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CHAPTER 23

Special Techniques Used in Ventilatory Support

OUTLINE

KEY TERMS

- Amplitude
- Electrical activity of the diaphragm (Edi)
- Heliox
- Neurally adjusted ventilatory assist (NAVA)

LEARNING OBJECTIVES

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

1. Discuss the benefits and disadvantages of airway pressure release ventilation (APRV)
2. Recommend initial settings for initiating APRV in patients with acute respiratory distress syndrome (ARDS).
3. Explain how the controls operate with the Vyaire 3100B High-Frequency Oscillatory Ventilator.
4. Recommend initial ventilator settings for an adult with the 3100B unit.
5. List types of medications that may be used in transitioning from volume-controlled continuous mandatory ventilation (VC-CMV) to high-frequency oscillatory ventilation (HFOV) for an adult.
6. Explain how the chest wiggle factor is influenced by HFOV settings.
7. Name pulmonary pathological conditions in which heliox therapy may be beneficial.
8. Compare the differences among set tidal volume (V_T), monitored V_T, and actual V_T delivery ($V_T$del) during heliox therapy.
9. Describe how heliox used with a mechanical ventilator may affect pressures and fractional inspired oxygen concentration (F_IO_2) monitoring and delivery.
10. Explain the procedure for using heliox cylinders with a mechanical ventilator.
11. Name at least four techniques that can help determine the correct placement of the esophageal Edi catheter.

12. Provide examples of how the Edi waveform can be of value in monitoring critically ill patients.
13. Discuss various factors that can cause a low Edi signal and a high Edi signal.
14. Describe the safety backup features and alarms available with neurally adjusted ventilator assist (NAVA).
15. Calculate an estimated pressure delivery when given the NAVA level, Edi peak, Edi minimum, and positive end-expiratory pressure (PEEP).
16. Explain what parameters (pressure, flow, volume, neural signal, time) are used to deliver a breath during NAVA ventilation.
17. Identify clinical situations in which NAVA should be used.

The ultimate goal of mechanical ventilation is to sustain life and do no harm. In an effort to achieve these outcomes, clinicians often search for alternative methods of treatment when standard procedures fail. Some of these techniques prove to be viable alternatives to standard practices and gain a foothold in clinical practice.

The inclusion of all recently explored and newly discovered techniques that can be used to ventilate and manage critically ill patients is beyond the scope of this text. We therefore present a discussion of four techniques that have received increased attention by critical care clinicians: airway pressure release ventilation, high-frequency oscillatory ventilation (HFOV) in the adult, heliox therapy, and monitoring the diaphragm's electrical activity and using that activity with the ventilator mode **neurally adjusted ventilatory assist (NAVA)**.

AIRWAY PRESSURE RELEASE VENTILATION

Airway pressure release ventilation (APRV) is a mode of ventilatory support designed to provide two levels of continuous positive airway pressure (CPAP) and allow spontaneous breathing at both levels when spontaneous effort is present.[1-6] APRV provides a moderately high level of pressure (P_{high}, 15–30 cm H_2O), which can be considered a baseline pressure. P_{high} is occasionally interrupted with a brief time at lower pressure (P_{low}, 0–15 cm H_2O). The brief interval at P_{low} is called release time (Fig. 23.1).

Both the high- and low-pressure levels are time triggered and time cycled when spontaneous efforts are not detected.[7] With current-generation ventilators, spontaneous breathing can be accompanied by patient triggering. During APRV, if the patient is not spontaneously breathing, the pressure curve looks like pressure-controlled inverse ratio ventilation (Fig. 23.2).[8] When the patient is spontaneously breathing, the patient's breathing activity can be monitored on the ventilator's display, particularly in the flow-time and volume-time scalars during ventilation (see Fig. 23.3).[9]

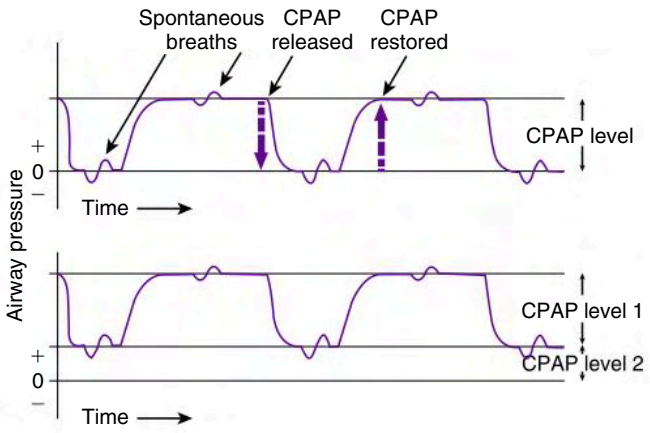

Fig. 23.1 *Top curve,* Pressure-time waveforms for airway pressure release ventilation (APRV) with a continuous positive airway pressure (CPAP) level 2 (P_{low}) of 0 cm H_2O. *Bottom curve,* Pressure-time waveform for APRV with a CPAP level 2 (P_{low}) set greater than 0 cm H_2O. Spontaneous efforts by the patient do not cycle the ventilator between low and high CPAP or high and low CPAP. A fixed time interval is set for the CPAP level 1 time and the CPAP level 2 times. (From Dupuis Y: *Ventilators: theory and clinical application,* ed 2, St Louis, MO, 1992, Mosby.)

OTHER NAMES

In Europe, APRV is referred to as bilevel airway pressure (BiPAP).[7] It has also been called variable positive airway pressure, intermittent CPAP, CPAP with release, pressure-controlled inverse ratio ventilation with spontaneous ventilation, upside-down intermittent mandatory ventilation, and biphasic CPAP.[7,10,11] In the United States, BiPAP is often used to describe a technique in which inspiratory positive airway pressure (IPAP) and expiratory positive airway pressure (EPAP) levels are set for noninvasive positive pressure ventilation (NIV), where the inspiratory/expiratory (I/E) ratio is normal.[7] (See Chapter 19 for more information on BiPAP with NIV.)

The Dräger Evita was the first ventilator in the United States to provide APRV. Subsequently other intensive care unit (ICU) ventilators, such as the Hamilton G-5, Puritan Bennett 840, Dräger Evita V500, CareFusion AVEA, and Getinge Servo-u APRV. Interestingly,

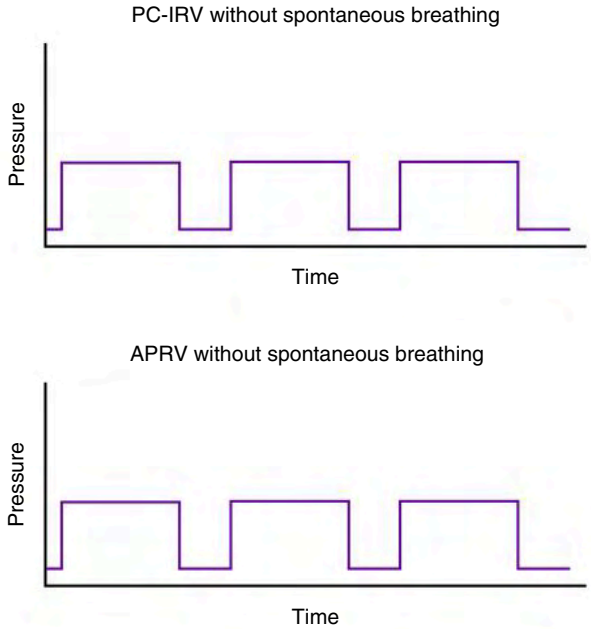

Fig. 23.2 Pressure-time curves for pressure control inverse ratio ventilation *(PC-IRV)* in an apneic patient *(top)* and airway pressure release ventilation *(APRV)* for an apneic patient *(bottom)*. The waveforms appear the same.

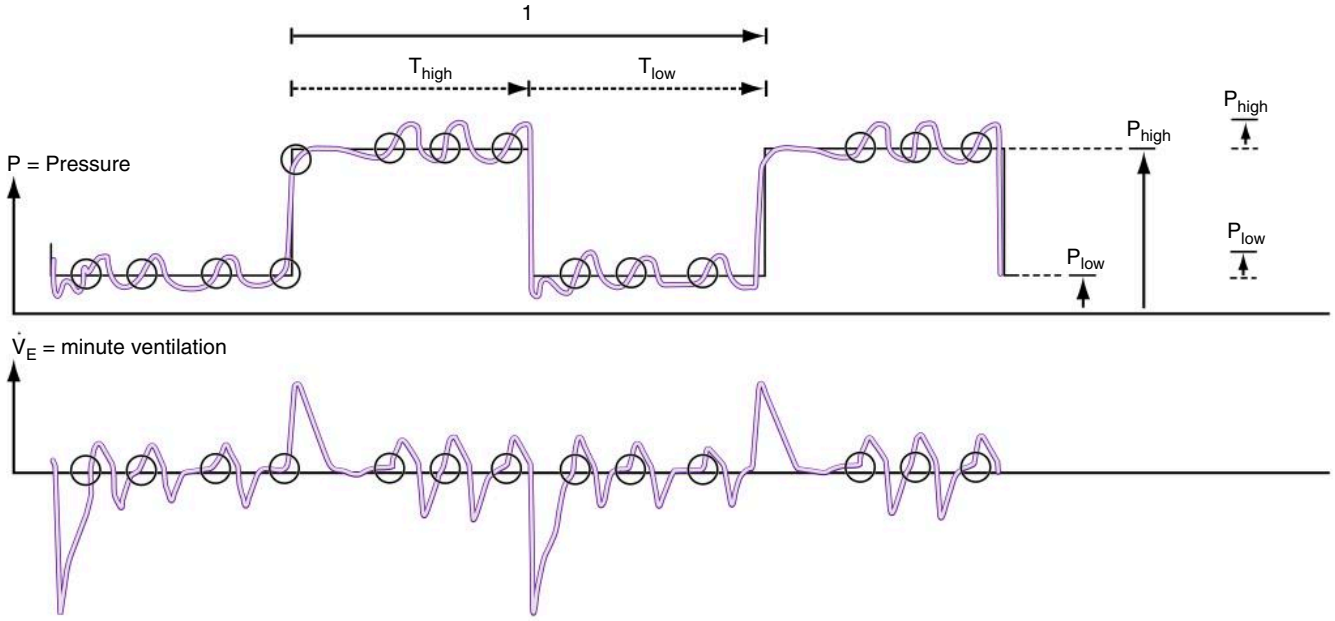

Fig. 23.3 *Top,* Pressure-time curve; *bottom,* flow-time curve during APRV, called Bi-Vent on the Servo-i ventilator. Inspiratory flows are in the upward direction and expiratory flows downward. Note the spontaneous breathing during the high pressure (P_{high}) setting and the low pressure (P_{low}) setting. (Courtesy Maquet, Inc., Bridgewater, N.J. Redrawn for this text.)

each of these manufacturers uses a different terminology for describing the APRV mode. For example, the Servo-i and Servo-u refers to APRV as Bivent; the Puritan Bennett 840/980 uses the term Bilevel; and the Hamilton G5 refers to APRV as Duo-PAP. The function of APRV may also be different with each ventilator. For example, the Puritan Bennett 840/980 Bilevel mode allows the setting of one pressure support level during both P_{high} and P_{low}. On the other hand, the Servo-i and Servo-u allows two levels of pressure support ventilation (PSV) to be set independently, one for P_{high} and a separate level for P_{low} (Key Point 23.1).[7,12]

***Key Point* 23.1** Because the function of ventilators is frequently changed with the addition of upgraded software versions, clinicians must become familiar with these upgrades on the ventilators they plan to use with patients.

ADVANTAGES OF AIRWAY PRESSURE RELEASE COMPARED WITH CONVENTIONAL VENTILATION

The original concept and the equipment for the application of APRV were first described by Stock and colleagues in 1987.[6,13] Subsequent investigations confirmed that APRV produced lower peak pressures, better oxygenation, less circulatory interference, and better gas exchange without compromising the patient's hemodynamic status compared with conventional ventilation for the treatment of acute respiratory distress syndrome (ARDS).[3,4,6,12,14-16] APRV requires a lower minute ventilation than volume-controlled continuous mandatory ventilation (VC-CMV), suggesting a reduction in physiological dead space.[9] It also provides better ventilation/perfusion $\dot{V}/\dot{Q}$ matching compared with PSV.[17] APRV improves gas exchange and lowers peak inspiratory pressure (PIP) in patients with ARDS compared with patients receiving volume-controlled inverse ratio ventilation (VC-IRV).[18] Compared with pressure-controlled inverse ratio ventilation (PC-IRV), APRV reduces peak and mean airway pressures, increases cardiac index, decreases central venous pressure, increases urine output, increases oxygen (O_2) delivery, and reduces the need for sedation and paralysis.[16,19] APRV also improves renal perfusion and function when spontaneous breathing is maintained.[20]

Because APRV can reduce airway pressure in patients with ARDS, it is also thought to be associated with a reduced risk for ventilator-induced lung injury (VILI). (Maximum alveolar pressure should be kept below 30 cm H_2O to protect the lung. See Chapter 17 for additional information on VILI.) APRV may recruit consolidated lung areas over time and prevent repeated opening and closing of alveoli.[9,12,21,22]

Patients receiving APRV have been shown to require less sedation and analgesia compared with patients receiving continuous mandatory ventilation. Thus APRV appears to reduce anxiety and pain and improve patient comfort.[23,24]

Preserving Spontaneous Ventilation

Many of the advantages of APRV are likely attributable to the preservation of spontaneous breathing.[1,12,24] For example, spontaneous breathing preserves the cyclic decrease in pleural pressure, which augments venous return, thereby improving cardiac performance.[17,25] Moreover, because the patient can breathe spontaneously, the need for sedation may be reduced.[19,23,24]

Normally the mechanical delivery of air tends to ventilate areas receiving the least amount of perfusion. During a positive pressure breath in a paralyzed patient, the diaphragm is displaced toward the abdomen, with the *nondependent* regions of the lung receiving the most ventilation. At the same time, dependent areas receive the greatest perfusion.[26,27] Maintaining spontaneous ventilation tends to improve $\dot{V}/\dot{Q}$ matching by preferentially providing ventilation to dependent lung regions that receive the greatest blood flow.[23,27]

During positive pressure ventilation, atelectasis can occur near the diaphragm when activity of this muscle is absent. However, if spontaneous breathing is preserved, the formation of atelectasis is offset by the activity of the diaphragm.[28] In addition, maintaining spontaneous breathing may prevent atrophy of the diaphragm associated with the use of mechanical ventilation and paralytic agents.[28] Fig. 23.4 illustrates spontaneous ventilation during APRV.

APRV and Airway Pressures During Spontaneous Breathing

In older-generation ventilators, the expiratory valve closed completely during inspiration and was therefore not interactive with the patient. Current ventilators have more active expiratory values, which tend to "float open" when inspiratory pressures slightly exceed set pressures during pressure-targeted ventilation.

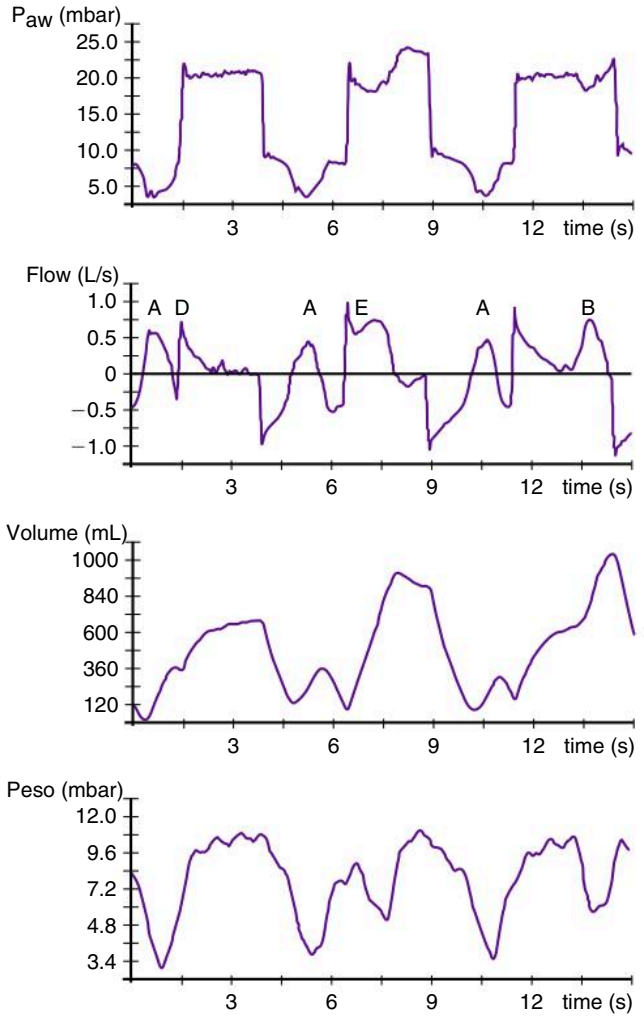

Fig. 23.4 Airway pressure release ventilation with curves for P_{aw}, flow, volume, and esophageal pressure (P_{eso}). P_{high} and P_{low} are both set at 2.5 seconds. Spontaneous breathing occurs throughout both phases. On the flow-time curve: *A,* spontaneous breaths at P_{low}; *B,* spontaneous breaths at P_{high}; *D,* a time-trigger transition from P_{low} to P_{high}; *E,* a simultaneous mandatory and spontaneous inspiration from P_{low} to P_{high}. (Redrawn from Neumann P, Golisch W, Strohmeyer A, et al.: Influence of different release times on spontaneous breathing pattern during airway pressure release ventilation, *Intensive Care Med* 28:1742–1749, 2002.)

As a result, current ventilators accommodate spontaneous breathing during APRV without pressure buildups. Because PIP during APRV does not significantly exceed the P_{high} level, complications associated with high pressures, such as overdistention of the lungs and reduced cardiac output, may be minimized (the high-pressure limit must still be set at a safe level).

DISADVANTAGES

Because APRV is a pressure-targeted mode of ventilation, volume delivery depends on lung compliance (C_L), airway resistance (R_{aw}), and the patient's spontaneous effort. The patient's volumes and gas exchange should be closely monitored. APRV does not completely support carbon dioxide (CO_2) elimination; rather, it relies on spontaneous breathing to ensure adequate CO_2 elimination.[1,7] With increased R_{aw} (increased time constants) such as occurs in patients with chronic obstructive pulmonary disease (COPD), the ability to eliminate CO_2 may be reduced because of limited emptying of the lung during the short release periods.

