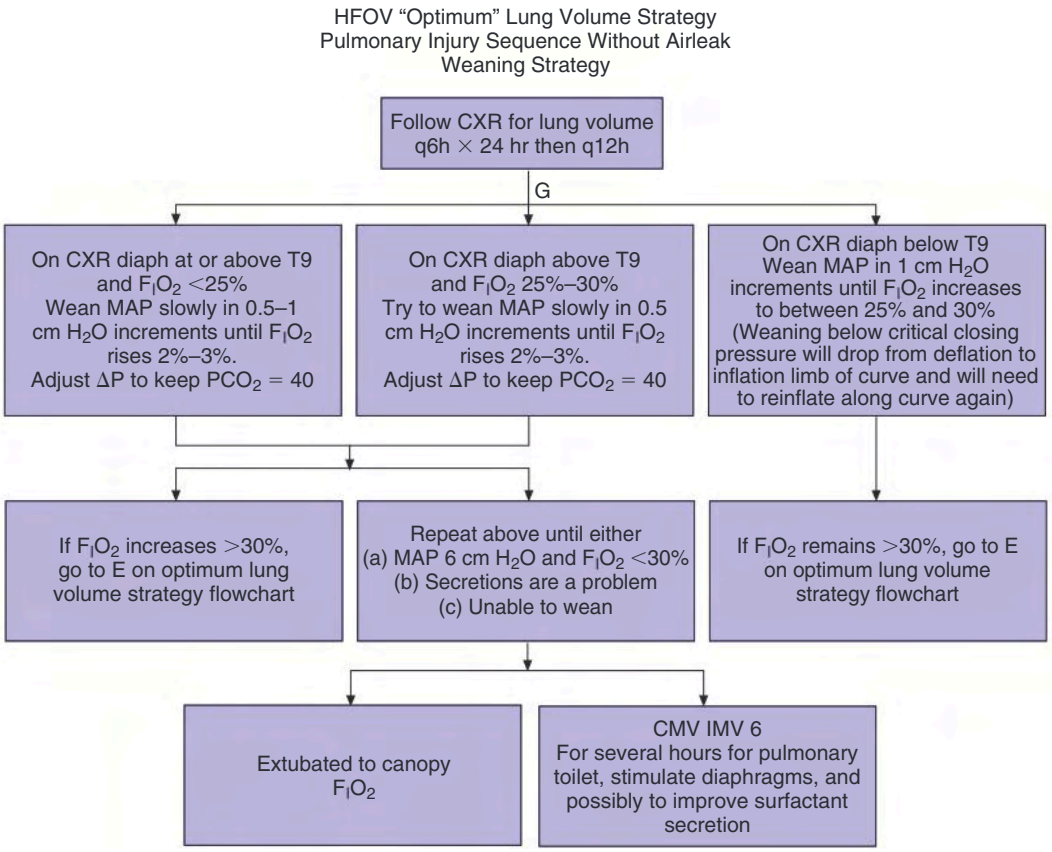

Fig. 22.33 Strategy flow chart for weaning from optimum lung volume high-frequency oscillatory ventilation. *CMV,* Continuous mandatory ventilation; *CXR,* chest radiograph; *F_IO_2,* fraction of inspired oxygen; *IMV,* intermittent mandatory ventilation; *MAP,* mean arterial pressure; *q6h,* every 6 hours; *q8h,* every 12 hours. (From Minton S, Gerstmann D, Stoddard R: Cardiopulmonary review PN 770118-001. Yorba Linda, Calif. Mettawa, IL, 1995, Vyaire Medical.)

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 pediatric patients. Compared with conventional ventilation, HFOV initiated earlier in the disease process may improve gas exchange and reduce VILI in pediatric patients with ARDS.[131]

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.[66] The time from start of weaning to extubation and the rate of extubation failure seemed to be unaffected by the use of written weaning protocols.[66] Routinely evaluating a patient for weaning readiness has been shown to be far more useful in facilitating timely ventilator discontinuation and extubation.[132] 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 (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 weaning readiness test (see Box 22.8) is usually conducted in patients whose sedation score would permit extubation. The patient's enteral feedings are stopped for the test, the F_IO_2 is reduced to 0.5, and PEEP is reduced to 5 cm H_2O. If the S_pO_2 is >95% on these settings or with a lower F_IO_2, the pressure support level is reduced to the minimum amount for the ET size (see Box 22.8). Once the patient has been placed on the minimum-pressure support level for the ET size, the respiratory rate and S_pO_2 are monitored. Increases in the respiratory rate above guideline parameters or a drop in S_pO_2 signals a failed test and suggests that additional support is needed.[66] Although the test is called an extubation readiness test, it is only one criterion in the decision on extubation. It is also 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 is often 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

