SECTION XIII ■ RESPIRATORY DISORDERS

CHAPTER 127 ■ HEART–LUNG INTERACTIONS
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KEY POINTS
1. Spontaneous ventilation is exercise.
   a. Failure to wean may connote cardiovascular insufficiency.
   b. Weaning is a cardiovascular stress test.
   c. Breathing loads both the heart and lungs.
2. Changes in lung volume alter autonomic tone, pulmonary vascular resistance, and at high lung volumes compress the heart in the cardiac fossa in a fashion analogous to cardiac tamponade.
   a. Low lung volumes increase pulmonary vasomotor tone by stimulating hypoxic pulmonary vasoconstriction.
   b. High lung volumes increase pulmonary vascular resistance by increasing transpulmonary pressure.
3. Spontaneous inspiration and spontaneous inspiratory efforts decrease intrathoracic pressure.
   a. Increasing venous return
   b. Increasing left ventricular afterload
4. Positive pressure ventilation increases intrathoracic pressure.
   a. Decreasing venous return
   b. The decrease in venous return is mitigated by the associated increase in intra-abdominal pressure
   c. Decreasing left ventricular afterload
   d. Abolishing negative swings in intrathoracic pressure selectively reduces left ventricular (LV) afterload without reducing venous return.

OVERVIEW
Perhaps the most obvious and least understood aspect of cardiopulmonary disease is the profound and intimate relation between cardiac and pulmonary dysfunction. Heart–lung interactions go in both directions. They include the effect of the circulation on ventilation wherein acute ventricular failure causes hypoxemia and ischemic respiratory failure; and the effect of ventilation on circulation wherein hyperinflation can induce tamponade and spontaneous inspiration acute heart failure. Although most references to heart–lung interactions usually refer to the effect of ventilation on the circulation, the opposite interactions also exist and are relevant to the bedside clinician.

Heart–lung interactions can be grouped into interactions that involve three basic concepts that usually coexist (1,2).

First, spontaneous ventilation is exercise, requiring O$_2$ and blood flow, thus placing demands on cardiac output, and producing CO$_2$, adding additional ventilatory stress on CO$_2$ excretion. Second, inspiration increases lung volume above resting end-expiratory volume. Thus, some of the hemodynamic effects of ventilation are due to changes in lung volume and chest wall expansion. Third, spontaneous inspiration decreases intrathoracic pressure (ITP) whereas positive pressure ventilation increases ITP. Thus the differences between spontaneous ventilation and positive pressure ventilation primarily reflect the differences in ITP swings and the energy necessary to produce them.

THE EFFECTS OF CARDIOVASCULAR DYSFUNCTION ON VENTILATION
Cardiogenic shock can induce hydrostatic pulmonary edema, impairing acute hypoxic respiratory failure. Circulatory shock, by limiting blood flow to the respiratory muscles, can induce respiratory muscle failure and respiratory arrest. These points underscore a fundamental aspect of ventilation, namely that it is exercise, and like any form of exercise, it must place a certain metabolic demand on the cardiovascular system (3). If cardiovascular reserve is limited, this metabolic demand may exceed the heart's ability to deliver O$_2$ to meet the increased metabolic activity associated with spontaneous ventilation. Thus, ventilator-dependent patients with cardiovascular insufficiency may not be able to wean from mechanical ventilation because the metabolic demand of spontaneous ventilation is too great. Since this increased stress occurs only during the weaning trial, such insufficiency may not be apparent prior to weaning attempts.

Under normal conditions, respiratory muscle blood flow is not the limiting factor determining maximal ventilatory effort even with marked respiratory efforts. Although ventilation normally requires less than 5% of total O$_2$ consumption (3), if the work of breathing is increased, such as in pulmonary edema, pulmonary fibrosis, or bronchospasm, the work cost of breathing can increase to 25% of total O$_2$ consumption (3–6). If cardiac output is limited, then blood flow to all organs including the respiratory muscles may be compromised, inducing both tissue hypoperfusion and lactic acidosis (7–10). Under these severe heart failure conditions respiratory muscle failure may develop despite high central neuronal drive (11). Supporting spontaneous ventilation by the use of mechanical ventilation will reduce O$_2$ consumption increasing SvO$_2$ for a constant
hemodynamic effects of ventilation and ventilatory maneuvers

Ventilation can profoundly alter cardiovascular function. The specific response will be dependent on myocardial contractility and preload reserve, circulating blood volume, blood flow distribution, autonomic tone, endocrinologic responses, lung volume, intrathoracic pressure (ITP), and the surrounding pressures for the remainder of the circulation (24,25).