Another problem is that not all ventilators that offer an APRV mode allow for patient triggering of breaths in the phase from P_{high} to P_{low} and P_{low} to P_{high}.[29-31] Consequently some patient discomfort and increased work of breathing (WOB) may occur during these interval changes.

Limited staff experience with this mode may make its implementation difficult in some cases. Adequate training time and offsite support services and backup from manufacturers are essential. The limited amount of clinical research data available for this method of ventilation also makes determining evidence-based application problematic.

INITIAL SETTINGS[21,32,33]

When initiating APRV, the practitioner sets high and low pressure levels and high and low time levels. Using the P_{high} control sets the upper level of CPAP. The lower pressure, or release pressure, is set with a P_{low} control. A timing control commonly called T_{high} is used to set the duration of P_{high}. The P_{low} time period is set with a timer for the low time period (i.e., T_{low} control).

The total cycle time (TCT) is based on the T_{high} and T_{low} settings. For example, if T_{high} is 5.0 seconds and T_{low} is set at 0.5 seconds, the TCT will be T_{high} plus T_{low}, or 5.5 seconds. The I/E ratio will be 5:0.5, or 10:1. The number of cycles per minute will be equal to 60 seconds divided by the TCT. In this example, 60 s/5.5 s = 11 cycles/min.

Tidal volume (V_T) and flow delivery depend on the same factors that affect V_T and flow delivery in pressure-targeted ventilation: lung characteristics, patient effort, and pressure gradient ($\Delta P = P_{high} - P_{low}$). The higher this gradient, the more rapid the expiratory flow during release time and the higher the V_T. Minute volume ($\dot{V}_E$) depends on the patient's spontaneous effort during all time intervals plus the release interval volume change. Some clinicians suggest a target minute ventilation of 2 to 3 L/min less than the $\dot{V}_E$ on conventional ventilation.[21]

Setting High Pressure

When changing a patient from a conventional mode of ventilation to APRV, the settings for the conventional ventilator mode can

serve as a guide to the APRV settings. For example, if the patient's plateau pressure (P_{plat}) is 25 cm H_2O during conventional ventilation, an initial setting of 25 cm H_2O for P_{high} is appropriate. Some clinicians use the mean airway pressure (P_{aw}) measured during conventional ventilation for the P_{high} setting. A setting of 15 to 30 cm H_2O is typically used. P_{high} generally establishes a P_{aw} intended to maintain oxygenation by restoring functional residual capacity (FRC). Overdistention of the lung must be avoided, so a P_{high} of 35 cm H_2O is probably the maximum accepted level.[12,21,34] Several clinical studies have noted the use of P_{high} up to 45 cm H_2O, but this level may be damaging to the lungs unless the chest wall compliance is reduced from abdominal distention or prone positioning.[9,21]

The use of P_{high} may help in the recruitment of collapsed alveoli and maintenance of these recruited units.[18] It can take as long as 8 hours or longer for recruitment to occur. This is reflected in a progressive improvement in oxygenation and C_L.[18]

Setting Low Pressure

Some practitioners recommend initially setting P_{low} at 0 cm H_2O. A P_{low} of 0 cm H_2O allows an unimpeded expiratory gas flow and a rapid drop in pressure.[21,32,33]

Setting High Time

T_{high} is set at a minimum of 4.0 seconds.[21,32] Values less than 4.0 seconds can affect the P_{aw} in a negative way, losing the benefits of the APRV mode. The goal is to create a nearly continuous positive pressure that recruits collapsed alveoli, maintains recruitment, and optimizes oxygenation and compliance.[21,32] T_{high} can then be progressively increased to 12 to 15 seconds in 0.5- to 2.0-second intervals until the oxygenation target is achieved.[21,32] Slow progression is better than the increase of the I/E ratio in large increments, such as 2:1 or 3:1.[5,8,21] For example, if T_{high} is 5 seconds and T_{low} is 0.5 seconds (a 10:1 ratio), and T_{high} is changed to 5.5 seconds, the ratio change is small (11:1).

Setting Low Time

During the release time, or T_{low}, the patient exhales a volume of gas. This allows ventilation and removal of CO_2 from the body. As soon as the release time is complete, the higher level of CPAP is restored. The optimal duration of the release time and the setting of P_{low} are functions of the time constant of the respiratory system.[5] V_T depends on the C_L, R_{aw}, duration of T_{low}, and amount of pressure drop.[1] Complete exhalation of V_T would occur in approximately four time constants, but the actual setting of T_{low} has conflicting views.

During early trials with APRV, T_{low} settings of 1 to 1.5 seconds were suggested.[13,32] It was found that in patients with acute lung injury, after 1 second, complete emptying of the lung occurred, and after 2 seconds, airway pressures remained stable with no further pressure leaving the lungs. For example, Davis and colleagues intentionally increased T_{low} to prevent auto-PEEP.[35] They suspected that the presence of auto-PEEP was undesirable. In addition, many early trials with APRV were performed on patients with relatively normal or nearly normal pulmonary compliance, which added the variable of different pathological conditions to the study of T_{low} settings.[4,13,34] Several studies showed no significant deterioration in oxygenation or lung mechanics with a long release time.[30,31] In fact, a release time of 1.0 second or less, in some cases, resulted in increases in partial pressure of arterial CO_2 (P_aCO_2) and dead space ventilation, indicating that ventilation was inadequate even when spontaneous breathing was present.[2,21,29]

What appears to be essential is that T_{low} is established on the basis of the patient's pulmonary condition. For example, short T_{low} settings appear to be appropriate for patients with ARDS. Too long a release time can interfere with oxygenation and allow lung units to collapse. In the view of some practitioners, alveolar recruitment is enhanced by preventing complete exhalation in the slower compartments of the lungs, thus producing regional auto-PEEP.[21] This may help maintain an open lung and avoid repeated collapse and reexpansion of alveoli.[36-38] Atelectasis in the injured lung can develop in seconds when airway pressure drops below a certain critical level.[21,34,39,40] In neonates with ARDS, this time may be as little as 0.2 seconds.[41]

Recently a general consensus for using APRV in patients with ARDS suggests setting T_{low} between 0.5 and 1.0 second, typically at 0.8 second.[21,32,33] The T_{low} setting should generate regional intrinsic PEEP (areas of trapped air) and enhance alveolar recruitment.[21,36] In actual practice, clinicians tend to rely on the evaluation of the expiratory gas flow waveform. When expiratory flow during a release period is approximately 50% to 75% of peak expiratory flow (T-PEFR), the airway pressure should be allowed to return to P_{high}.[21,32]

T_{low} settings vary considerably among patients. Indeed, patients may require different settings as their lung condition changes. Establishing T_{low} should therefore be performed with caution and careful patient monitoring.

ADJUSTING VENTILATION AND OXYGENATION[21,32,33]

Ventilation and P_aCO_2 are both determined by the release time and V_T exchange during T_{low} and by the patient's spontaneous ventilation. If the P_aCO_2 is low and the patient's spontaneous rate is low, the machine rate can be reduced by lengthening T_{high} and/or lessening T_{low}. This is done until either the spontaneous rate increases significantly or respiratory acidosis occurs. If the patient's P_aCO_2 levels rise or tachypnea occurs, the rate can be increased.

If respiratory acidosis occurs, the patient should be evaluated for the level of sedation. It may be appropriate to reduce the level of sedation administered and allow more spontaneous ventilation. After sedation evaluation and adjustment, if CO_2 remains high, an increase in P_{high} may be necessary. For example, suppose a patient has a pressure gradient of 20 cm H_2O ($P_{high} - P_{low}$; 20 − 0 cm H_2O) and this gradient produces a V_T during release of 400 mL. Imagine the patient's pulmonary edema gets worse and the patient's lungs are less compliant. For the same pressure gradient, the V_T is now 300 mL. The P_{high} level should be raised to increase the pressure gradient ($P_{high} - P_{low}$) and effectively increase the V_T level. In general, P_{high} should not be allowed to increase above 30 cm H_2O to avoid high alveolar pressures.

Another strategy for improving ventilation is shortening T_{high}. A shorter T_{high} will result in more releases per minute and increase the patient's minute ventilation. Increasing T_{high} should be done with caution because the T_{high}/P_{high} combination (higher pressure for a longer time) is what increases the mean airway pressure and patient's oxygenation. If T_{high} is shortened so that the breath release is increased to more than 12 releases/min, this may compromise oxygenation.[32]

Another option for improving minute ventilation is to ensure that the release volume is optimized. If the T-PEFR is greater than 75% and oxygenation is acceptable, consider increasing T_{low} by increments of .05 to 1.0 second to achieve a T-PEFR of 50%. The APRV Clinical Scenario shows an example case of how changing T_{low} can increase V_T release. T_{low} should not be extended to the point at which closure of lung units can occur (derecruitment).

Another alternative is to allow increased P_aCO_2 levels (permissive hypercapnia) and use sedation because this condition is uncomfortable for the patient. However, hypercapnia in APRV patients may not develop because these patients can achieve adequate ventilation on their own.

Oxygenation can generally be improved by increasing P_{high} or F_IO_2. P_{high} should not exceed 30 cm H_2O to avoid overdistention injury to the lung. An additional strategy is to increase T_{high}. The increase of T_{high} and P_{high} increase the mean airway pressure. The clinician should be sure that T_{low} is set optimally for the patient. Oxygenation also seems to be enhanced when spontaneous ventilation is present. Using the prone position may also provide improvement in oxygenation if maintaining an adequate P_aO_2 is difficult (e.g., requiring high pressures and high F_IO_2 values).[35,37] Once oxygenation has improved, reduction of F_IO_2 is generally recommended until an F_IO_2 of less than 0.5 is achieved. Then P_{high} can be reduced gradually.

Clinical Scenario: APRV

A 73-year-old patient with a diagnosis of severe pulmonary fibrosis is awaiting a lung transplant. He is on APRV and has a pH of 7.27 and a P_aCO_2 of 63 mm Hg. V_T during a release with T-PEFR of 75% ranges from 190 to 230 mL. T_{low} is set at 0.6 seconds. Oxygenation is currently acceptable. T_{low} is progressively increased to 0.8 (T-PEFR of 50%), and the V_T increases to 350 to 375 mL. The resulting ventilation change is a pH of 7.35 and a P_aCO_2 of 55 mm Hg. This is an example of how lengthening T_{low} can result in a higher V_T and improvement in ventilation in a patient with respiratory acidosis.

DISCONTINUATION

Withdrawal of APRV can begin once the patient's lung condition has improved enough so that support can be safely reduced. The technique for reducing support is to adjust P_{high} and T_{high}. P_{high} should be reduced 2 to 3 cm H_2O at a time and T_{high} lengthened in 0.5- to 2.0-second increments, depending on the patient's tolerance.[32,33] (Simultaneously, T_{low} may be maintained or reduced.) P_{high} is slowly decreased until it meets P_{low}. P_{low} may be elevated slightly during the same process. During the lowering of P_{high}, some clinicians add pressure support to help compensate the spontaneous breathing efforts.[7] P_{high} and P_{low} pressures are intended to meet at the desired baseline, at which time the patient is essentially maintained at traditional CPAP or CPAP plus pressure support. Before switching to CPAP, the P_{high} should be approximately 14 to 16 cm H_2O or less and the T_{high} 12 to 15 seconds (Table 23.1).[21,32]

As the patient's condition improves, compliance will also generally improve. At P_{high} under improving conditions, the FRC is quite high and the patient tries to breathe spontaneously at this high lung volume. Therefore reducing the P_{high} allows the patient to achieve an adequate V_T at an appropriate FRC more easily. If tachypnea or respiratory acidosis occurs, or if the patient uses accessory muscles to breathe, the process should be stopped and the settings adjusted to more appropriate values.

TABLE 23.1 Example of Weaning APRV Settings in an Uncomplicated Case of Acute Lung Injury

P_{high} (cm H_2O)	T_{high} (s)	P_{low} (cm H_2O)	T_{low} (s)	Calculated P_{aw} (cm H_2O)
35	4.0	0	0.8	29.2
33	4.5	0	0.8	28.0
30	5.0	0	0.8	25.9
28	5.5	0	0.8	24.4
26	6.0	0	0.8	22.9
23	7.0	0	0.8	20.6
20	8.0	0	0.8	18.2
18	10.0	0	0.8	16.7
15	12.0	0	0.8	14.1

Following the final settings, the patient was transitioned to CPAP of 12 cm H_2O.
APRV, Airway pressure release ventilation; P_{aw}, mean airway pressure.
From Frawley PM, Habashi NM: Airway pressure release ventilation: theory and practice, *AACN Clin Issues* 12:234–246, 2001.

Although APRV promises benefits such as lower PIP, better oxygenation, and reduced hazards associated with high airway pressure, there is limited evidence to support these benefits.[7] However, APRV is worth pursuing because of its potential for lung recruitment and improved $\dot{V}/\dot{Q}$ ratios and its possible minimization of the compromising effects of positive pressure on cardiovascular function, particularly in patients with ARDS.

HIGH-FREQUENCY OSCILLATORY VENTILATION IN THE ADULT

High-frequency oscillatory ventilation (HFOV) has been proposed as an alternative method of respiratory support in adult patients with ARDS. Although HFOV has been used to treat neonates for some time, its use in the adult population has risen. The increased use of HFOV is based on the idea that it offers an alternative method of improving oxygenation in adult patients with ARDS and at the same time reduces the risks for VILI.

Theoretically HFOV by its design helps recruit the lung and avoid lung injury.[42-45] V_Ts with HFOV are quite small, with low alveolar pressure changes. Similarly, mean airway pressure (mP_{aw}), which is comparable to PEEP, is maintained at an adequate level to recruit the lung, at the same time avoiding repeated collapse and expansion of alveoli.[43] Overdistention of the lungs is avoided. Chapter 22 reviews the various forms of HFV, their mechanisms of action, and application in the newborn population. This section focuses on HFOV as used in the adult.

Although HFOV has been used since the late 1950s, its use in adults has only recently gained a solid footing in clinical practice.[43,44-53] The slow adoption of using HFOV in the management of adults with ARDS may be the result of several factors. First, a commercial high-frequency oscillator with sufficient power to ventilate an adult was not available until the 1990s.[54] Second, adult practitioners accustomed to using conventional ventilators were

reluctant to use this technology. Indeed, in spite of its popularity among neonatal practitioners, actual outcome benefits of HFOV compared with conventional ventilation remained controversial and not well established.[39,55-57]

Two early series of case studies used high-frequency oscillation (HFO) in adult patients as a rescue technique after conventional ventilation.[57,58] In these studies, patients were maintained on high levels of conventional support (high PEEP, high PIP, high P_{plat}, and high F_IO_2) before being switched to HFO. Both studies showed an improvement in gas exchange when patients were switched from conventional ventilation to HFO. Mortality rate in both studies was directly related to the length of time on conventional ventilation.

In a prospective, randomized, controlled trial comparing HFOV and pressure control ventilation in adults with ARDS, HFOV was shown to be as effective and safe as pressure control ventilation. The 30-day mortality rate for patients in the HFOV group was 37%, and the mortality rate for patients receiving conventional ventilation was 52%. Although better survival appeared to occur in the HFOV group, this finding was not statistically significant.[59]

Evidence that clearly demonstrates improved survival in adults with ARDS using HFOV is not currently available. Although HFOV may improve P_aO_2 in some patients and thereby allow F_IO_2 to be lowered, this improvement may only be transitory.[60]

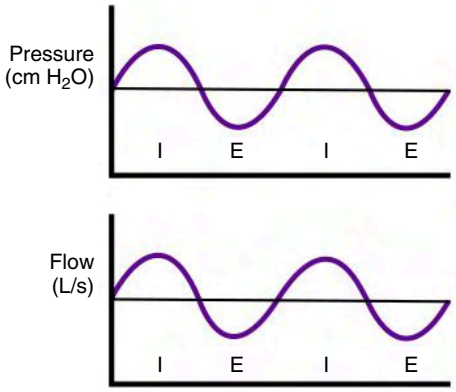

Fig. 23.5 The sinusoidal waveform generated by an oscillator. *I*, Inspiration (gas flow into the patient); *E*, expiration (active gas flow out of the patient). (From Cairo JM, Pilbeam SP: *Mosby's respiratory care equipment,* ed 8, St Louis, MO, 2010, Mosby.)

TECHNICAL ASPECTS

HFOV is a method of high-frequency ventilation (HFV) in which gas is oscillated at high frequencies (3–15 hertz [Hz]). The Vyaire 3100B High-Frequency Oscillatory Ventilator (Vyaire Medical, Mettawa, IL) is an example of an adult HFO currently available. It was approved by the U.S. Food and Drug Administration (FDA) and introduced in 2001. Technical aspects of this specific ventilator are described elsewhere.[61]

HFOs are produced by a reciprocating piston with the 3100B. The resulting flow waveform approximates a sine wave (Fig. 23.5). With HFOV, pressure is positive in the airway during the inspiratory phase (forward stroke) and negative during the expiratory phase (return stroke). Thus inspiration and expiration are active, resulting in bulk flow rather than jet pulsations.

The diaphragm-shaped piston used in this device is magnetically driven, much like a stereo speaker. Gas is oscillated back and forth by the action of the diaphragm. The amplitude of the wave, which is set by the power control, determines the forward and backward excursion of the piston and thus helps determine V_T. A low-compliance plastic circuit provides humidified bias flow of gas to the patient (Fig. 23.6).