BOX 22.8 Extubation Readiness Test[116]

Procedure

1. Temporarily stop enteral feedings.
2. Reduce the fractional inspired O_2 (F_IO_2) to 0.5.
3. Reduce the positive end-expiratory pressure (PEEP) to 5 cm H_2O.
4. Evaluate the O_2 saturation by pulse oximetry (S_pO_2):
   a. If the S_pO_2 is below 95% and the F_IO_2 is less than 0.5, increase the F_IO_2 to 0.5.
   b. If the S_pO_2 is above 95%, change to pressure support ventilation (PSV) at the minimal amount for the endotracheal (ET) tube size used:
      - 3 to 3.5 mm: 10 cm H_2O
      - 4 to 4.5 mm: 8 cm H_2O
      - 5 mm or larger: 6 cm H_2O
   c. Monitor the S_pO_2, effective tidal volume (V_T), and respiratory rate *(f)*.

Assessment

The patient is *potentially* ready for extubation if:

- The S_pO_2 is >95%.
- The effective V_T is >5 mL/kg.
- The respiratory rate is within the goal range for the patient's age (see chart) for up to 2 hours:

Age	Goal Range (breaths/min)
Younger than 6 mo	20–60
6 mo–2 y	15–45
2–5 y	15–40
Older than 5 y	10–35

number of patients who had an uneventful ventilator course and planned extubation nevertheless had a failed extubation.[63,127]

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 (see Chapter 20). A small number of studies support the use of dexamethasone as a prophylactic treatment to prevent 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.[133,134] This protocol is sometimes 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 alveolar gas. About 90% of human surfactant is made up of phospholipids, about 60% of which is di-palmitoyl-phosphatidylcholine (DPPC). DPPC, other phospholipids, and neutral lipids produce the surface-active effects in the lungs. The distribution of surfactant is thought to be caused by 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.[135,136]

Two strategies are suggested for surfactant replacement therapy: prophylactic therapy and rescue therapy. **Prophylactic therapy** consists of surfactant administration immediately at birth or soon after for infants who are at risk for developing RDS. **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_IO_2 of 1.0. Each partial dose is usually 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_T and waveforms are noted and settings adjusted appropriately. If the patient is placed on an HFV, the F_IO_2 and P_{aw}, S_pO_2, and ABG values are evaluated (see previous section on HFV). 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, O_2 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 the pharynx because of a leak around the ET. Even without tube obstructions, hypoxemia may worsen.[135] Some patients develop prolonged periods of apnea after surfactant dosing.[136]

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.[137] 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.[135]

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_{aw} delivered by the ventilator.[134] This situation underscores the importance of monitoring V_T and using VSV after the immediate postdosing period. Monitoring for changes in the shunt is crucial after surfactant replacement in patients with patent ductus arteriosus (PDA), particularly newborns with BPD.[137] Theoretically the reduced pulmonary vascular resistance produced by improved oxygenation can increase the left-to-right shunting caused by the PDA.[138-140] 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.[141] The studies showed an initial improvement in the P_aO_2/F_IO_2 ratio in most subjects, but sustained improvement beyond 48 hours of treatment has not been achieved. More recent experiments with a novel breath-synchronized nebulizer for surfactant aerosol delivery have shown improved drug delivery and better response in gas exchange than liquid instillation for RDS[142] and ARDS.[143]

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 $\dot{V}/\dot{Q}$ matching and reduce physiological shunt (see Chapter 13). 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.[144]

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 INOMax DS_{IR} (Ikaria, Clinton, NJ), a pneumotachometer incorporated into an injector module is placed in line 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_2) and the F_IO_2.[145] 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.[146] Pulmonary tissue toxicity is a well-known side effect and results when NO combines with O_2 and forms the reddish-brown gas NO_2. When NO_2 is exposed to NO, dinitrogen trioxide (N_2O_3) is produced and reacts with water, forming either nitrous or nitric acid, both of which are toxic to the alveolar epithelium.[147] The higher the concentration of O_2, the greater is the potential for the development of toxic levels of NO_2.

Many clinical situations that require administration of INO also require high concentrations of O_2 in the gas mixture. In such cases, even low-dose NO can produce toxic levels of NO_2,[148] thus underscoring the importance of NO/NO_2 monitoring in the inhalation circuit. However, when NO is administered at low doses, it usually reacts slowly with O_2, and the formation of toxic products is small. Nonetheless, close monitoring is essential to control the therapeutic level of NO and avoid excessive levels of NO_2.