To understand this issue better one must understand, at least in part, the relation between airway pressure (Paw) and ITP, the transpulmonary pressure. Paw is relatively easy to measure (26,27), whereas ITP is not. Positive pressure ventilation-induced increases in Paw do not necessarily equate to proportional increases in ITP. The primary determinants of the hemodynamic responses to ventilation are due to changes in ITP and lung volume (28), not Paw. The relation between Paw, ITP, pericardial pressure (Ppc) and lung volume varies with spontaneous ventilatory effort, as well as lung and chest wall compliance. Lung expansion during positive pressure inspiration pushes on the surrounding structures, distorting them and causing their surface pressures to increase, increasing both Ppl and Ppc (29). Only lung and thoracic compliance determine the relation between end-expiratory Paw and lung volume in the sedated and paralyzed patient. However, if a ventilated patient actively resists lung inflation or sustains expiratory muscle activity at end-inspiration, then end-inspiratory Paw will exceed resting Paw for that lung volume. Similarly, if the patient actively prevents full exhalation by expiratory braking, then for the same end-expiratory Paw, lung volume may be higher than predicted from end-expiratory Paw values. At end-expiration, if the respiratory system is at rest, Paw equals alveolar pressure and lung volume is at functional residual capacity. If incomplete exhalation occurs, then alveolar pressure will exceed Paw. The difference between measured Paw and alveolar pressure is called intrinsic positive end-expiratory pressure (PEEP). Finally, if chest wall compliance decreases, as may occur with increased abdominal pressure, both Paw and ITP will increase for the same tidal breath.

Since the heart is fixed within a cardiac fossa and cannot be displaced in any direction, juxtacardiac Ppl will increase more than lateral chest wall or diaphragmatic Ppl during inspiration. Pcc is the outside pressure to LV intraluminal ventricular pressure determining LV filling. Pcc and ITP may not be similar nor increase by similar amounts with the application of positive Paw, if the pericardium acts as a limiting membrane (30,31). With pericardial restraint, as in tamponade, Pcc exceeds juxtacardiac Ppl (32). With progressive increases in PEEP, juxtacardiac Ppl will increase toward Pcc levels, whereas Pcc will initially remain constant. Once these two pressures equalize, further increases in PEEP by increasing lung volume will increase both juxtacardiac Ppl and Pcc in parallel. Thus, if pericardial volume restraint exists, as may occur with acute cor pulmonale or tamponade, then juxtacardiac Ppl will underestimate Pcc.

The presence of lung parenchymal disease, airflow obstruction, and extrapulmonary processes that directly alter or influence wall-diaphragmatic contraction or intra-abdominal pressure may also alter these interactions. Static lung expansion occurs as Paw increases because the transpulmonary pressure (Paw relative to ITP) increases. If lung injury induces alveolar flooding or increased pulmonary parenchymal stiffness, then greater increases in Paw will be required to distend the lungs to a constant end-inspiratory volume (9,29,33). Thus, the primary determinants of the increase in Ppl and Pcc during positive pressure ventilation are lung volume change and chest wall compliance, not Paw change (34). Since acute lung injury (ALI) is often nonhomogeneous, with aerated areas of the lung displaying normal specific compliance, increases in Paw above approximate 30 cm H2O will overstress these aerated lung units (35). Vascular structures that are distended will have a greater increase in their surrounding pressure than collapsible structures that do not distend (36). Despite this nonhomogeneous alveolar distention, if tidal volume is kept constant, then Ppl increases equally, independent of the mechanical properties of the lung (33,37,38). Thus, under constant tidal volume conditions, changes in peak and mean Paw will reflect changes in the mechanical properties of the lungs and patient coordination, but may not reflect changes in ITP. Thus, one cannot predict the amount of change in ITP or Pcc that will occur in a given patient as PEEP is varied. Accordingly, assuming some constant fraction of Paw transmission to the pleural surface as a means of calculating the effect of increasing Paw on ITP is inaccurate and potentially dangerous if used to assess transmural intrathoracic vascular pressures. However, if the patient
has a pulmonary artery catheter in situ, then one can estimate end-expiratory ITP. The clinician has the ability to measure on-PEEP and end-expiratory vascular pressures by calculating the airway pressure transmission index to the pleural space (39) or by briefly removing PEEP while these pressures are directly measured (38). The ratio of end-inspiratory-to-end-expiratory pulmonary artery diastolic pressure (reflecting ITP changes) to Paw (reflecting alveolar pressure changes) defines the pulmonary transmission index. If one assumes that lung compliance is linear over the given tidal volume, then the product of this transmission index and PEEP represents the end-expiratory ITP.