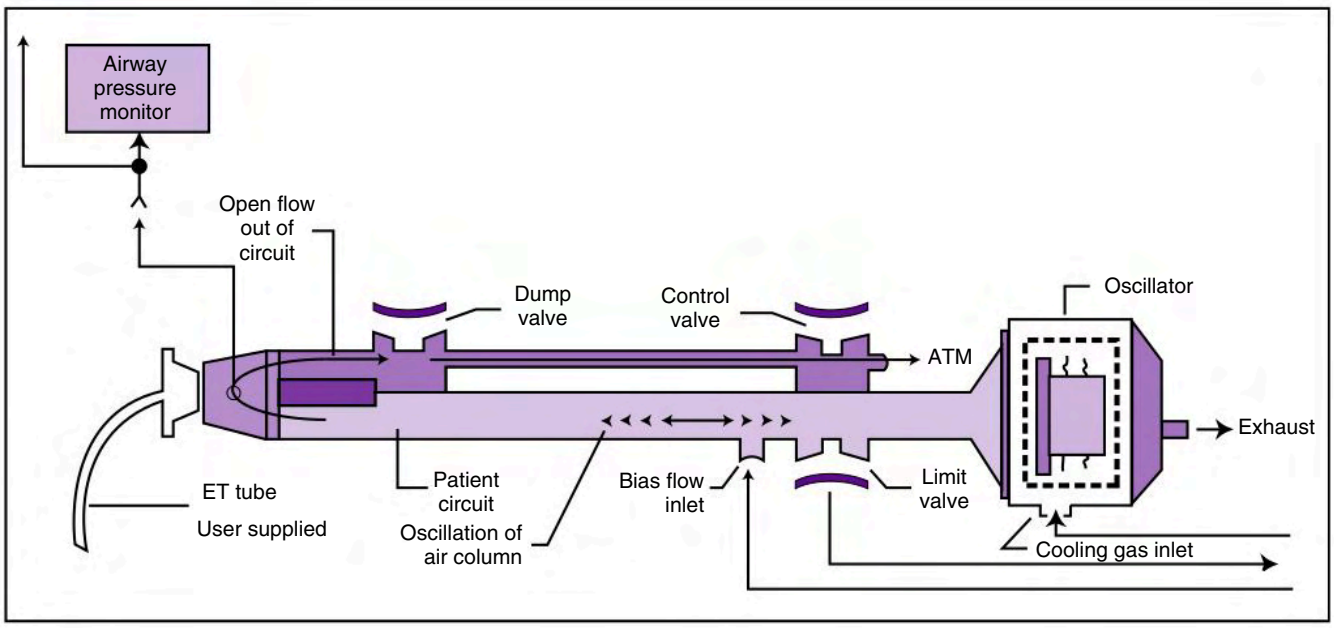

Fig. 23.6 Schematic of the Vyaire 3100B High-Frequency Oscillatory Ventilator patient circuit. A dump valve located on the expiratory limb opens when the oscillator (piston) is off. The mP_{aw} control valve is located at the end of the expiratory limb. As the pressure to this valve is increased, the outflow of gas is restricted and the mP_{aw} increases. The limit valve located on the inspiratory limb is used to limit inspiratory pressure. When the set pressure limit is exceeded, this valve opens. (Courtesy Vyaire Medical, Mettawa, Ill.)

INITIAL CONTROL SETTINGS

Because the use of HFOV is still being researched, the following recommendations for initial settings come from available published studies.[54,55,59]

Parameters that are set in HFOV include the following:

- mP_{aw} (analogous to $\overline{P}_{aw}$ on conventional ventilation)
- Power (the peak-to-trough pressure difference generated by the piston movement)
- Frequency (in hertz)
- Inspiratory time percentage
- Flow (bias flow)

Mean Airway Pressure

The mP_{aw} is a direct control located on the front panel of the Vyaire 3100B High-Frequency Oscillatory Ventilator (Fig. 23.7). It is generated by a continuous flow of gas past the variable resistance of a mushroom valve. In the 3100B, the balloon, or mushroom, valve is located in the expiratory limb of the circuit (see Fig. 23.6). As the balloon is inflated, the outflow is restricted and the airway pressure increases. The mP_{aw} must be kept at the target value because it can drift. This drift can occur when other controls that affect mP_{aw} are changed.

The mP_{aw} directly affects P_aO_2 by changing lung volume. Initially, mP_{aw} is set at 3 to 5 cm H_2O above the observed P_{aw} during conventional ventilation. A starting range is typically 25 to 30 cm H_2O. As the mP_{aw} is increased, the volume in the lungs increases and the patient's diaphragm is displaced downward. For example, one goal of mP_{aw} in the neonatal population is to position the diaphragm between the eighth and ninth thoracic vertebrae on a chest radiograph. This position usually correlates with good lung expansion and an acceptable P_aO_2; it may also correlate with adequate lung expansion in the adult, that is, visualization of the ninth posterior rib above the level of the diaphragm in the midclavicular line on the chest radiograph.[59,62] The mP_{aw} should not be reduced during the first 24 hours, to allow an adequate time for as many lung units as possible to be recruited. An exception to this is if mP_{aw} is initially set too high. As previously mentioned, assessment of mP_{aw} is done by observing the expansion of the thorax on a chest radiograph (Key Point 23.2). If hemodynamic monitoring is being used, further assessment of mean arterial pressure may include determining the ability of the right side of the heart to fill in the presence of the continuous high pressure in the thorax.

Key Point 23.2 Wide swings in the mP_{aw} value during ventilation may indicate leaks in the system, water in the expiratory circuit, or that the patient is making spontaneous breathing efforts.

Establishing appropriate mP_{aw} during HFOV has been studied in an animal model.[63] Setting mP_{aw} at 1.5 times the lower inflection point of a static pressure—volume curve was sufficient to reestablish pre—lung injury P_aO_2.

Amplitude

Amplitude influences the level of ventilation (P_aCO_2) and can be adjusted by the power control, which sets the amount of piston displacement and resulting amplitude of the oscillating waveform (Fig. 23.8).[64] As amplitude is increased, the pressure gradient (ΔP) increases and V_T increases. (In HFOV, the equation dictating ventilation is not $f \times V_T$, but $f \times V_T^2$.)[62] Adjusting the power control changes the amount of power going to the piston and the amount of piston displacement or forward-backward movement. Piston displacement affects ΔP created by a piston (oscillator) excursion (see Fig. 23.6). The settings control panel ranges from 1.0 to 10. It is adjusted by using a control located adjacent to the amplitude readout (see Fig. 23.7). A starting power setting of 6 to 7 is typical for adults. The appropriateness of the power setting is determined by observing chest wall movement or "chest-wiggle factor" (CWF). Increased amplitude is also associated with increased chest wall movement. The CWF should be visible from the level of the clavicle to the midthigh. (Assessment of chest wiggle is difficult in obese patients.) Placing a pencil or tongue depressor on the leg at the midthigh level may provide a visual indicator of the wiggle at thigh level. In practice, some clinicians have found that a setting that results in a ΔP of 60 to 70 cm H_2O around the mP_{aw} settings is a good starting point until arterial blood gas (ABG) results can be obtained.[64] Another recommended starting point for amplitude used by some clinicians is to determine the P_aCO_2 before HFOV and add 20 to this value. For example, if P_aCO_2 on conventional ventilation was 60 mm Hg, starting amplitude will be 80.

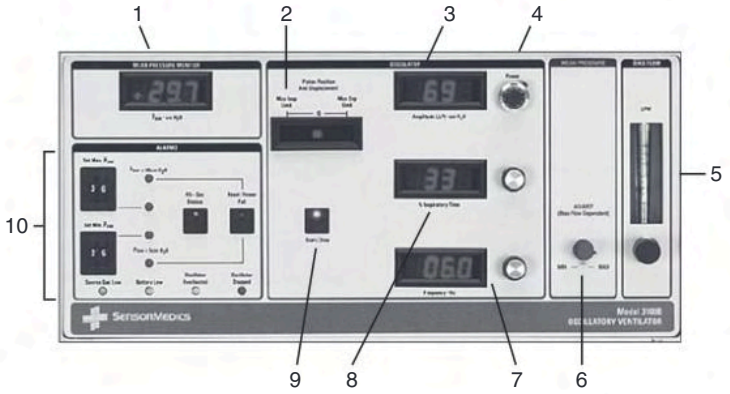

Fig. 23.7 Control panel of the Vyaire 3100B High-Frequency Oscillatory Ventilator. *1*, Display of mP_{aw}; *2*, indicator of piston movement; *3*, amplitude display; *4*, power control knob; *5*, bias flow display and control; *6*, adjustment for mP_{aw}; *7*, frequency control and display; *8*, T_I% control and display; *9*, on/off control for oscillating piston; *10*, alarm settings and indicators panel. (Courtesy Vyaire Medical, Mettawa, Ill.)

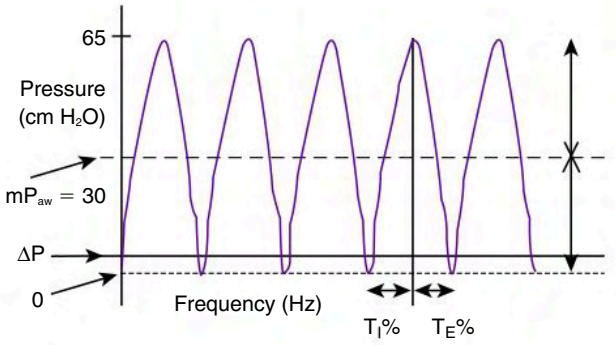

Fig. 23.8 The waveform for pressure-time produced during high-frequency oscillatory ventilation. The oscillations occur above and below the set mP_{aw} (30 cm H_2O). The amplitude of this oscillating pressure (ΔP) is approximately 70 cm H_2O, or 35 cm H_2O above and below the mP_{aw} setting. *T_E%,* Expiratory time percent; *T_I%,* inspiratory time percent.

In addition to the actual power setting, other factors that affect the amplitude include the endotracheal tube (ET) size, R_{aw}, and C_L. Changes in R_{aw} and C_L will affect the amplitude delivery.[65] For example, at a specific power setting, a *measured* amplitude increase could be caused by an increase in R_{aw} or a decrease in C_L. The amplitude measured at the airway is significantly higher than the pressures delivered to the lung. The size of the ET, branches of the bronchial tree, and distance from the piston can attenuate pressure delivery.

Frequency

The frequency setting controls the time allowed for the piston to move forward and back. It is a secondary control for ventilation. The available range is 3 to 15 Hz (180–900 cycles/min). Reducing the frequency causes a greater volume displacement and thus a larger V_T and higher $\dot{V}_E$. Conversely, increasing the frequency results in a smaller volume displacement, a smaller V_T, and lower $\dot{V}_E$. An initial setting for an adult is 5 to 6 Hz (300–360 cycles/min). One management strategy to reduce P_aCO_2 is to lower the frequency setting. (NOTE: It is not typically set lower than 3.) Generally, once the frequency is established, it is usually not changed during the course of treatment. Occasionally some clinicians may change the frequency after changes in amplitude are made in an effort to alter the patient's $\dot{V}_E$ (low frequency tends to increase $\dot{V}_E$, and high frequency tends to decrease $\dot{V}_E$).

Inspiratory Time Percentage

The T_I percentage (T_I%) represents the portion of the respiratory cycle that the piston spends in a forward motion. An initial setting of 33% (I/E of 1:2) is usually appropriate for an adult. The T_I% control may also assist in CO_2 elimination, but to a lesser extent than amplitude and frequency. For some patients, a T_I% of 50% may improve ventilation (CO_2 removal) and lung recruitment. Once this parameter is set, it is generally not changed.

Bias Flow

The bias flow control is usually the first parameter set when starting HFOV. A typical range for an adult is 25 to 40 L/min (e.g., 30 L/min). Changing the bias flow affects mP_{aw}. If flow is inadequate, the set mP_{aw} cannot be reached. Low flow may result in an increase in P_aCO_2 from inadequate washout of exhaled gas. If the set bias flow is too high, damping of active exhalation may inhibit CO_2 elimination. The backward motion of the piston is responsible for active exhalation (Key Point 23.3). With a high bias flow the piston may only remove air from the circuit. This may also increase the P_aCO_2 and result in auto-PEEP.

***Key Point* 23.3** In HFOV, during the expiratory phase the backward movement of the piston actively moves air out of the system.

Additional Settings

The F_IO_2 is usually set at 1.0 and decreased in increments of 0.1 (range, 0.05–0.10), as tolerated, to maintain the target range for P_aO_2 or O_2 saturation (S_pO_2). F_IO_2 may also be set similar to the way it is set with conventional ventilation and titrated based on S_pO_2 and ABGs. An ABG should be obtained 30 to 60 minutes after any change in mP_{aw}, amplitude, or frequency.[59]

Before transferring a patient from a conventional ventilator to the oscillator, the F_IO_2 may be increased to 1.0. After transfer, a recruitment maneuver (RM) is recommended because the patient is briefly disconnected from the ventilator (Box 23.1).[64,65] If the patient becomes disconnected, the RM should be repeated. Some institutions perform an RM on a regular basis, such as every 12 hours, if an F_IO_2 of more than 0.4 is required to maintain oxygenation. This would be appropriate as long as the patient responded to an RM with an improvement in oxygenation (Key Point 23.4).[54]

***Key Point* 23.4** Lung recruitment may be an important aspect of HFOV, whether HFOV is being used as a rescue technique or as a primary mode to prevent VILI.

BOX 23.1 Recruitment Maneuver for Use During High-Frequency Oscillatory Ventilatory[a]

To perform a recruitment maneuver:

- Increase the F_IO_2 to 1.0.
- Stop the piston (pressure from the oscillation of the piston can be transmitted to the peripheral lung in addition to the increased setting of mP_{aw} during the maneuver).[b]
- Turn the mP_{aw} up to 40 cm H_2O for 40 to 60 seconds, or 10 cm H_2O above the mP_{aw} setting from an already established mP_{aw}, while the piston is stopped.
- Discontinue the procedure if the patient becomes hypoxemic (S_pO_2 <85%) or hemodynamically compromised (mean blood pressure <60 mm Hg, a drop of blood pressure of >20 mm Hg from baseline, an increase in heart rate to >140 beats/min, a fall in heart rate to <60 beats/min, or development of a new arrhythmia).
- After the procedure, turn the mP_{aw} to an appropriate initial setting or back to the previous setting.

[a] A recruitment maneuver should not be performed if a pneumothorax or bubbling chest tube (active air leak) is present.

[b] Neonatal tubes are so small that only about 5% of the ΔP is transmitted to the distal end of the endotracheal tube (ET). However, in adults with large ETs, up to 20% of the ΔP can be transmitted to the carina level.

BOX 23.2 Indications and Precautionary Use of the Vyaire 3100B High-Frequency Oscillatory Ventilation

Indications

The 3100B is indicated for use in ventilatory support and treatment of selected patients (≥35 kg) with acute respiratory failure.

Contraindications

No contraindications exist for the use of the 3100B.

Precautionary Use

The 3100B HFO should be used with caution in the following patient conditions:

- Unstable cardiovascular status
- Acute bronchospasm
- Severe acidosis (the 3100B HFV is not designed to ventilate so much as oxygenate)
- Pregnancy
- Chronic obstructive pulmonary disease or asthma requiring the use of aerosolized bronchodilators

A few hours after initiation of HFOV, a chest radiograph should be obtained to determine the appropriateness of the mP_{aw} setting. The chest radiograph is taken while the piston is turned on because the information desired is how well recruited the lungs are on the current setting. If more than 11 posterior ribs are showing, mP_{aw} needs to be slowly decreased in increments of 2 cm H_2O until an appropriate inflation point is reached.

INDICATION AND EXCLUSION CRITERIA

Patients with ARDS weighing at least 35 kg and who are not responding to mechanical ventilation may be candidates for HFOV. Failure may be defined as the presence of unresponsive severe hypoxemia (F_IO_2 >0.6; PEEP 10 cm H_2O or more, with a P_aO_2 ≤65 mm Hg) or a significant risk for VILI.[59] Indications for HFOV depend on the institutional policy. Additional indications might include the following:

- A diagnosis of H1N1 influenza infection with ARDS
- Air leaks in patients
- Early intervention to recruit the lungs
- Clinical staff comfort with using the equipment

Criteria for the use of the Vyaire 3100B High-Frequency Oscillatory Ventilator are listed in Box 23.2. Another exclusion criterion involves patients requiring aerosolized medications. Use of the small-volume ultrasonic-type nebulizers or a vibrating mesh nebulizer may provide a solution to the problem of aerosol delivery during HFOV.[66] Further study of this topic is certainly warranted.

MONITORING, ASSESSMENT, AND ADJUSTMENT

The oscillator should be calibrated according to the manufacturer's recommendations before use. Because patients need to be suctioned regularly (at least every 12 hours), a closed-suction catheter system should be placed in the patient circuit. The connection for a closed-suction catheter is typically placed at a right angle in the circuit. This may affect the oscillatory pressure waveform; therefore chest excursions should be assessed after placing the system in the circuit. A suction catheter is still required to help maintain airway patency. Some clinicians use an inline O_2 analyzer for continuous monitoring of delivered F_IO_2.

The 3100B uses a standard heated humidifier with a closed feed system to maintain water level, which can provide adequate humidification. The water-feed system should be pressurized to keep water flowing into the device. Humidification can be verified when condensate appears at the distal end of the inspiratory circuit. The circuit should be kept free of water. Because of condensate, frequent checking of the circuit is required.

To transition from conventional ventilation to HFOV, sedation and analgesics may be required. The patient is sedated because vigorous spontaneous breathing efforts should be avoided during HFOV. A strong spontaneous inspiration may reduce circuit pressures below 5 cm H_2O, which can result in desaturation. A reduction in pressure this low would also trigger a disconnect alarm and shut off the oscillator piston. For this reason, patients are typically sedated with a combination of benzodiazepines (e.g., midazolam) and a narcotic (e.g., fentanyl).[60,64] Paralysis with such agents as pancuronium, cisatracurium, or vecuronium may also be required, particularly for the first 30 minutes or so of the procedure. (See Chapter 15 for information on these medications.) When a paralyzing agent is used, train-of-four monitoring is recommended to reduce the risk for prolonged neuromuscular blockade. Ulnar nerve placement avoids the chest, upper air area, and thigh areas—where wiggling is present—and might interfere with monitoring. (See Chapter 15 for information on train-of-four monitoring.)

Once the patient is receiving HFO, the clinician should listen for bilateral breath sounds to determine whether the intensity of breath sounds is equal over both lungs. A reduction in intensity may indicate a pneumothorax or malposition of the ET. Breath and heart sounds may be difficult to hear, so other forms of assessment should be performed (e.g., determination of C_L) once HFOV is initiated. To listen for heart sounds, the piston must be momentarily stopped while the CPAP level is maintained. Disconnecting the patient would cause derecruitment of the lungs and is therefore not recommended (Case Study 23.1).

The CWF should also be checked hourly. CWF may change with ET obstruction, right main-stem intubation, a pneumothorax, or an accidental extubation.[55]

Rotation of the patient is more difficult with HFOV than during conventional ventilation and must be performed cautiously in order not to disconnect the patient or cause an accidental extubation. A circuit disconnection with a loss of pressure will cause the oscillator to stop. For this reason, a manual resuscitator

Case Study 23.1

Patient Assessment During HFOV

A patient receiving ventilation by HFOV exhibits a sudden drop in S_pO_2, a reduction in CWF, tracheal deviation to the left, and a sudden drop in mP_{aw}. What is the most likely cause of these sudden changes?

with a PEEP valve attached should be kept at the bedside. (NOTE: A circuit disconnection may occur where the expiratory valve tubing attaches to the circuit.[64]) Continuous lateral rotation may also be safely accomplished using a specialized rotating bed (e.g., RotoRest, KCI Medical, Kidlington, UK).