Methemoglobinemia develops primarily through the oxidation of NO when it comes in contact with oxyhemoglobin. Methemoglobin occurs naturally, and its level is normally 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 <2%.

Studies of INO's effectiveness at reducing intrapulmonary shunt and improving $\dot{V}/\dot{Q}$ matching suggest that the drug is most effective when used with HFV.[149,150] 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 22.8).[151] (See the Evolve website for this text and for additional information on NO therapy.)

Case Study 22.8

Determining Appropriateness of Nitric Oxide Therapy

A 33-hour-old infant with respiratory distress has just been transferred to the newborn intensive care unit (ICU). Mild cyanosis is developing, and the S_pO_2 percentage is in the low 60s on supplemental O_2. 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 is eventually intubated, sedated, and paralyzed. An umbilical artery catheter is placed, and administration of dopamine and fluids is initiated. Arterial blood gas values show refractory hypoxemia, a low P_aCO_2, and metabolic acidosis. The patient is placed on mechanical ventilation with 100% O_2.

The ICU team is leaning toward a diagnosis of persistent pulmonary hypertension 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?

SUMMARY

- Mechanical ventilation in 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 amount of energy required to breathe.
- Neonatal and pediatric patients have smaller lungs, higher R_{aw}, lower C_L, 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 retrosternal retractions.
- The Silverman-Andersen 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 S_pO_2 and transcutaneous CO_2 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 $\dot{V}/\dot{Q}$ mismatching, decreased pulmonary blood flow, increased pulmonary vascular resistance, and decreased cardiac output.
- NIV, also known as *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 and 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 and reduce WOB with acute hypoxemia, neuromuscular disorders, and conditions that cause abdominal distention. It is also used to relieve the airway obstruction associated with obstructive sleep apnea or airway lesions such as laryngotracheal malacia.
- 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, 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-controlled ventilation is the most widely used mode of ventilation in neonates and pediatric patients. The pressure-controlled breath can be triggered by pressure or flow and is terminated on the basis of time. It can be used during IMV and CMV breath types.
- Monitoring inspiratory pressure, PEEP, T_I, T_E, I/E ratio, V_T, frequency, mean airway pressure, and inspired O_2 concentration are all necessary with pressure-controlled ventilation.
- Volume-controlled ventilation has been used in older children and adults with ARDS and neonates and premature neonates with RDS.
- The most widely used form of dual-control mode used in neonates and pediatric patients is PRVC, and it is commonly used in patients with CMV or IMV breath types.
- VSV is well suited to infants and pediatric patients as target V_T 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, and (4) alleviate pulmonary inflammation (biotrauma).
- HFV can be complicated by pulmonary injury sequence, which includes RDS, PIE, pulmonary air-leak syndrome, O_2 toxicity, and development of BPD.
- 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.
- HFOV has become the most widely used high-frequency technique for infants and pediatric patients because both inspiration and expiration are active, gas flow is sinusoidal rather than triangular, bulk flow rather than jet pulsations is delivered, and V_T 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 NO_2 therapy.
- Prophylactic therapy consists of surfactant administration immediately at birth or soon after for infants who are at risk for 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 $\dot{V}/\dot{Q}$ 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 S_pO_2 is 90% on room air. ABG values reveal a pH of 7.34, a P_aCO_2 of 28 mm Hg, and a P_aO_2 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 nasopharyngeal (NP) tube for 2 days. The NP tube is connected to a ventilator set to deliver CPAP at 6 cm H_2O with a flow rate of 8 L/min. Over the past 2 hours, the infant's breathing frequency increased from about 40 breaths/min to about 60 breaths/min. F_IO_2 had to be increased from 0.25 to 0.45 because of decreasing S_pO_2 values. Which of the following actions should be taken?
A. Increase the flow rate
B. Increase the CPAP level
C. Change the NP tube
D. Intubate the infant and begin TCPL

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, heart rate is 190 beats/min, temperature is 38.8° C, blood pressure is 70/44 mm Hg, and S_pO_2 is 83% on a nonrebreathing O_2 mask. Breath sounds are distant, but coarse rales can be heard bilaterally. ABG values reveal a pH of 7.26, P_aCO_2 at 64 mm Hg, and P_aO_2 at 55 mm Hg on the nonrebreathing mask. Which of the following interventions would be appropriate based on this information?
A. Intubate the patient and begin CPAP
B. Place the patient on a BiPAP system with supplemental O_2
C. Maintain the patient on the nonrebreathing 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 T_I are best determined by:
A. Placing the infant on the ventilator and adjusting PIP and T_I to obtain the desired V_T
B. Manually ventilating the infant while noting the PIP and T_I that achieve the best S_pO_2 and lung aeration
C. Placing the infant on the ventilator and adjusting PIP and T_I to obtain the desired S_pO_2
D. Manually ventilating the infant while monitoring the waveform, noting the PIP and T_I that produce the best waveform