ADJUSTING SETTINGS TO MAINTAIN ARTERIAL BLOOD GAS GOALS

Oxygenation during HFOV is achieved primarily by maintaining the mP_{aw} at a level sufficient to obtain optimal lung inflation.[59] At a set level of amplitude, increases in mP_{aw} result in increases in lung volume and available alveolar surface area. As long as cardiac output is not adversely affected, oxygenation generally improves (Fig. 23.9). (See Chapter 13 for lung recruitment strategies.) Conversely, decreases in mP_{aw} result in a decrease in oxygenation if lung derecruitment occurs.

An overall goal for oxygenation in patients with ARDS is a P_aO_2 of 55 to 80 mm Hg and an S_pO_2 of 88% to 95%.[67] If an F_IO_2 greater than 0.6 is required to maintain these goals, mP_{aw} can be increased by 2 to 3 cm H_2O (maximum, 45 cm H_2O) until the F_IO_2 can be decreased to less than 0.6.[59] Changes should be made slowly (every 30 minutes) to allow the opportunity for determining whether alveolar recruitment and improvement in S_pO_2 and P_aO_2 has occurred. (In neonates, alveolar recruitment may require up to 25 minutes.[59] The time required in adults is not currently known.) Before making these changes, an RM is recommended (see Box 23.1) unless a pneumothorax or an active air leak is present, and the chest drainage system to compensate for the leak is bubbling.

If severe hypoxemia persists, inhaled nitric oxide might be added or the patient may be placed in the prone position.[68]

To decrease P_aO_2, F_IO_2 is reduced in increments of 0.05 to 0.10, until an F_IO_2 of 0.6 is reached. Once F_IO_2 is 0.6 or less, reductions in F_IO_2 can be alternated with reductions in mP_{aw} (increments of 2–3 cm H_2O). Weaning of mP_{aw} should be done carefully and slowly until a minimum level of 20 to 24 cm H_2O is reached and F_IO_2 is less than 0.5. These suggested changes are one approach to targeting the oxygenation goals. Some institutions may have different approaches.

Key Point 23.5 The ET can block part of the pressure (volume) transmission to the lower airways. The smaller the ET, the more the pressure is damped. With a large ET more pressure (volume) is transmitted to the lungs, providing better ventilation.[59]

The maintenance of ventilation (pH and P_aCO_2) may be more difficult with an oscillator than with a conventional ventilator. During HFOV, $\dot{V}_E$ and V_T delivery are affected by several factors, including the pressure amplitude of oscillation (ΔP), frequency, ET size, amount of ET cuff leak, and lung characteristics (C_L and R_{aw}) (Key Point 23.5).[59]

As the piston moves, it applies cyclical pressure above and below the set mP_{aw} (see Figs. 23.8 and 23.9). As the power setting is increased and ΔP increases, V_T and $\dot{V}_E$ increase. This improves CO_2 elimination and lowers P_aCO_2. Decreasing P_aCO_2 can generally be accomplished in most patients by changing only the amplitude to decrease the ΔP. The power setting can be increased in increments of 0.5 to 1.0 until the maximal setting is reached (10 is the maximum on the control of the 3100B). Observing changes in CWF can assess power settings.

In some cases a change in frequency may also be required to alter P_aCO_2. Decreasing the frequency (hertz) increases the time of piston movement. In addition, as frequency is reduced, the attenuation of the wave signal through the ET is diminished and a larger ΔP occurs (Key Point 23.6).[64] This increases the delivered

Key Point 23.6 An upward adjustment of frequency results in an increase in P_aCO_2, whereas a downward adjustment of frequency results in a decrease in P_aCO_2.[59]

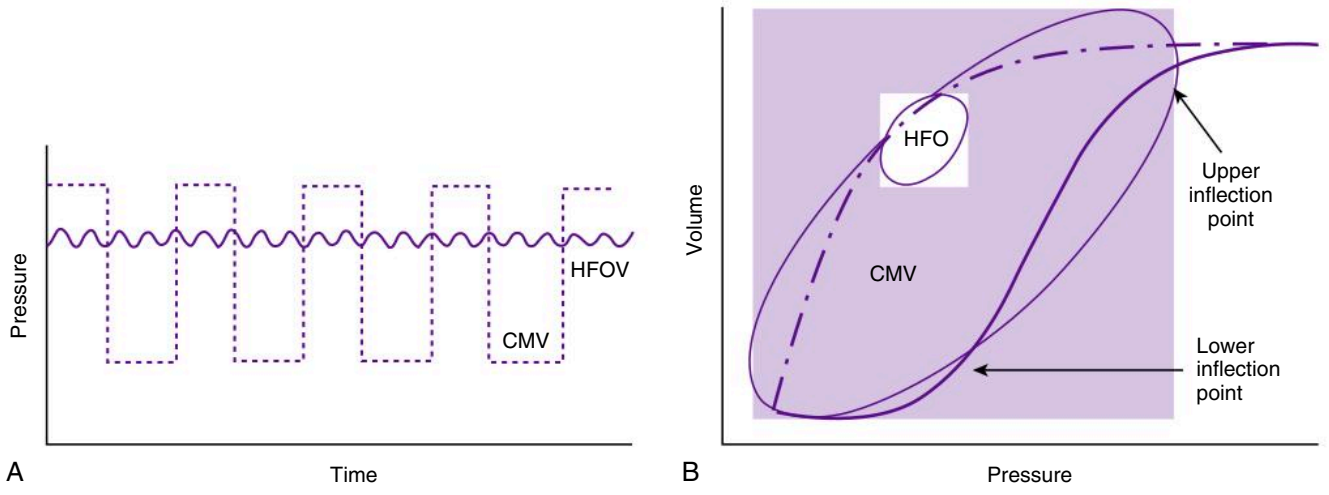

Fig. 23.9 (A) The pressure-time curve contrasting tidal variations in pressure with conventional mechanical ventilation *(CMV) (dashed line)* and high-frequency oscillatory ventilation *(HFOV) (solid line)*. (B) Pressure-volume loop showing inflation *(solid line)* and deflation *(dashed line)* comparing the potential for overdistention and alveolar collapse during tidal breathing with CMV and HFOV *(small oval)*. (From Ferguson ND, Stewart TE: The use of high-frequency oscillatory ventilation in adults with acute lung injury, *Respir Care Clin N Am* 7:647–661, 2001.)

BOX 23.3 Example of the Effect of Changing Amplitude and Frequency on Tidal Volume in High-Frequency Oscillatory Ventilation

Suppose the initial amplitude produces a ΔP of 50 cm H_2O, and the frequency is set at 8 Hz and the inspiratory time at 33%. If the amplitude is increased such that ΔP is increased to 60 cm H_2O, this can result in an increase in tidal volume (V_T). The same change in V_T can be accomplished by keeping the amplitude the same while decreasing the frequency from 8 Hz to 4 Hz.

V_T and the distal transmission of the ΔP to the lungs, thereby enhancing ventilation. Decreasing the frequency is accomplished in increments of 1 to 2 Hz until a minimum of 3 Hz is reached.[64,68] Box 23.3 provides an example of the effect of changing amplitude and frequency in HFOV.[69]

Conversely, increasing the frequency reduces V_T and CO_2 elimination and results in a higher P_aCO_2. Increasing the frequency too much with HFO may actually lead to air trapping and an increase in P_aCO_2. Thus adjustments in frequency should be done cautiously. "Adequate" ventilation may be a P_aCO_2 that keeps the pH greater than 7.20 and allows permissive hypercapnia.

If high P_aCO_2 persists in spite of changes in amplitude and frequency, an increase in V_T can be accomplished by increasing T_I% from an initial setting of 33% to 50%. Notice that this may also increase mP_{aw} and the risk for air trapping.

When amplitude, frequency, and T_I% have been optimally adjusted and CO_2 remains elevated, providing a deliberate cuff leak may increase the flow of gas through and around the ET and augment CO_2 elimination.[64] To set a cuff leak, the practitioner removes some of the air from the cuff until the mP_{aw} decreases slightly. The cuff leak is kept at this level, and the mP_{aw} is restored to its prior setting by increasing the bias flow. To confirm the cuff leak, the pilot balloon is squeezed to observe the measured mP_{aw}. Squeezing the pilot balloon should cause a rise of approximately 5 cm H_2O in mP_{aw}. ABG evaluation is performed after a cuff leak adjustment (15–30 minutes) to confirm that the leak was effective in lowering P_aCO_2.[59] Box 23.4 summarizes ventilator adjustments for reducing P_aCO_2 during HFOV.[59,69] To increase P_aCO_2, the previous steps can be reversed. (If the frequency [hertz] is increased, mP_{aw} may decrease.)

If a severe rise in P_aCO_2 occurs abruptly, the respiratory therapist should assess the patient immediately by manually ventilating the patient and passing a suction catheter through the ET to determine whether the ET or the airway is obstructed. The ventilator circuit should also be inspected for tube crimping or obstructions (Key Point 23.7).[64]

BOX 23.4 Steps to Reduce P_aCO_2 During High-Frequency Oscillatory Ventilation

1. Increase amplitude (ΔP) incrementally.
2. If hypercapnia persists and amplitude (power setting) is at maximum, decrease frequency by 1 Hz to a minimum of 3 Hz.
3. If hypercapnia persists, increase the inspiratory time to 50%.
4. If hypercapnia still persists, try increasing the cuff leak.

***Key Point* 23.7** A significant increase in P_aCO_2 can occur as the result of a few centimeters of a suction catheter from a closed-suction system remaining in the airway. Narrowing the lumen of the ET reduces effective ventilation during HFOV.[64]

RETURNING TO CONVENTIONAL VENTILATION

Once the patient is stable with an mP_{aw} of 20 to 24 cm H_2O and a S_pO_2 of 88% or more on an F_IO_2 less than 0.5, the patient can be checked for readiness to switch back to conventional ventilation. Readiness can be evaluated by observing a resolution of initial lung injury, tolerance of suctioning (i.e., if significant desaturation does not occur), and tolerance of manual ventilation on 50% O_2 and PEEP, 10 cm H_2O or greater (S_pO_2 remains more than 90% during the procedure).[59] If the patient does well during these procedures, he or she can then be transferred to a conventional ventilator.

Some clinicians transition patients to PC-CMV with low V_T settings (6 mL/kg ideal body weight [IBW] and high PEEP levels, 10–15 cm H_2O).[59,64] Others favor modes such as APRV, which allows a close matching of airway pressures to the mP_{aw} on HFVO during the transition.[64]

In summary, HFOV has been available for some time for the management of infants and may be a viable option for managing adult patients with ARDS.[54,70-76] When HFOV is used, timing is important. HFOV should be started early to avoid VILI associated with conventional ventilation. (See Chapter 17 for information on VILI.) The role of HFOV in the management of adults with ARDS needs to be verified with prospective, randomized, controlled trials.[70,77]

HELIOX THERAPY AND MECHANICAL VENTILATION

Helium (He)-O_2 mixtures (**heliox**) have been used in the treatment of a number of disorders that cause airway obstruction (Box 23.5). It is approved by the FDA as a diagnostic gas; the FDA has also cleared a number of devices for the therapeutic use of heliox mixtures.[78-80]

BOX 23.5 Disorders Treated With Heliox

- Postextubation stridor
- Asthma
- Tracheobronchitis
- Viral laryngitis
- Laryngotracheal bronchitis
- Bronchiolitis
- Tumors (laryngeal or mediastinal)
- Foreign body aspiration
- Vocal cord paralysis
- Dyskinesia
- COPD

BOX 23.6 Properties of Helium

Except for hydrogen, helium (He) is the most abundant element in the universe. On earth it can be found only in natural gas fields in North America (United States and Canada).[75] He is odorless and colorless. It has the second lowest density of any known gas, second only to hydrogen. The density of He is 0.18 g/L. Nitrogen's density is much higher, at 1.25 g/L, and O_2 is even higher, at 1.43 g/L. Air is 1.29 g/L. He has the lowest melting point of any element and a boiling point close to absolute zero. In addition, He has a high thermal conductivity.

BOX 23.7 Equations Describing Flow-Through Tubes

The Reynold's number for a flow through a pipe is defined as:

$$Re = (2 \times \dot{V} \times \rho)/(\pi \times r \times \mu)$$

where *Re* is Reynold's number, $\dot{V}$ is the flow (in milliliters per second), ρ is the density of the gas (in grams per milliliter), *r* is the radius of the tube (in centimeters), and μ is the gas viscosity (in grams per centimeter per second).[81]

Poiseuille (laminar) flow is experimentally found to occur for a Reynold's number less than 2000. At larger Reynold's numbers ($\geq$4000), flow becomes turbulent.

In *laminar* conditions the following applies: $\Delta P = k^1 (\dot{V})lam$, where ΔP is the pressure difference, k^1 is the coefficient of linear resistance, (which equals $8\eta l/\pi r^4$; where η is the gas viscosity in grams per centimeter per second, *l* is the length of the tube in centimeters, and *r* is the radius of the tube in centimeters), and $(\dot{V})lam$ is the laminar flow (in milliliters per second). Rewritten as Poiseuille's equation for laminar flow,

$$\Delta P = (8/\eta\dot{V})/(\pi \times r^4)$$

In *turbulent* conditions, $\Delta P = k\,(\dot{V}turb^2)$, where $\dot{V}turb^2$ is turbulent flow (in milliliters per second) and *k* is the coefficient of nonlinear resistance and equals $fl/4\pi^2r^5$ (where *f* is the friction factor dependent on the Reynold's number and roughness of the tubing wall). In a small tubing, $f = 0.316/Re^{1/4}$, where *r* is the radius (in centimeters).

The effect of He is based on its low density. Box 23.6 lists some of the physical properties of He.[81] The low density of He reduces turbulent flow in obstructed airways and decreases respiratory muscle load and dyspnea. In cases of severe reactive airway disease (e.g., asthma), heliox delivery may reduce the WOB through partially obstructed airways.[81,82]

Another key property of He regarding its medical application is the fact that it is an inert gas (Key Point 23.8).

Key Point 23.8 Because He is an inert gas, it will not react with human tissue or with pharmaceutical agents.

GAS FLOW THROUGH THE AIRWAYS

Because heliox is effective only when turbulent flow is present, it is important to understand where and when turbulence is likely to occur. Normal airflow in the larynx and trachea is turbulent and is the predominant gas flow pattern in the larger airways of the first and second generation of bronchi (mainstem and lobar bronchi) during normal, quiet breathing. The transition to laminar flow occurs somewhere between the third and eleventh generations of bronchi, depending on the rate of gas flow (Box 23.7).[81-83]

During laminar flow in the small airways, the driving pressure is proportional to flow and is influenced by the viscosity of the gas being breathed. Density does not affect the pressure—flow relation in laminar flow. During turbulent flow the driving pressure is proportional to the flow squared, and gas density is more important than viscosity as a determinant of flow.[81] As a low-density gas, He provides a greater flow for a given ΔP when flow is turbulent. Heliox is therefore expected to be more beneficial in diseases affecting the larger airways and partially obstructed airways, where flow is turbulent. When He reduces the Reynold's number to less than 2000, laminar flow predominates, thereby reducing R_{aw}.

HELIOX IN AVOIDING INTUBATION AND DURING MECHANICAL VENTILATION

With an acute exacerbation of asthma, central airways are narrowed from mucus plugging, mucosal edema, and smooth muscle constriction, increasing turbulent flow and R_{aw}. In the treatment of spontaneously breathing, nonintubated patients with severe asthma episodes, heliox has been shown to increase peak inspiratory flow rate. It may also reduce the risk for respiratory muscle fatigue until bronchodilator and corticosteroid therapy become effective (Key Point 23.9).[81,84] Heliox has also been shown to reduce the incidence of pulsus paradoxus in patients experiencing a severe asthma exacerbation.

Early in a severe asthma exacerbation, the obstruction is located in the central airways. As time progresses (more than 96 hours), however, the peripheral airways become obstructed with secretions and edema. Thus heliox is more likely to be beneficial early in the process.[83] When heliox is required in treating asthma, it may be required for up to 12 to 24 hours and has been used in some facilities up to 5 days.[83,85] In patients with COPD or small airway disease, heliox may not be helpful in reducing R_{aw} if it occurs in the peripheral regions of the lungs.[83]

At present no guidelines exist for the indications and timing of heliox in patients with severe asthma. Some clinicians recommend using heliox when aggressive treatment with bronchodilators fails to improve the patient's condition within 30 minutes.[81] Others suggest intubation and mechanical ventilation when the patient's assessment deteriorates into acute respiratory failure and to consider heliox only if the patient is then stabilized.[83] Regardless of approach, heliox does *not* replace aggressive bronchodilator and

Key Point 23.9 Heliox ventilation does not cure the cause of partial airway obstruction. It should be used only as an adjunct therapy to reduce work of breathing until the obstruction can be alleviated.[81]

corticosteroid therapy (See Clinical Scenario on severe asthma).[81,82]

Clinical Scenario: Severe Asthma

A 24-year-old woman vacationing in Florida was admitted to a local emergency department with status asthmaticus. She was conscious on admission and indicated she had taken 80 puffs from an albuterol metered-dose inhaler (MDI) before going to the emergency department but had no relief of her symptoms. ABGs obtained while she was breathing enriched O_2 through a nonrebreathing mask were pH = 6.98, P_aCO_2 = 125 mm Hg, and P_aO_2 = 78 mm Hg. The patient was able to request heliox verbally. She had previously been treated with heliox successfully. Although the respiratory care department did not routinely perform this procedure, they were willing to comply.[81,82]

The patient was intubated and mechanically ventilated. She received sedation and paralyzing agents. Heliox was obtained from another local hospital and instituted within 2 hours of intubation. PIP dropped dramatically from 80 cm H_2O to 40 cm H_2O when heliox was instituted. The patient required ventilation for less than 3 days. No pneumothorax occurred during ventilation.

POSTEXTUBATION STRIDOR

In postextubation stridor, heliox can be used in addition to racemic epinephrine to reduce the WOB until the airway inflammation subsides. This may help avoid reintubation. In cases in which stridor does not diminish after 24 to 36 hours of therapy designed to reduce upper airway inflammation, the patient may have a more serious problem, such as tracheal webbing.[83]

DEVICES FOR DELIVERING HELIOX IN SPONTANEOUSLY BREATHING PATIENTS

Heliox can be effectively delivered using a mask system, in conjunction with aerosolized bronchodilators, and with invasive and noninvasive mechanical ventilation. To achieve an optimal effect, the concentration of the He generally used is at least 60% to 70%.[83] The most commonly used concentration of heliox is 80:20 (80% He and 20% O_2). This mixture contains the greatest amount of He, and thus the lowest-density gas, without providing subambient levels of O_2 (Key Point 23.10).

***Key Point* 23.10** It is *never* advisable to use a 100% He cylinder in any heliox setup because of the risk that the cylinder might accidentally be used as the only gas source.

Mask Heliox

Heliox can be delivered through a mask system by two different techniques. In the first technique, two flow meters are used. One flow meter is connected to O_2 and one to a heliox cylinder. Tubing from each flow meter is connected to a T-piece that is then attached to the desired mask, usually a nonrebreather. (The one-way valve of the rebreather mask should be in place.) To set the desired F_IO_2, an O_2 analyzer is placed in line at the T-piece to determine the proper setting of each of the two flow meters. The second technique for mask delivery involves connecting the heliox directly to the nonrebreathing mask and using a nasal cannula to administer O_2. A major drawback of this latter case is analysis of the F_IO_2 and the amount of He delivered. Monitoring with pulse oximetry is essential in both cases.

Case Study 23.2

Calculating Gas Flows During Heliox Therapy

A patient is receiving 70:30 He/O_2 through a flow meter. The flow is set at 10 L/min. What would be a close estimate of the actual flow of gas?

One of the more challenging aspects of both mask systems is to determine accurately the actual flow of He delivered. Although the regulators used to connect to the heliox cylinders are specific for the cylinder, the flow meters used for delivery are usually O_2 or airflow meters. Because these flow meters are calibrated to the density of O_2 and not He, the flow displayed on the flow meter will not be accurate for indicating heliox flow. Conversion factors exist for the most common heliox mixtures that can be used to determine the actual liter flow. The factor is multiplied by the actual flow reading. The correction factor for an 80:20 heliox mixture is 1.8. A 70:30 mixture has a factor of 1.6. A 60:40 mixture has a conversion factor of 1.4. For example, if 5 L/min is displayed on the O_2 flow meter attached to an 80:20 heliox mixture, the actual flow will be 9 L/min ($5 \times 1.8 = 9$) (Case Study 23.2).

In many hospitals, respiratory therapists chart the actual flow displayed on the flow meter when recording flows in the patient's chart rather than the corrected flow. The respiratory care staff should be aware of the flow delivery differences. Regardless of whether actual or corrected flow is recorded, the patient's ventilatory and oxygenation status is the most important indicator that determines whether the heliox therapy is effective.[81]

Heliox should not be delivered by nasal cannula alone. Delivering heliox through the nasal cannula produces high flows that can be irritating, cold, and drying and can deliver undetected, inadvertent PEEP. Determining whether the therapy is relieving the patient's hypoxia or meeting minute ventilation needs is also difficult.

Heliox should not be used with tents or hoods. Tents and hoods leak and lose a lot of air. In addition, He is light and will migrate to the top of the device. Because of gas layering, some situations may produce subambient O_2 concentrations (<21%) to the patient.[86,87]

Nasal CPAP for infants may be used with heliox, but the clinician must ensure the CPAP device will work effectively and safely with He and make any necessary adaptations to the therapy. Blenders with CPAP setups that use heliox are not advised. Because of the way the blender functions, it typically has a high gas consumption.[88]

Cost and Gas Consumption During Heliox Therapy

Patients receiving heliox therapy can consume from two to six cylinders in a 24-hour period depending on the device and heliox

concentration. Therefore an adequate quantity of the cylinders should be kept in stock along with appropriate regulators and wrenches. These heliox cylinders typically range in price from $55 to $155 in the United States but can cost upward of $275 per cylinder in Europe.[81]

Heliox and Aerosol Delivery

Aerosol delivery with heliox can be easily achieved with some simple modifications to the mask system previously described. A Y-piece can be placed between the reservoir bag and mask, and then a nebulizer can be attached to the open limb of the Y-piece (Fig. 23.10). The nebulizer can be driven by either heliox or O_2, depending on the patient's F_IO_2 requirement. A similar setup uses two flow meters hooked to one H-cylinder of heliox. One flow meter powers the reservoir bag and mask, and the other powers the nebulizer.

Heliox appears to improve the nebulizer's ability to deliver albuterol, as well as aerosol deposition.[89,90] For patients receiving heliox to reduce the WOB associated with increased R_{aw}, the administration of aerosolized bronchodilators with an MDI may also substantially improve drug delivery.[91,92]

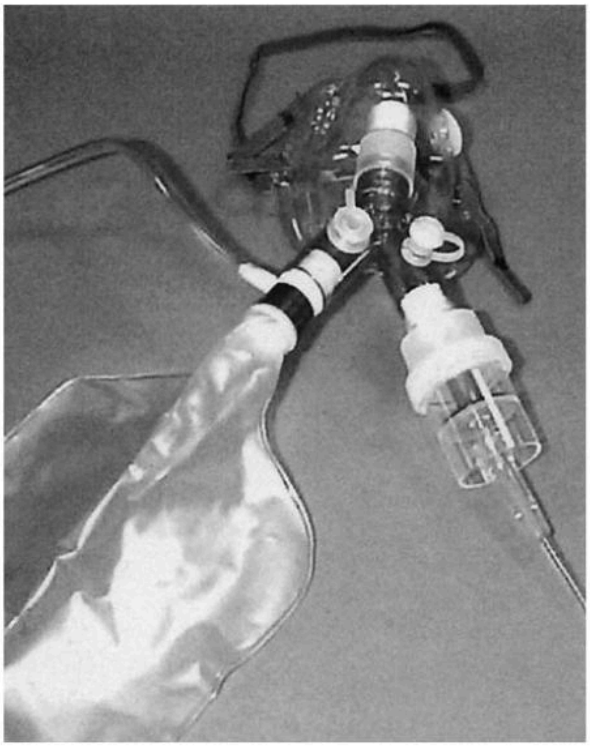

Fig. 23.10 A modified nonrebreathing mask for delivering heliox. A ventilator Y-connector allows inclusion of a standard small-volume nebulizer to provide aerosol therapy without interrupting the heliox delivery. (From Ritz R: Methods to avoid intubation, *Respir Care* 44:686—699, 1999.)

MANUFACTURED HELIOX DELIVERY SYSTEM

In 2004, Datex-Ohmeda, Inc., a division of GE Healthcare (Madison, WI), released the Aptaér Heliox Delivery System for sale in the United States (Fig. 23.11). This device delivers heliox to patients with severe airflow obstruction. It uses premixed heliox gases from a single gas cylinder (60:40—80:20 He/O_2 mixtures). A regulator attaches directly to the cylinder. A second cylinder can be attached to the back of the unit to provide quick changeover when one is depleted.

Fig. 23.11 The Aptaér Heliox Delivery System. (Courtesy Datex-Ohmeda, Inc., GE Healthcare, Madison, Wis.)

Delivery of the gas to the patient is through a coaxial patient circuit, which consists of two tubes, one inside the other. One tube conducts inspired gas (inner tube), and the other expired gas (Fig. 23.12). The circuit is attached to the patient by a sealed face mask.

Medication delivery is provided by the Aeroneb Professional Nebulizer System (Aerogen, Mountain View, CA), which is incorporated into the Aptaér patient circuit (see Fig. 23.12). The nebulizer system is an electronic micropump. It uses a piezoelectric element to vibrate a plate, which contains 1000 funnel-shaped openings. The vibration frequency is 100,000 cycles/s or approximately one tenth the frequency of ultrasonic nebulizers. Movement and vibrational frequency of the plate containing the funnel-shaped openings govern particle size. The Aeroneb consistently produces a mist with a small particle size (2.4-μm mean mass aerodynamic diameter).

The mode of operation of the Aptaér Heliox Delivery System is pressure support. Controls include a support pressure range of 3 to 20 cm H_2O, trigger sensitivity of −0.1 to −1.5 cm H_2O, a rise time adjustment with settings from 1 to 10 (10 being the most rapid rise to the set pressure), and the ability to adjust flow cycle from 5% to 75%. Monitored parameters include respiratory rate (0—122 breaths/min) and an airway pressure waveform display (pressure/time) (Fig. 23.13). Alarms are also available and include a low respiratory rate alarm (2—20 breaths/min), a high respiratory rate alarm (10—99 breaths/min), and a minimum pressure alarm (1—15 cm H_2O).

A dedicated system for providing heliox therapy has several advantages. The equipment needed for therapy is contained in a single cart. The Aptaér system conserves gas, and thus cylinders last longer than typical constant flow cylinder setups. The unit is equipped to provide visual displays for pressure and alarms to

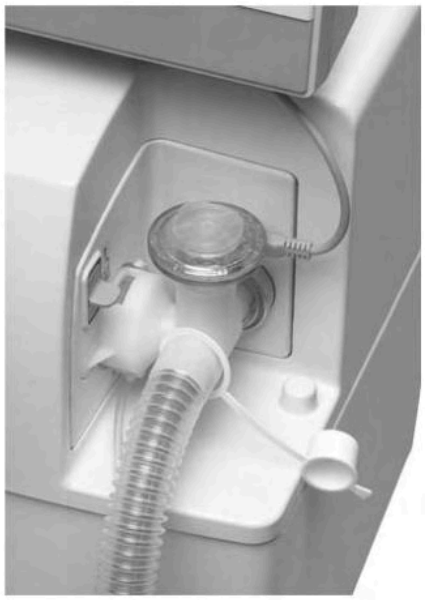

Fig. 23.12 The patient circuit demonstrating a coaxial construction with the inspiratory limb inside the expiratory limb. The Aeroneb Professional Nebulizer System from Aerogen, which is incorporated into the Aptaér Heliox Delivery System, is connected by a cable to the delivery system. (Courtesy Datex-Ohmeda, Inc., GE Healthcare, Madison, Wis.)

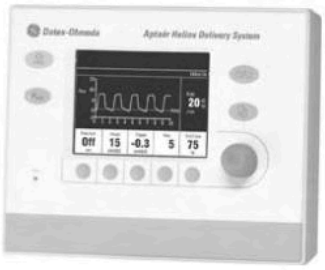

Fig. 23.13 The monitor and control screen of the Aptaér Heliox Delivery System. (Courtesy Datex-Ohmeda, Inc., GE Healthcare, Madison, Wis.)

monitor high and low respiratory rate in addition to low pressure. It also provides gas delivery by using a pressure support mode. Additional studies are needed to determine whether the Aptaér unit provides significant advantages over traditional methods of providing heliox therapy.

HELIOX AND AEROSOL DELIVERY DURING MECHANICAL VENTILATION

If a nebulizer is used for inline delivery of medications during mechanical ventilation with heliox, powering the nebulizer with O_2 and the ventilator with heliox may be advisable. The use of heliox to power the nebulizer may require a high flow and not be as effective in producing an aerosol mist.[87] The heliox in the circuit is sufficient to carry the medication to the patient. During mechanical ventilation, He:O_2 concentrations of 50:50 or more increase delivery of a bronchodilator (albuterol) from an MDI by more than 50%, compared with O_2 or air.[91,92] For this reason aerosolized medications are better delivered by MDI to ventilated patients when heliox is used.

Heliox With a Mechanical Ventilator

Delivering heliox through a ventilator has been found to be effective but presents special challenges. Because of its low density, volume and flow delivery measurements will not be accurate with most ventilators. Calculations and display of any parameter based on a flow measurement will therefore be incorrect. Ventilator values based on flow measurements are less accurate than values based on pressure measurements.[93,94] Accurate measurement of volume with heliox requires a device that is not density dependent, such as a volume displacement spirometer. However, this type of monitoring is not practical.

Delivery of heliox through mechanical ventilators can also interfere with other key functions of the ventilator, such as gas-mixing devices, inspiratory and expiratory valve function, triggering and cycling mechanisms, automatic leak compensation, monitors, and, most notably, minute volume alarms.

During volume ventilation, most ventilators will show a discrepancy between the set V_T ($V_T$set) and the actual delivered V_T ($V_T$del). The magnitude of this difference, in general, is inversely related to the F_IO_2. In other words, the lower the F_IO_2 (i.e., the higher the He percentage), the more $V_T$set exceeds $V_T$del.[94] In most ventilators a linear correlation occurs between $V_T$set and $V_T$del for any given F_IO_2. Because of this, a correction factor may be applied to the $V_T$set to obtain an actual $V_T$del for any set F_IO_2 (Box 23.8).[94-104]

Although no $V_T$set in pressure ventilation exists with heliox, discrepancy with $V_T$del as reported by the ventilator and the actual $V_T$del delivered to the patient does occur. During pressure control in most ventilators, inspired V_T reported by the ventilator ($V_T$insp) will underestimate true $V_T$del by an amount equal to that between $V_T$set and $V_T$del in volume control ventilation.[94] Because pressure ventilation is not based on flow measurements, it may be more suitable for use with mechanical ventilation with heliox. PEEP is also not dependent on flow measurements and is therefore not affected by heliox.[100]

Whatever the technical difficulties, the benefits to patients who require heliox outweigh the problems. Use of heliox reduces PIP in volume ventilation and also helps reduce P_aCO_2. To safeguard against overinflation, P_{plat} must be limited (see the Clinical Scenario on heliox).[105]

Clinical Scenario: Heliox

Case 1

A 14-year-old girl with asthma is admitted to the emergency department and given continuous nebulizer therapy with albuterol (40 mg/h), intravenous corticosteroids, intravenous terbutaline, and an aminophylline infusion to therapeutic levels. The patient is intubated, sedated, paralyzed, and placed on mechanical ventilation. With the F_IO_2 set at 0.70, the ABGs on appropriate ventilator settings are pH = 7.11, P_aCO_2 = 123 mm Hg, and PaO_2 = 68 mm Hg. End-tidal CO_2 is above the upper limit of the scale (>99 mm Hg). Heliox is started with 60% He and 40% O_2 (60:40 mixture). Within minutes the P_aCO_2 drops to 63 mm Hg, PaO_2 rises slightly to 75 mm Hg, and pH returns to the normal range.[106]

BOX 23.8 Ventilator Function With Heliox for Specific Models

CareFusion AVEA *(CareFusion, Viasys Corp, San Diego, CA):* No significant difference in tidal volume (V_T) delivery was detected with volume control, pressure control, or pressure support ventilation, with and without heliox ratios of 80:20 and 60:40. Accuracy of volume delivery was clinically acceptable. At 80:20, V_T delivered was within manufacturer specifications of ±(10% + 0.2 mL) of actual setting. V_T delivered and measured was reasonably accurate. In pressure control mode, PEEP and PIP were within ±1 cm H_2O of actual. Accuracy of F_IO_2 readings was acceptable, at less than 3% variability.[94-96]

Hamilton Veolar and Hamilton Galileo *(Hamilton Medical, Bonaduz, Switzerland):* $V_T$del was consistently higher than $V_T$set. The lower the F_IO_2 (the higher the helium [He]), the more the $V_T$del exceeded the $V_T$set. V_{Texh} underestimated actual V_T.[94,97]

Hamilton G-5: With the Heliox option, the Hamilton G-5 delivers a specific set V_T and displays accurate volumes for inhaled and exhaled volume delivery. It can only be used with an 80:20 mixture of He/O_2.

Puritan Bennett 840/980 *(Medtronics, Minneapolis, MN):* Will not cycle at all (i.e., heliox cannot be used with the 840). The ventilator recognizes a low-density gas when heliox is connected to the air connect and alerts the operator. This helps protect the patient from inappropriate gases being used, but it also eliminates the possibility of using this ventilator for heliox therapy.[94,97,100]

Servo-u *(Getinge, Göteborg, Sweden):* Cycled consistently with all heliox mixtures tested in volume control and pressure control ventilation. The O_2 analyzer was within ±3%, and the blender was within ±1%. Set V_T factor (0.95) was consistent for all heliox concentrations tested. V_{Tinsp} was within acceptable limits. The ventilator uses ultrasonic technology to measure expiratory gas flows. Exhaled V_T display was erratic at 80:20 and 70:30 (He/O_2) but consistent at 60:40 and 50:50. V_{Texp} read lower than actual delivered V_T. As the He concentration decreased, the accuracy recovered.

Correction factors can be used to determine $V_T$del by using $V_T$set. For example, $V_T$del = $V_T$set multiplied by the factor (factor for 21%, 30%, 40%, and 50% O_2 is 0.95). Correction factors can only be used to determine $V_T$del by using V_{Texh} for high F_IO_2 values (40% and 50%). For example, $V_T$del = V_{Texh} × 1.37 (for 40% O_2). At high F_IO_2 values (low He percentage), no correction factor is available. With high He concentrations at a set V_T of 500 mL, the low $\dot{V}_E$ alarm was breeched and could not be disabled.[98-100]

Servo-i without heliox option: Cycled consistently with all heliox mixtures tested in volume-controlled and pressure-controlled ventilation. The O_2 analyzer was within ±3%, and the blender was within ±1%. Set V_T factor (0.95) was consistent for all heliox concentrations tested. V_{Tinsp} was within acceptable limits. The ventilator uses ultrasonic technology to measure expiratory gas flows. Exhaled V_T display was erratic at 80:20 and 70:30 (He/O_2) but consistent at 60:40 and 50:50. V_{Texp} read lower than actual delivered V_T. As the He concentration decreased, the accuracy recovered.

Correction factors can be used to determine $V_T$del by using $V_T$set. For example, $V_T$del = $V_T$set multiplied by the factor (factor for 21%, 30%, 40%, and 50% O_2 is 0.95). Correction factors can be used only to determine $V_T$del by using $V_T$exh for high F_IO_2 values (40% and 50%). For example, $V_T$del = $V_T$exh × 1.37 (for 40% O_2). At high F_IO_2 values (low He percentage), no correction factor is available. With high He concentrations at a set V_T of 500 mL, the low alarm was breeched and could not be disabled.[98-100]

Servo-i, Servo-u with heliox option: With the Heliox option, the Servo ventilators deliver a specific set V_T and display accurate volumes for inhaled and exhaled volume delivery. It can only be used with an 80:20 mixture of He/O_2.[103-104]

Dräger Dura-2 *(Dräger Medical, Inc. Telford, PA):* $V_T$del was greater than $V_T$set. V_{Texp} read higher than actual delivery. A correction factor is available to determine actual volume delivery during VC-CMV with 70:30 (He/O_2) and varying O_2 settings (21%, 30%, 35%). At the heliox concentration with a set F_IO_2 of 0.21, for example, the correction factor is 1.38. Divide the desired V_T by 1.38 to get the V_T value to set. The flow sensor and the O_2 analyzer must be turned off. The expiratory flow monitoring malfunction alarm is activated if it is not turned off. (Autoflow was turned off in this study.)[102] In the Dura-2, the higher the He delivered, the lower is the actual delivered F_IO_2 compared with the F_IO_2 set.