9. A 3.5-kg newborn with a diagnosis of group B streptococcal pneumonia is intubated with a 3-mm internal diameter 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 H_2O, PEEP = 4 cm H_2O, F_IO_2 = 1.0, set frequency = 20 breaths/min
Actual frequency = 50 to 55 breaths/min
V_{Tinsp} = 45 to 50 mL, V_{Texh} = 12 to 15 mL, set T_I = 0.6 second, actual T_I = 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 T_I

10. 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 H_2O, PEEP = 6 cm H_2O, frequency = 22 breaths/min, T_I = 0.6 second
F_IO_2 = 0.3, flow rate = 8 L/min
ABG values are pH = 7.26, P_aCO_2 = 66 mm Hg, P_aO_2 = 78 mm Hg

Additional data are as follows:
V_{Texh} = 7 to 10 mL, $\dot{V}_E$ = 1.88 L/min
S_pO_2 = 95%, BP = 68/42 mm Hg

On the basis of these data, which of the following ventilator control manipulations would be most appropriate?
A. Increase the T_I
B. Increase the flow
C. Increase the frequency
D. Increase the PIP

11. A 2-year-old patient intubated with a 4-mm internal diameter nasal ET is recovering from surgical repair of a ventricular septal defect and has been weaned from volume ventilation on IMV to PSV (Servo-i ventilator). Since the changeover to this mode, the ventilator at times seems to trigger on and cycle off rapidly, making the patient uncomfortable and 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

12. A newborn patient of 29 weeks' gestational age has RDS. She weighs 950 g. She is receiving conventional mechanical ventilation at a P_{aw} of 16 cm H_2O. The patient is to be changed to HFOV. Which of the following settings would you initially select?
A. P_{aw} = 18 cm H_2O; frequency = 15 Hz
B. P_{aw} = 16 cm H_2O; frequency = 15 Hz
C. P_{aw} = 18 cm H_2O; frequency = 10 Hz
D. P_{aw} = 16 cm H_2O; frequency = 10 Hz

13. An 18-month-old, 15-kg child with a diagnosis of ARDS has been mechanically ventilated for 5 days. The patient initially received VC-IMV but now is receiving PCV at the following settings:
PIP = 37 cm H_2O, PEEP = 8 cm H_2O, P_{aw} = 16.4 cm H_2O
Frequency = 40 breaths/min, T_I = 0.9 second, F_IO_2 = 1
ABG values are: pH = 7.29, P_aCO_2 = 53 mm Hg, P_aO_2 = 46 mm Hg, S_aO_2 = 79%

Additional data are as follows:
V_{Texh} = 75 to 85 mL
$\dot{V}_E$ = 2.92 L/min

On the basis of these data, which of the following would be most appropriate?
A. Increase the PEEP, maintain the PIP, and give sodium bicarbonate ($NaHCO_3$) to normalize the pH
B. Change to high-frequency ventilation
C. Maintain the present settings but give $NaHCO_3$ to normalize the pH
D. Change to a high V_T/low *f* strategy

14. 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 F_IO_2 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 F_IO_2 as soon as possible
B. Reduce the P_{aw} even if the F_IO_2 must be increased
C. Increase the P_{aw} and wean the F_IO_2 as much as possible
D. Switch to conventional ventilation

15. A 640-g newborn is receiving HFOV at the following settings:
P_{aw} = 19 cm H_2O, F_IO_2 = 0.28, frequency = 15 Hz, amplitude (P) = 34 cm H_2O
ABG values are pH = 7.56, P_aCO_2 = 23 mm Hg, P_aO_2 = 85 mm Hg

On the basis of these data, which of the following would be most appropriate?
Maintain the current settings
Reduce the amplitude
Reduce the frequency
Reduce the P_{aw}

16. A full-term, 3-kg infant is on HFJV at the following settings:
PIP = 22 cm H_2O, PEEP = 11 cm H_2O, P_{aw} = 12 cm H_2O
Frequency = 420 cycles/min, jet T_I = 0.02 second, F_IO_2 = 0.4
ABG values are: pH = 7.3, P_aCO_2 = 55 mm Hg, P_aO_2 = 90 mm Hg

On the basis of these data, which of the following control changes should be made first?
Increase the jet T_I
Maintain the current settings
Reduce the frequency
Reduce the PEEP

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

18. 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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