Dräger E-4 *(Dräger Medical, Inc., Telford, PA):* $V_T$del was higher than $V_T$set, and this difference was linear; with $V_T$set higher than 500 mL, the relation between set and delivered was nonlinear. This problem was alleviated by inactivating the leak compensation mechanism. The ventilator gives a high-priority inoperative alarm, which cannot be silenced, but it requires inactivation of the inspiratory flow monitor. The flow sensor must be removed to accomplish ventilation with heliox. $V_T$del was consistently higher than $V_T$set so that a correction factor could be calculated from $V_T$del average. In the Evita-4, the higher the He delivered, the lower is the delivered F_IO_2 compared with the F_IO_2 set.[94,97]

eVent Medical Inspiration Mechanical Ventilator *(eVent Medical, Inc., San Clemente, CA):* Cycled consistently in volume control and pressure control ventilation. The O_2 analyzer within ±1% and the blender was within ±5%. Conversion factors are available.[101]

Case 2

One day postoperatively, a 10-year-old boy is extubated and exhibits severe stridor and respiratory distress. A nebulizer treatment with racemic epinephrine fails to relieve the stridor. The child is started on dexamethasone (Decadron), but it typically takes approximately 4 hours or more for it to become effective. Rather than reintubate the child, heliox is started (80:20 mixture). The stridor is immediately relieved. Five hours later, the stridor resolves apparently because of the steroid administration and heliox is discontinued.[106]

Technical Considerations in Heliox Delivery

In many cases the expiratory flow–sensing devices must be disconnected or disabled when heliox is used during mechanical ventilation. A clinician must always be aware of how the ventilator will function with heliox before it is used for ventilating a patient.

Delivery of heliox with mechanical ventilation is achieved by connecting the compressed air inlet hose directly to a 50-psi, 80:20 heliox gas source. In preparation for using heliox with a ventilator, two cylinders are recommended, preferably yoked together so that when one is empty, the respiratory therapist can quickly switch to the second cylinder. Cylinder trolleys, appropriate heliox

Key Point 23.11 It is absolutely essential that the label on the cylinder be checked for content accuracy. It must read that the cylinder contains medical-grade (U.S.P. [United States Pharmacopeia]) He and O_2. The color code for heliox cylinders is typically brown and green, but do not trust the color of the cylinder.

regulators, and wrenches should also be on hand.[87] Early version ICU ventilators consumed approximately four to six cylinders a day. Newer ICU ventilators, such as the Servo-u and the Hamilton G-5, conserve a greater amount of gas and may use only two to three cylinders a day.[103,104] The clinician must be sure that a sufficient number of cylinders are available (Key Point 23.11). The minimum concentration of heliox recommended for use is 80:20. As previously mentioned, a 100% He cylinder should never be connected to a ventilator because of the risk for asphyxia.

Any heated-wire flow-measuring device is likely to alarm, so a ventilator with a heated-wire flow monitor should not be used or the monitoring device should be disconnected. For example, the Dräger Evita XL uses a heated-wire device for measuring exhaled V_T. Failure to disconnect this device will result in continuous alarm activation. The Evita XL also has leak compensation, which should be turned off for the same reason.

As with normal ventilation, the gas should be heated and humidified. A heated-wire circuit should be used with the humidification system because of the high thermal conductivity of He. (NOTE: The circuit may need to be wrapped in a plastic sleeve to help retain heat and avoid activating the temperature alarms on the humidifier.)

Other potential problems must be considered. For example, an O_2 analyzer must be used to determine actual O_2 delivery during mechanical ventilation with heliox, because the setting displayed on the front panel may not be accurate. In some ventilators that have been studied for this function, the actual delivered F_IO_2 is approximately the same as the set F_IO_2 (e.g., Hamilton G-5, Servo-u). The F_IO_2 delivered may also vary from the set value as the He concentration changes in some ventilators (e.g., Dräger E-4), resulting in activation of the ventilator's internal O_2 analyzer alarm. These can be disabled or disconnected. If they are, some other form of O_2 monitoring should be provided, such as an O_2 analyzer equipped with an alarm. Activation of the O_2 alarm is often what alerts the respiratory therapist to a technical problem.

Pressure during ventilation is not a problem because pressure is not density dependent. Thus pressure ventilation is easily accomplished. In pressure control ventilation with a variety of ventilators (see Box 23.8), $V_T$del is equal to V_T measured for a range of pressures and F_IO_2 values.[94]

As previously mentioned, heliox has a high thermal conductivity, which can affect the accuracy of the hot wire sensor. With hot wire flow monitoring, the higher the He percentage, the greater the flow is overestimated. The Dräger E-4 uses hot wire flow measuring, but it should be used with caution (see Box 23.8).

Another example of the effect of ventilator valve function on effective ventilation with heliox involves ventilators that use variable orifice pneumotach (e.g., Hamilton G-5). The pneumotach is located at the ventilator's Y-connector and measures both inspiratory and expiratory flows. The variable orifice pneumotach relies on turbulent flow for accuracy. Because high He concentrations result in laminar flow, this phenomenon lowers the resistance through the device and reduces the ΔP across the device, causing the variable orifice pneumotach to underestimate the true flow.[94]

Because the difference between $V_T$exp and $V_T$del (see Box 23.8) is usually linear and is a function of F_IO_2, correction factors can be used to determine actual $V_T$del.[94] In some of the ventilators studied, the lower the F_IO_2 (the higher the He percentage), the larger the discrepancy between the reported $V_T$exp on the ventilator and the actual $V_T$del. With other ventilators this relation is nonlinear and the displayed $V_T$exp is much higher than the actual $V_T$del. At these high He concentrations, a correction factor is not available.

Only ventilators that can provide safe breath delivery should be used for heliox therapy in patients on ventilation. The practitioner must keep in mind that changes can be made to ventilator function at any time by the manufacturer. These changes may affect ventilator performance differently during heliox delivery.

Heliox and NIV

NIV with heliox has been used with both critical care and portable ventilators.[94] When heliox is used with some portable ventilators, ventilators titrated with heliox may exhibit erratic triggering and cycling, although this finding may not be clinically significant. Concentrations up to 60% He can be delivered. Gas consumption may be high. For example, a cylinder containing 4500 L of heliox being used at a flow of 18 L/min will last approximately 4 hours. With continuous therapy, this would result in the use of six cylinders in 24 hours, resulting in a cost of about $500 per day.[86] This cost may be offset by a reduction in ventilator days, days in the ICU, and overall hospital costs.

Despite the fact that the benefit is modest, NIV with heliox has been reported to be beneficial for patients with COPD.[107,108] Furthermore, although the benefit of heliox is seen more often in acute upper and central airway obstruction, such as in acute asthma, the ability of heliox to alleviate air trapping and unload the muscles is believed to benefit patients with COPD. This may in turn significantly reduce the length of hospital stay for this patient group. However, combining heliox and NIV is technically challenging. Importantly, the use of NIV for the treatment of acute asthma has not been well documented.[109]

Safe use of heliox during management of patients with airflow obstruction is possible if the physical properties (low density, high thermal conductivity) are kept in mind. These factors will influence WOB, aerosol delivery, and mechanical ventilator function. F_IO_2 and V_T delivery may be altered during heliox delivery with a mechanical ventilator. This is particularly true when hot wire technology is part of the ventilator's gas delivery or monitoring system. In most ventilators, the set V_T and delivered V_T parallel each other, and a correction factor can be used to determine the desired V_T setting. Exhaled V_T displayed may be higher or lower than the $V_T$del depending on the type of ventilator. Practitioners should study the ventilators they plan to use for heliox therapy and carefully determine equipment and cylinder requirements before using this therapy with a patient.

MONITORING THE ELECTRICAL ACTIVITY OF THE DIAPHRAGM AND NEURALLY ADJUSTED VENTILATORY ASSIST

For decades clinicians have used electrocardiograms (ECGs) to monitor the electrical activity of the heart. Indeed, all patients in

an ICU have ECG monitors with alarms in place to alert personnel of dangerous cardiac events. In contrast, clinicians have only recently gained the ability to monitor a patient's neural respiratory drive at the bedside.

A new method has been introduced to clinically monitor neural control of respiration through measurements of **electrical activity of the diaphragm (Edi)**. The principle governing this new technique is that depolarization of the diaphragm depends on the transmission of a neural signal from the brainstem to the diaphragm. Edi measurements can therefore be used to evaluate a patient's neural control of spontaneous breathing.

REVIEW OF NEURAL CONTROL OF VENTILATION

The respiratory controller governs a person's drive to breathe. Afferent information from the peripheral and central chemoreceptors, along with neural stretch receptors and volume receptors, and information from the prefrontal cortex of the brain are integrated and processed by the respiratory center located within the brainstem. The resulting efferent neural signal is propagated through the phrenic nerve to the diaphragm. The diaphragm then depolarizes and contracts. The greater the intensity of the neural stimulation and the greater the number of nerve fibers involved, the stronger is the electrical activity measured on the muscle. In healthy individuals, the greater the neural stimulation to the diaphragm, the greater is the muscle's electrical activity, and the greater is the diaphragm's contractile strength. This is not necessarily true in a patient with pulmonary hyperinflation, in which the Edi may increase but the strength of contraction does not increase proportionately.[110]

Previously, the only way to assess diaphragmatic activity in the clinical setting was to use an esophageal balloon to measure esophageal pressure (see Chapter 10). Although measurement of esophageal pressure (P_{es}) can provide measurements of thoracic pressure changes associated with diaphragmatic movement, its use is not without problems. For example, the accuracy of thoracic pressure measurements can be adversely affected by inappropriate positioning of the catheter balloon within the thorax, inaccurate volume filling of the catheter balloon, and patient positioning.[111,112] Consequently, a complete in vivo assessment of diaphragm function in human subjects has remained unavailable.

Without the ability to monitor the diaphragm function, it is not possible to fully address a number of the problems associated with patients on ventilation. For example, does the patient demonstrate any diaphragmatic activity, or is the patient too heavily sedated or overventilated to breathe spontaneously? Is the diaphragm activated in synchrony with the triggering and cycling of the ventilator, or is patient-ventilator asynchrony present? It is believed that having the ability to monitor a patient's diaphragmatic activity at the bedside could provide clinicians with valuable information about the patient's central drive to breathe.

DIAPHRAGM ELECTRICAL ACTIVITY MONITORING

History of Diaphragm Electrical Activity Monitoring

During the past 50 years, groundbreaking research has resulted in the introduction of a technique that allowed scientists to study the function of the diaphragmatic muscle in humans by means of electromyography.[110] Early laboratory studies in the 1960s showed a correlation between the activity of the phrenic nerve and the electrical activity of the diaphragm.[113] Although these researchers paved the way, it would take several more decades of clinical investigation before the ability to monitor the diaphragm's electrical activity in the clinical setting was possible.[114,115]

The Edi Catheter: Its Characteristics and Placement

In 1998, Sinderby and Beck developed a catheter (Edi catheter) that allows measurements and monitoring of the diaphragm's electrical activity in the clinical setting. The catheter is basically a nasogastric tube with miniaturized electrodes near the distal tip* (Fig. 23.14).

The Edi catheter is inserted into the esophagus through the oral route. The electrodes are positioned within the esophagus at a level that is adjacent to the diaphragm (crucial muscle of the diaphragm). This allows the electrodes to detect an Edi (or EAdi) signal. (NOTE: The distal tip itself resides in the stomach. Thus the catheter can also be used for feeding the patient or administering medications, just as a regular nasogastric [NG] or orogastric [OG] tube.)

The Edi catheter is made of medical-grade polyurethane. A barium strip is embedded along the length of the catheter for radiographic identification, although a chest radiograph is not essential for placement accuracy.[116] The Edi catheter contains a total of 10 stainless steel bipolar electrodes. The spacing between electrodes varies on the basis of the size and length of the catheter.

A variety of catheter sizes are available for different patient sizes (premature infant to adult sizes) (Table 23.2). The Edi catheter is inserted in a manner similar to any NG or OG tube. Once placed, the catheter can be connected to the ventilator with a cable. As Fig. 23.14 illustrates, the cable connects the Edi catheter electrodes to a module located in a side slot of the Servo-I, Servo-u, and Servo-n ventilators.

Several procedures can be used to establish that the catheter is properly positioned within the esophagus and stomach. First, the centimeter marking at the nose or mouth will coincide with a manufacturer-recommended calculation used to determine the appropriate depth of insertion of the catheter. Second, the clinician can listen over the epigastric area with a stethoscope while injecting air though the catheter feeding port. Hearing gas movement helps confirm the location of the catheter tip in the stomach. Third, there is a catheter positioning screen on the ventilator that helps establish the exact location of the electrodes within the esophagus and stomach. Fourth, the clinician can confirm the correct catheter position by performing an expiratory pause (end-expiratory occlusion) maneuver. When the patient attempts to inhale against the closed system during the pause, the Edi signal is positive (increases) and the airway pressure waveform decreases. (Additional information on these waveforms will be presented later in this section.) Fifth, a CO_2 detector can be used at the proximal end of the catheter to monitor the presence of CO_2. The presence of elevated levels of CO_2 would suggest that the catheter is in the trachea and not the

*The Edi catheter and the ability to measure the Edi is exclusively available through the Servo-i, Servo-u, and Servo-n ventilators; (Getinge, Göteborg, Sweden) uses the abbreviation Edi, rather than EAdi, which is the abbreviation that appears in the medical literature.

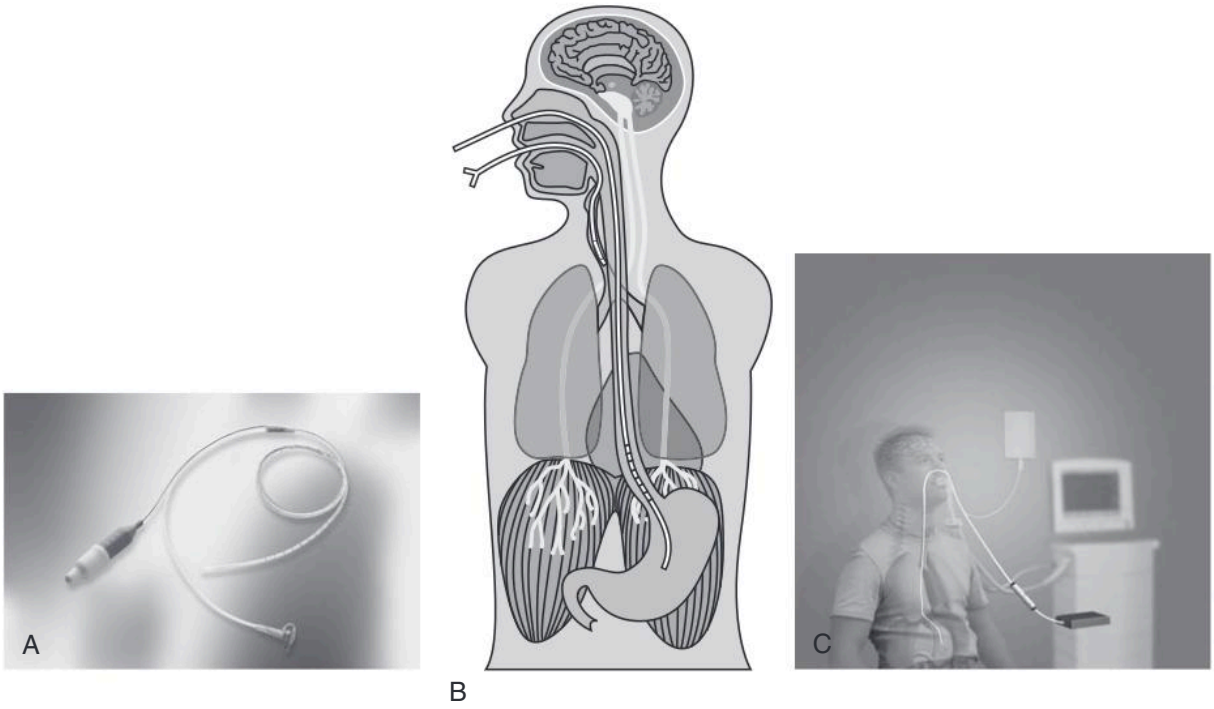

Fig. 23.14 (A) Example of an Edi catheter. (B) The catheter in the esophagus and stomach. Notice the electrodes span a certain distance so that some electrodes are located above the diaphragm and others below the diaphragm. (C) The electrical connector from the catheter attaches to a cable connecting the catheter to the ventilator *(image on bottom)*. (*A* and *C*, Courtesy Maquet, Inc., Wayne, N.J. *B,* Courtesy Dr. C Sinderby, St-Michael's Hospital, Toronto, Canada.)

stomach. Finally, a chest radiograph can be performed if hospital policy required it. Once the catheter is in place, the Edi waveform is displayed (Fig. 23.15).

TABLE 23.2 Edi Catheter Specifications

EDI CATHETER GUIDE FOR SELECTING CORRECT CATHETER BASED ON PATIENT SIZE					
Patient height (cm)	<55	<55	45–85	76–160	>140
Patient weight (kg)	0.5–1.5	1.0–2.0	NA	NA	NA
French/cm	6/49	6/50	8/100	12/125	16/125 8/125

NA, Not applicable.

The Edi waveform is basically sinusoidal in appearance. A digital display of the maximum Edi measured (Edi peak) and minimum Edi (Edi min) is provided on the right side of the screen. Values are reported in microvolts (μV).

The resting Edi peak for a healthy individual ranges from a few μV to about 10 μV. An Edi min is typically close to 0 μV. These values vary depending on the individual. The Edi μV measurements are small values compared with the heart's electrical activity, which has an electrical amplitude 10 to 100 times that of the diaphragm.[110] The Edi will increase in normal individuals, for example, when the diaphragm is required to work harder, which occurs when an individual exercises.[117] Individuals with chronic respiratory insufficiency, such as COPD, commonly have an Edi signal 5 to 7 times stronger than that of a normal individual.[118-119]

Occasionally a catheter will be placed in the correct position, but there is no measurable Edi waveform or signal. An Edi signal may not be detectable on a mechanically ventilated patient for a number of reasons. For example, there may be no central respiratory drive because of sedation, hyperventilation, or brain injury.

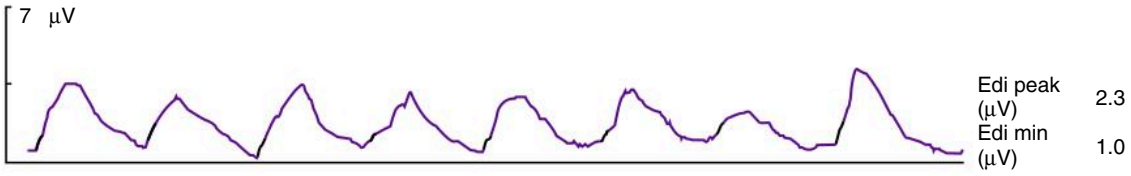

Fig. 23.15 An Edi waveform. Note the Edi peak (2.3 μV) and Edi min (1.0 μV) displayed at the lower right corner of the screen. (See text for additional information.)

An absent Edi may also have an anatomical reason, such as a diaphragm hernia or a severed phrenic nerve (e.g., postthoracic surgery). Even a conduction failure can result in an absent Edi, when the impulse is not conducted from the phrenic nerve to the diaphragm as a result of use of a paralytic agent, or a disease is present that blocks electrical impulse conduction.[120] Interestingly, Bordessoule and colleagues proposed that the Edi catheter can be used to evaluate recovery from paralysis.[121]

It should be apparent from this discussion that the ability to monitor the Edi can be a valuable tool to assess ventilatory function in critically ill patients. As you will see in the following section, Edi can also be used to detect the presence of patient-ventilator asynchrony.

Detecting Patient-Ventilator Asynchrony Using the Edi Catheter

As discussed in Chapter 18, patient-ventilator asynchrony is a common problem among patients receiving mechanical ventilation. It has been estimated that 25% of patients on mechanical ventilation exhibit asynchrony, which can increase the length of time that a patient requires ventilatory support.[121-124]

Patient-ventilator asynchrony can lead to a number of complications. When a patient "fights" the ventilator, this can potentially result in damage to the diaphragm (ventilator-induced diaphragm dysfunction). This loss of diaphragmatic force-generating capacity is specifically related to the use of mechanical ventilation.[125] It is a common practice among many clinicians in the ICU to sedate the patient when asynchrony is present. However, sedation can significantly reduce a patient's spontaneous breathing efforts and therefore diaphragm activity. Lack of use of the diaphragm during mechanical ventilation can lead to severe diaphragm atrophy, which can occur in as short a time as 18 to 69 hours.[125,126]

Another example of asynchrony is referred to as *double triggering*. The etiology of double triggering was unknown, and many practitioners thought the ventilator was unable to provide the flow demand required by the patient during a breath. By using two techniques to evaluate diaphragm activity during double triggering, it is possible to determine what the patient needs versus what the ventilator is delivering (Fig. 23.16).[127,128]

The phenomenon of double trigger can occur regardless of the type of ventilator being used.[127,128] By looking at Fig. 23.16 and using either an esophageal balloon or the Edi catheter to monitor

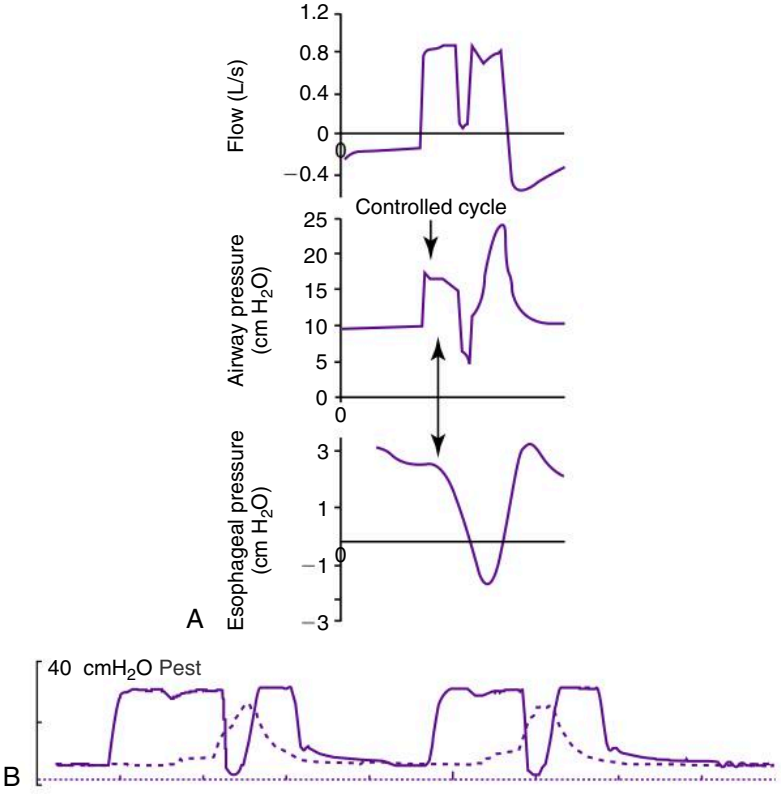

Fig. 23.16 (A) The flow, pressure, and esophageal pressure curves of a patient on mechanical ventilation showing a double trigger, two consecutive breath cycles separated by a short expiratory time. Note the patient's effort indicated by the decrease in esophageal pressure. The patient effort occurs after a mandatory breath has started and continues past the end of this breath. The pressure and flow curves decrease in apparent response to a patient effort. This seems to result in a second mandatory breath. (B) The pressure-time curve shows double-triggered breath *(solid line)*. The Edi waveform *(dashed line demonstrates what the patient actually wants.)* The patient's neural effort starts much later than the mandatory breath provided by the ventilator. The ventilator's mandatory breath seems to end. There is a short expiratory time indicated by the pressure-time curve, but the patient's diaphragm has only begun to taper off. The ventilator has a delayed response to the patient's effort and attempts to give another breath, while the patient is trying to exhale. (See text for additional information.) *(A,* Redrawn from Thille AW, Brochard L: Double triggering during assisted mechanical ventilation: is it a controlled, auto-triggered or patient-triggered cycle? *Intensive Care Med* 33:744—745, 2007. *B,* Redrawn from screen capture provided by Daniel D. Rowley, RRT-NPS, RPFT, FAARC, Pulmonary Diagnostic & Respiratory Therapy Services, University of Virginia Health System; Charlottesville, Va.)

diaphragm activity, it becomes apparent that the patient is making an inspiratory effort while a mandatory breath delivery is already in progress. The ventilator is simply not coordinated with the activity of the patient's diaphragm and respiratory center. (As mentioned earlier, a common practice in these situations is to use sedation as a means of stopping the patient from "fighting" the ventilator. However, it is important to recognize that sedation can prolong mechanical ventilation with the potential for many of its associated complications.[129])

Using the Edi Waveform to Interpret Ventilator Synchrony

It is now possible to monitor ventilator graphics and measurements of Edi activity at the same time. This can help the clinician determine how well the set mode and parameters are in sync with the patient's drive to breathe. Fig. 23.17 shows an example of the Edi compared with the pressure-time graphic during VC-CMV. The figure illustrates another example of patient-ventilator asynchrony. Notice that the ventilator breath delivery is not in synchrony with the Edi waveform. The ventilator is completely out of synchrony with the patient in terms of both when the breath starts (trigger) and when it ends (cycle).

Fig. 23.18 shows an example of a patient receiving pressure support/CPAP mode on the Servo-i, which is purely a patient-triggered mode. Notice how low the Edi Peak is in this figure. One can see that the patient is triggering the breath by observing the pressure, flow, and volume curves. Remember that with PSV, all breaths are patient triggered. However, there is no Edi waveform because the diaphragm is not depolarizing and contracting. How is this possible? There are two possible explanations. First, the patient may be using accessory muscles to trigger the ventilator. A physical assessment of the patient would determine whether this is true. Second, the ventilator could be autotriggering, which the therapist could also evaluate. In this case the patient was actually using accessory muscles to breathe, as noted by his physical examination.

Notice the ventilator settings in the example shown in Fig. 23.18 as follows: pressure support above PEEP is 11 cm H_2O, PEEP is 5 cm H_2O, and the F_IO_2 is set at 28%. The patient's respiratory rate is about 24 breaths/min and V_T is about 300 mL. It would appear that this patient is ready to have the level of support reduced as he is progressing toward weaning. However, when a spontaneous breathing trial was attempted, the patient failed. Under normal circumstances, we might not know why he failed. However, having the Edi signal information, it is possible to determine that the diaphragm is not active. It may be necessary, in this case, to further reduce the support level to stimulate the patient to use his diaphragm so that he can be successfully weaned.

Other types of asynchrony that may be evaluated include wasted efforts, where the patient wants to take a breath but does not receive one from the ventilator; mismatching of the neural rate with the ventilator delivery rate; and fixed levels of ventilator assist,

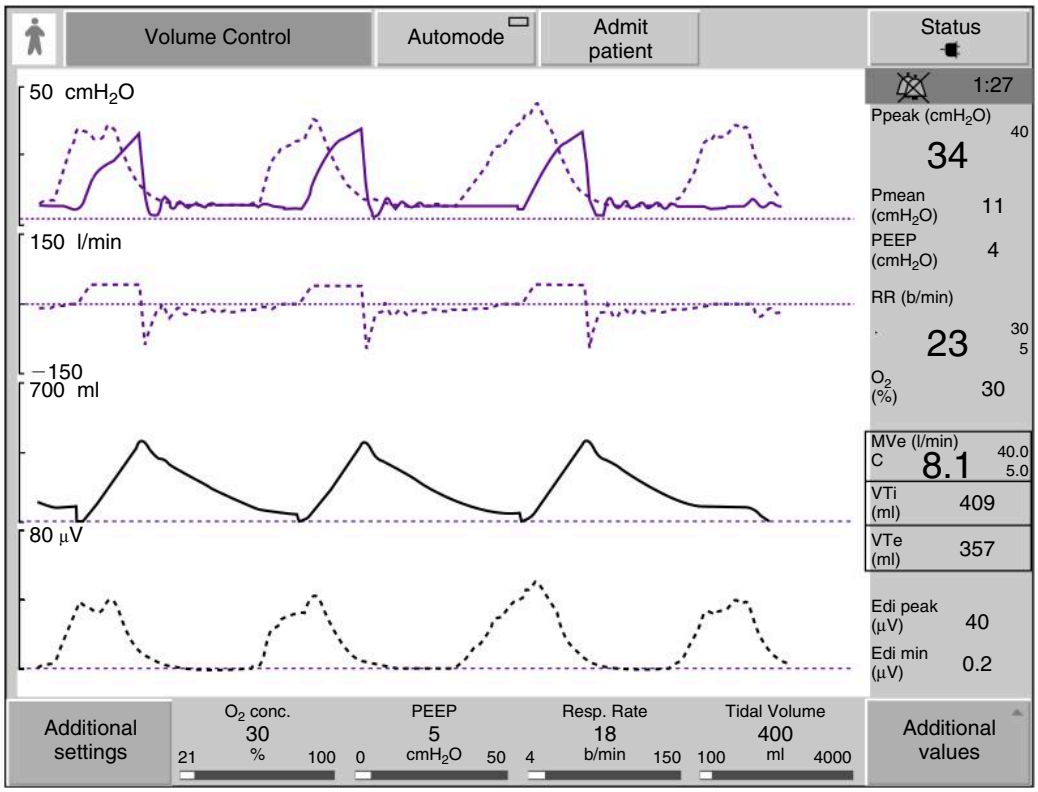

Fig. 23.17 Main screen on the Servo-i ventilator showing VC-CMV *(called "volume control")* waveforms. The top curve is the pressure-time curve *(solid line)* graph along with the Edi waveform. Note this shadow or ghost Edi waveform is identical in shape to the Edi—time waveform *(dashed line)* at the bottom of the screen. The second waveform is flow-time, the third volume-time, and the fourth the Edi waveform. Notice the lack of synchrony between what the patient wants *(Edi signal)* and what the ventilator delivers as shown in the pressure-time curve. (See text for additional information.) (Screen capture provided by Daniel D. Rowley, RRT-NPS, RPFT, FAARC, University of Virginia Health System, Charlottesville, Va.)

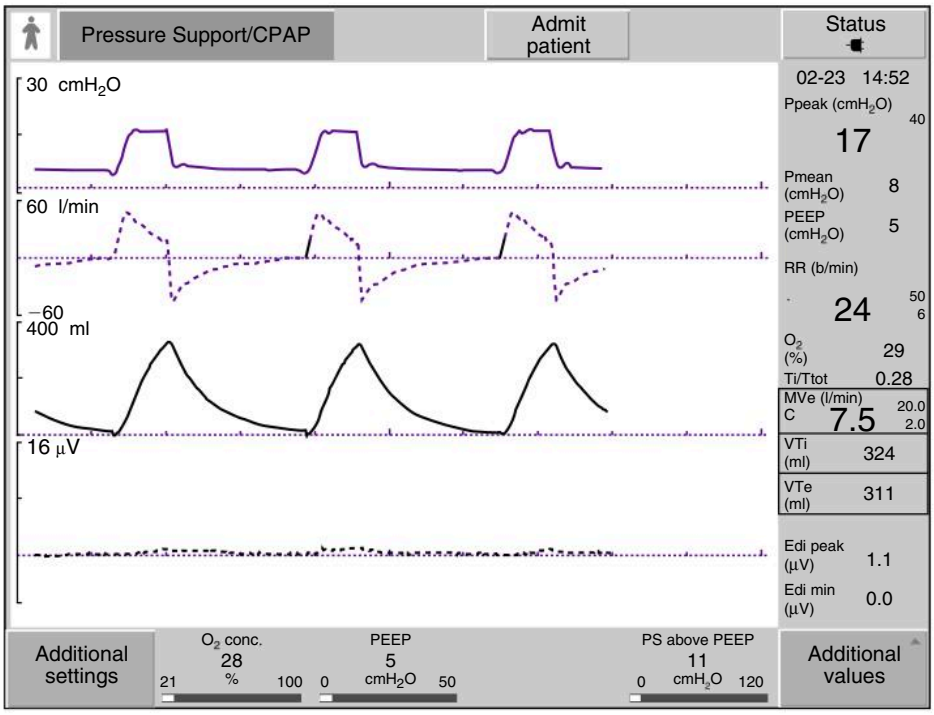

Fig. 23.18 Main screen on the Servo-i ventilator during pressure support/CPAP mode showing pressure, flow, volume, and Edi waveforms, from *top* to *bottom*. Note the Edi value is nearly zero. (See text for additional information.) (Screen capture provided by Daniel D. Rowley, RRT-NPS, RPFT, FAARC, University of Virginia Health System, Charlottesville, Va.)

such as a set pressure in PSV, where the level of assist does not correspond to the variable drive of the patient.

NEURALLY ADJUSTED VENTILATORY ASSIST

At this point, it would be reasonable to pose the question if you have the ability to monitor the Edi, why not use that signal to control the mechanical ventilator? The high incidence of patient-ventilator asynchrony described earlier suggests that current ventilator modes and controls do not always provide the best outcomes for our patients. We currently depend on the ventilator to pneumatically measure a patient effort, using flow or pressure signals, to trigger a breath. The patient must rely on the operator's judgment to set an appropriate V_T, pressure, flow, and T_I.

The motivation for the development of NAVA was to allow the spontaneously breathing patient to have greater control of his or her V_T, T_I, and flow. In 2007 Maquet received approval from the FDA to include NAVA as a mode of ventilation on the Servo-i ventilator. NAVA uses the Edi, a reflection of the neural respiratory output to the diaphragm, to control triggering, breath delivery, and cycling of the ventilator.[110] A comparable mode available on the Puritan Bennett 840/980 is proportional assist ventilation (PAV), which was described in Chapter 5. Both NAVA and PAV provide breath delivery in proportion to the patient's demand.[130] The primary difference is that NAVA uses an electrical signal, the Edi, whereas PAV uses a pneumatic signal to deliver the next breath.

Whereas the Edi catheter can be used in most ventilated patients to facilitate monitoring of the diaphragm, the NAVA mode is specifically for use in patients who are capable of spontaneous breathing, that is, patients who have an Edi signal.[110] Patients who demonstrate the following conditions would be excluded from the use of the NAVA mode:

- Heavily sedated and/or paralyzed
- Damaged brain center
- Absence of phrenic nerve activity
- Diseases that prohibit neuromuscular transmission
- Presence of apnea (NOTE: Some premature infants may experience periods of apnea, but because NAVA has a backup mode, this does not necessarily exclude them from using this technique.)

Using NAVA Ventilation

The following section describes how the NAVA mode can be set up by the operator. Once the patient has an Edi catheter in place, as described earlier, it is connected to the ventilator by means of a cable that leads to a module in the side of the ventilator (see Fig. 23.14). The operator then selects the NAVA mode for ventilating the patient. Triggering of a breath occurs when a 0.5-µV deflection occurs in the Edi waveform below the Edi min of the previous breath. It has been shown that complete trigger synchrony is achieved with NAVA, compared with other modes.[131]

Delivery of pressure during inspiration is based on the strength of the Edi signal and the level of NAVA support ("NAVA level") set by the operator. The NAVA level determines the amount of pressure delivered by the ventilator in proportion to the Edi. The easiest way to think about it is that the pressure delivery is similar to pressure support. The higher the numerical value of the NAVA level (0–15 cm H_2O/µV), the greater the support provided by the ventilator. The pressure varies constantly during breath delivery as the Edi varies (i.e., it varies in proportion to changes in the Edi and

the neural demand for a breath).[132,133] An estimated peak pressure that will be delivered during the breath is based on the following equation[134]:

$$NAVAP_{peak} = \text{NAVA level} \times (\text{Edi peak} - \text{Edi min}) + \text{PEEP}$$

For example, if the NAVA level is set at 1 cm $H_2O/\mu V$, the Edi peak is 10 μV, the Edi min is 0 μV, and the PEEP is 5 cm H_2O, the estimated P_{peak} delivered for this particular NAVA-supported breath will be equal to 1 cm $H_2O/\mu V \times (10\ \mu V - 0\ \mu V) + 5$ cm $H_2O = 15$ cm H_2O. Because the Edi varies throughout inspiration, the amount of pressure delivered will also vary throughout inspiration. The Edi signal is measured 60 times per second, so ventilator response is rapid (Fig. 23.19).

The ventilator provides a "NAVA Preview" tool that allows the operator to estimate an appropriate NAVA level based on the patient's current mode of ventilation. For example, if the patient has acceptable blood gas results on a specific mode such as VC-CMV, the "NAVA Preview" helps the operator set a NAVA level that will be comparable with the current pressure and volume deliveries.

What if the NAVA level was accidentally set at 10 cm $H_2O/\mu V$? What would be the estimated P_{peak}? Using the same values from the previous example, the Edi peak is 10 μV, the Edi min is 0 μV, and the PEEP is 5 cm H_2O. Tthe estimated P_{peak} delivered for this particular NAVA supported breath would theoretically be equal to 10 cm $H_2O/\mu V \times (10\ \mu V - 0\ \mu V) + 5$ cm $H_2O = 105$ cm H_2O! However, what happens in a case to avoid this high pressure is first the ventilator cycles and starts filling the lungs. As the lungs fill with air, the stretch receptors in the lungs send signals by way of the vagus nerve to the respiratory centers of the brain. The more the receptors are stretched, the more the nerve is stimulated and the extent of the breath is inhibited (Hering-Breuer Reflex).[135] The brain then sends a signal to stop neural transmission through the phrenic nerve to the diaphragm, resulting in the cessation of diaphragmatic activity. The respiratory system is therefore self-protective and the patient would not receive 105 cm H_2O. The process is extremely rapid. Additionally, there is the usual protection from the set peak pressure limit. But the pressure delivery stops long before the upper pressure limit would be reached. It is important to note that this protective response is only for patients with intact vagal reflexes and respiratory centers. Some patients may not have an intact vagal response. For example, post–lung transplant patients do not have an intact vagal reflex from the lungs. Also, in some patients who require a high CO_2 stimulation to breathe, or if the respiratory center has been impaired (injury or medications), these protective reflexes may be overridden. (In these instances, the Edi catheter may still be of benefit in monitoring the neural activity.)

Inspiration during a NAVA breath ends or cycles when the Edi decreases to 70% of the Edi peak. This represents a time when the Edi is in the process of decreasing to the Edi min (baseline) and the patient's effort is ending.

Alarms and Safety Features in NAVA

When a patient is on ventilation in the NAVA mode, a variety of safety features are available. The clinician can set two different

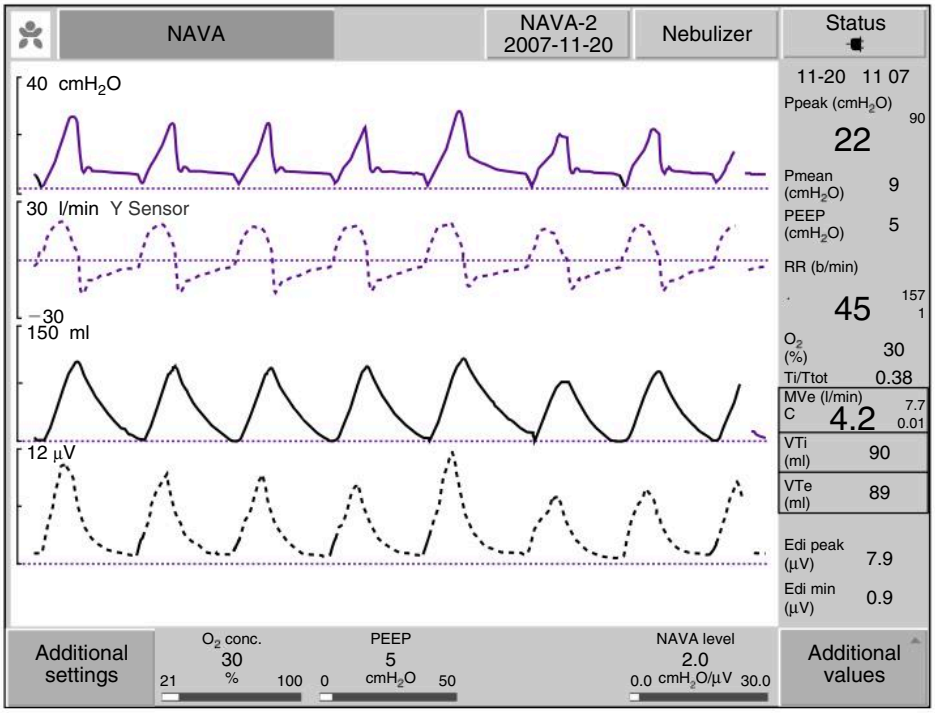

Fig. 23.19 The main screen on the Servo-i ventilator during NAVA ventilation of an infant. The waveforms, from top to bottom, are pressure, flow, volume, and time. Notice how the breath delivery is in complete synchrony with the patient's Edi signal *(drive to breathe)*. Also note that pressure, flow, and volume delivery vary breath to breath, a phenomenon that occurs during natural breathing. NAVA level is set at 2 cm $H_2O/\mu V$. NAVA $P_{peak\ est}$ = NAVA level × (Edi peak − Edi min) + PEEP; NAVA $P_{peak\ est}$ = 2 cm $H_2O/\mu V \times (7.9\ \mu V - 0.9\ \mu V) + 5$ cm H_2O; NAVA $P_{peak\ est}$ = approximately 19 cm H_2O. Displayed P_{peak} is 22 cm H_2O. This slightly higher P_{peak} represents the previous breath. (Screen capture provided by Daniel D. Rowley, RRT-NPS, RPFT, FAARC, University of Virginia Health System, Charlottesville, Va.)

backup modes. The first backup mode is pressure support. In the event that the Edi signal is lost, for example, if the catheter is pulled out or moved within the esophagus or if the Edi signal and pneumatic signal are not in sync, the ventilator will automatically switch to pressure support. An audible alarm and message will notify the operator if this is an asynchrony issue or loss of the Edi signal.

If the patient becomes apneic for some reason, for example, if a large amount of sedation is administered, the backup apnea mode is pressure control. The operator can set the pressure control level above PEEP, the rate and inspiratory time for backup pressure control mode.

The typical ventilator alarms discussed previously in Chapter 7 are also available during NAVA ventilation (i.e., upper pressure limit, high and low PEEP, high and low minute volume, high and low rate alarm, and high and low O_2 percentage).

Results of Initiating NAVA Ventilation

Several studies have reviewed changes that occur in ventilator parameters after a patient is switched to NAVA. It has been noted that often patients tend to relax and appear more comfortable after they are switched to NAVA.

When the ventilator is switching from a traditional mode of ventilation to NAVA, the mean ventilating pressures decrease, V_T tends to decrease and stabilizes at about 4 to 6 mL/kg IBW, and the respiratory rate tends to increase. The minute ventilation appears to remain fairly constant, unless the patient was previously hyperventilated or hypoventilated.[136] Once NAVA is initiated, blood gases appear to return to the patient's normal levels.[137] Oxygenation and compliance may also improve.[136,138] With NAVA the patient establishes his or her own transpulmonary pressures, volume, and respiratory rate.[110] In addition, NAVA allows a patient's respiratory center to maintain the biologically variable rhythm generation, compared with other modes such as pressure support, in which the amount of support remains constant based on what the operator has selected for the patient.[139] NAVA is not affected by leaks, and in fact is now available as noninvasive NAVA (Servo-i version 5.0 and 5.0 options).

The operator can adjust the NAVA level of support on the basis of patient assessment. In general, if the NAVA level is increased, support from the ventilator increases and the Edi signal tends to decrease. This is true only if the patient does not want the assist. The patient may accept the higher pressure and keep Edi the same. Conversely, as the NAVA level is decreased, the Edi will generally increase, but again this can vary among patients.[140] These findings, of course, will vary depending on the patient's pathology. Remember, it is the patient's respiratory center that is controlling ventilation during NAVA.

Weaning from NAVA

Clinicians should approach weaning from NAVA as with any mode of ventilation. The first step is to perform a weaning assessment of the patient to determine whether he or she is ready to be weaned (see Chapter 20). If the criteria for weaning have been met, the level of support should then be progressively reduced. Weaning protocols in some institutions may call for a spontaneous breathing trial in which all support is withdrawn. In this case the NAVA level can be set at zero and PEEP set at zero. The Edi waveform provides an excellent opportunity to evaluate the patient's response to withdrawal of support and helps determine whether the patient is ready for weaning and extubation.

The Edi catheter can remain in place after extubation by placing the ventilator in the standby mode. This allows the clinician to continue monitoring the Edi during the postextubation period. For example, the clinician can use the Edi to monitor the patient's response to the use of CPAP or a high-flow nasal cannula after extubation.[141]

Evaluating NAVA

NAVA has been shown to be a safe mode of ventilation, even in premature infants.[142] However, some clinicians argue that NAVA, unlike other modes of ventilation, requires the invasive placement of an NG tube. This is true and should be taken into account.[143] Part of the decision to use this mode should of course be based on whether use of an NG or OG tube is contraindicated for a patient. For example, an NG or OG tube might be contraindicated in a patient with head or facial injury. On the other hand, if the patient is likely to need an NG or OG tube, use of the Edi catheter may be appropriate. The Edi catheter is more expensive than a traditional NG tube. It has been suggested, however, that the initial cost associated with purchasing these catheters may be offset by the benefits of greater patient comfort and reduced length of ventilatory support.

The use of NAVA and the Edi catheter provides a novel approach to mechanical ventilation. The idea that the operator relinquishes control of volume, pressure, flow, and rate delivery to the patient is indeed a new concept. It may be difficult for some clinicians to embrace because it is "out of the box" thinking compared with traditional ventilatory support techniques.

Early studies suggest that NAVA and the Edi catheter offer a promising alternative for the management of critically ill patients receiving mechanical ventilation.[144,145] The Edi waveform provides valuable information about a patient's respiratory center function, which in turn could ultimately improve patient-ventilator synchrony. It should be stated, however, that evidence-based practice dictates that additional studies will be required to better define the application and importance of NAVA in clinical practice.

SUMMARY

- APRV is a mode of ventilatory support designed to provide two levels of continuous positive airway pressure (CPAP) and allow spontaneous breathing at both levels when spontaneous effort is present.
- APRV is referred to as *bilevel airway pressure, variable positive airway pressure, intermittent CPAP, CPAP with release, pressure-controlled inverse ratio ventilation with spontaneous ventilation, upside-down intermittent mandatory ventilation,* and *biphasic CPAP.*
- APRV provides lower peak pressures, better oxygenation, less circulatory interference, and better gas exchange without compromising the patient's hemodynamic status compared with conventional ventilation in ARDS.
- Because APRV is a pressure-targeted mode of ventilation, the patient's minute ventilation and gas exchange should be closely monitored. This is the result of the fact that volume delivery depends on lung compliance (C_L), airway resistance (R_{aw}), and the patient's spontaneous effort.
- When initiating APRV, the practitioner sets two pressure levels and two time levels. The upper level of CPAP is set with the

P_{high} control, and the lower CPAP level, or release pressure, is set with a P_{low} control. The duration of P_{high} is set with the T_{high} control, and the release time period is set with the T_{low} control.

- When changing from a conventional mode to APRV, the settings used with the conventional ventilator can serve as a guide to the APRV settings.
- Ventilation and P_aCO_2 are determined by the release time and V_T exchange during T_{low}, as well as by the patient's spontaneous ventilation. Oxygenation can generally be improved by increasing T_{high}, P_{high}, or F_IO_2.
- HFOV has been proposed as an alternative to conventional ventilation in patients with ARDS because it is thought that it offers an alternative method of improving oxygenation in adult patients while simultaneously reducing the risks for VILI.
- With HFOV, pressure is positive in the airway during the inspiratory phase (forward stroke) and negative during the expiratory phase (return stroke). Thus both inspiration and expiration are active, resulting in bulk flow rather than jet pulsations.
- Oxygenation during HFOV is primarily achieved by maintaining the mP_{aw} at a level sufficient to obtain optimal lung inflation. $\dot{V}_E$ and V_T delivery are affected by several factors, including the pressure amplitude of oscillation, frequency, ET size, amount of ET cuff leak, and lung characteristics.
- Heliox therapy has been used in the treatment of disorders that cause airway obstruction, such as asthma and tracheobronchitis. It does not cure the cause of airway obstruction but reduces the patient's WOB until the obstruction can be alleviated.
- Heliox therapy does *not* replace aggressive bronchodilator and corticosteroid therapy in the treatment of asthma.
- Heliox can be delivered using a mask system, in conjunction with aerosolized bronchodilators, and with both invasive and noninvasive mechanical ventilation. Heliox should not be delivered by nasal cannula alone. The flow through the nasal cannula is high and can be irritating, cold, and drying and can lead to undetected, inadvertent PEEP.
- When delivering heliox through a ventilator, volume and flow delivery measurements will not be accurate with most ventilators. Heliox can also interfere with gas-mixing devices, inspiratory and expiratory valve function, triggering and cycling mechanisms, automatic leak compensation, monitors, and minute volume alarms.
- The Edi catheter allows measurement and monitoring of the diaphragm's electrical activity in the clinical setting.
- An Edi may not be detected if the patient is overly sedated, hyperventilating, or receiving neuromuscular blocking agents. The impulse also may not be detected in patients with diaphragmatic hernia, in those with a severed phrenic nerve (e.g., postthoracic surgery), and after brain injury.
- NAVA uses the Edi measurement to control triggering, breath delivery, and cycling of the ventilator in spontaneously breathing patients. The operator can adjust the NAVA level of support based on patient assessment.
- Delivery of pressure during inspiration is based on the strength of the Edi signal and the level of NAVA support set by the operator.
- NAVA is not indicated for patients who are heavily sedated, demonstrate brainstem damage, have an absence of phrenic nerve activity, or a condition that prohibits neuromuscular transmission.
- In the event that the Edi signal is lost or the patient becomes apneic during NAVA, the ventilator will automatically switch to pressure support, which the operator can set as the backup mode.
- Early studies suggest that NAVA may provide an effective and novel approach to mechanical ventilation of critically ill patients.

REVIEW QUESTIONS *(See Appendix A for answers.)*

1. Which of the following are considered a benefit of APRV compared with conventional mechanical ventilation in patients with ARDS?
1. Less circulatory interference
2. Better gas exchange
3. Higher PIP
4. Improved oxygenation

A. 1 only
B. 1 and 3 only
C. 2 and 4 only
D. 1, 2, and 4

2. The benefits of APRV compared with conventional ventilation have been primarily associated with which of the following?
A. Preservation of spontaneous breathing
B. Reduction in shunt fraction
C. Less use of sedatives and analgesics
D. Augmentation of venous return

3. Disadvantages of APRV compared with conventional ventilation include:
1. Unfamiliarity of staff with the technique
2. Potential for patient-ventilator asynchrony if patient efforts are not matched with the ventilator
3. Difficulty with CO_2 elimination in patients with increased R_{aw}
4. Augmented renal perfusion when spontaneous breathing is maintained

A. 1 only
B. 2 only
C. 3 and 4
D. 1, 2, and 3

4. Which of the following represent appropriate initial settings when using APRV in patients with ARDS?
1. P_{high} of 15 to 25 cm H_2O
2. P_{low} of 10 to 15 cm H_2O
3. T_{high} of 4 seconds or more
4. T_{low} of 0.5 to 1.0 seconds

A. 2 only
B. 1 and 2
C. 2 and 3
D. 1, 3, and 4

5. The initial mP_{aw} set for HFOV in the adult is generally
A. 3 to 5 cm H_2O above the P_{aw} used with the conventional ventilator
B. 10 cm H_2O above PEEP used with the conventional ventilator

C. 15 to 25 cm H_2O
D. 30 cm H_2O

6. Which of the following controls govern the amount of displacement of the oscillating piston during HFOV?
1. Amplitude (power)
2. T_I%
3. Frequency
4. mP_{aw}
   A. 1 only
   B. 3 only
   C. 1, 2, and 3
   D. 1, 2, and 4

7. During the transition from conventional ventilation to HFOV, the patient is often:
A. Asked to remain still
B. Provided heavy sedation
C. Extubated and fitted with a face mask
D. Moved to a different unit of the hospital

8. Which of the following statements are true regarding CWF seen during HFOV?
1. CWF should be present from the clavicles to the midthigh.
2. When CWF is seen, the patient is given a paralyzing agent.
3. Auscultation of heart sounds and breath sounds is difficult when CWF is present.
4. When CWF is present, the patient must be disconnected from HFOV for breath sounds to be evaluated.
   A. 2 only
   B. 4 only
   C. 1 and 3
   D. 1, 2, and 4

9. Heliox therapy is most often used in which of the following pulmonary pathologies?
A. ARDS
B. Asthma
C. Pulmonary embolism
D. Pneumonia

10. Because of its density characteristics, heliox administered through a ventilator will affect which of the following?
A. Pressure monitoring
B. Inspiratory time
C. Volume displays
D. PEEP

11. Which of the following is advisable when using heliox during mechanical ventilation?
A. Keep two large heliox cylinders in or near the patient's room.
B. Use at least one 100% helium cylinder to save gas consumption.
C. Use a 60:40 heliox delivery to get the most effect from gas density.
D. Use a heated-wire volume-monitoring device.

12. Heliox cannot be used in which of the following circumstances?
A. During NIV
B. In patients allergic to helium gas
C. During aerosolized medication delivery
D. When appropriate equipment is not available to deliver heliox safely

13. The Edi catheter can be used for feeding, gastric evacuation, and what other function?
A. Monitoring the electrical activity of the diaphragm
B. Monitoring the esophageal pressure
C. As an optional airway
D. As a cardiac pacemaker

14. Edi signal reflects what physiological parameter?
A. Esophageal pressure
B. Upper airway pressure
C. Electrical activity of the diaphragm
D. Neural activity of the phrenic nerve

15. The NAVA backup safety features include which of the following?
A. Backup volume control
B. Backup pressure control
C. Volume support above PEEP
D. IMV plus pressure support

16. A NAVA level is set at 1 cm $H_2O/\mu V$. The Edi peak is 8 μV and the Edi min 0 μV. PEEP is set at 5 cm H_2O. What is an estimated pressure delivery to the patient based on these parameters?
A. 6 cm H_2O
B. 1 cm H_2O
C. 13 cm H_2O
D. It cannot be determined with the information given.

17. A low Edi signal may be caused by which of the following?
A. Hypoventilation
B. Oversedation
C. Atelectasis
D. Increased work of breathing

18. As with pressure support, the NAVA mode can be used in which of the following situations?
A. Paralysis
B. Heavy sedation
C. Injury to the respiratory brain centers
D. Spontaneous breathing

19. Which of the following are true regarding using the Edi catheter for monitoring?
1. The Edi can be monitored after extubation to evaluate postextubation therapy.
2. The Edi can be used to monitor if the diaphragm is active.
3. The Edi can be used to assess a current mode of ventilation to evaluate synchrony.
4. The Edi can be used to stimulate the diaphragm.
   A. 1 only
   B. 2 only
   C. 3 and 4
   D. 1, 2, and 3

20. Which of the following is true regarding NAVA?
A. Ventilator synchrony is improved.
B. Breaths are flow-cycled.
C. NAVA is a time-triggered mode.
D. The amount of pressure delivered during a single breath is constant.

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