HEMODYNAMIC EFFECTS OF CHANGES IN LUNG VOLUME

Changing lung volume alters autonomic tone, pulmonary vascular resistance, and at high lung volumes, compresses the heart in the cardiac fossa, limiting absolute cardiac volumes analogous to cardiac tamponade. However, unlike tamponade where Ppc selectively increases in excess of Ppl, with hyperinflation both juxtacardiac Ppl and Ppc increase together.

Autonomic Tone

Cyclic changes in lung volume induce cyclic changes in autonomic inflow. The lungs are richly innervated with integrated somatic and autonomic fibers that originate, traverse through, and end in the thoracic cavity. These neural pathways mediate many homeostatic processes through the autonomic nervous system that alter both instantaneous cardiovascular function and steady-state cardiovascular status (40,41). Lung inflation to normal tidal volumes (<10 mL/kg) induces parasympathetic withdrawal, increasing heart rate. This inspiration-induced cardiac acceleration is referred to as respiratory sinus arrhythmia (42). The presence of respiratory sinus arrhythmia connotes normal autonomic control (43) and is used in diabetics with peripheral neuropathy to assess peripheral dysautonomia (44). Inflation to larger tidal volumes (>15 mL/kg) decreases heart rate by a combination of both increased vagal tone (45) and sympathetic withdrawal. The sympathetic withdrawal also creates arterial vasodilation (46–50). This inflation–vasodilatation response induces expiration-associated reductions in LV contractility in healthy volunteers (51), and in ventilator-dependent patients with the initiation of high-frequency ventilation (40) or hyperinflation (48). Humoral factors, including compounds blocked by cyclo- oxygenase inhibition (52), released from pulmonary endothelial cells during lung inflation may also induce this depressor response (53–55). However, these interactions do not appear to grossly alter cardiovascular status (56). Although oversaturation of aerated lung units in patients with acute lung injury (ALI) may induce such cardiovascular depression, unilateral lung hyperinflation (unilateral PEEP) does not appear to influence systemic hemodynamics (57). Thus, these cardiovascular effects are of uncertain clinical significance.

Ventilation also compresses the right atrium and through this mechanical effect alters control of intravascular fluid balance. Both positive pressure ventilation and sustained hyperinflation decrease right atrial stretch stimulating endocrinologic responses that induce fluid retention. Plasma norepinephrine, plasma renin activity (58,39), and atrial natriuretic peptide (60) increase during positive pressures by calculating the airway pressure transmission index to the pleural space (39) or by briefly removing PEEP while these pressures are directly measured (38). The ratio of end-inspiratory-to-end-expiratory pulmonary artery diastolic pressure (reflecting ITP changes) to Paw (reflecting alveolar pressure changes) defines the pulmonary transmission index. If one assumes that lung compliance is linear over the given tidal volume, then the product of this transmission index and PEEP represents the end-expiratory ITP.

Chapter 127: Heart-Lung Interactions

Pulmonary Vascular Resistance

Ventilation alters pulmonary vascular resistance, and thus pulmonary arterial pressure. Right ventricular (RV) ejection performance is markedly limited by increases in RV ejection pressure because the right ventricle has thin walls that cannot distribute increased wall stress. Sudden increases in pulmonary arterial pressure can induce cardiovascular collapse. This is the common cause of cardiovascular collapse, for example, with massive pulmonary embolism. The mechanisms inducing changes in pulmonary vascular resistance with changing lung volume are often complex, often conflicting, and include both humoral and mechanical interactions. Increasing lung volume occurs because transpulmonary pressure increases. For example, although obstructive inspiratory efforts, as occur during obstructive sleep apnea, are usually associated with increased RV afterload, the increased afterload is due primarily to either increased vasomotor tone (hypoxic pulmonary vasoconstriction) or backward LV failure and not lung volume–induced changes in pulmonary vascular resistance (63,64). RV right ventricle, like LV afterload, can be defined as the maximal RV systolic wall stress during contraction (65). Thus, it is a function of the maximal product of end-diastolic radius of curvature (a function of end-diastolic volume) and transmural pressure (a function of systolic RV pressure) during ejection (66). Systolic RV pressure equals transmural pulmonary artery pressure. Increases in transmural Ppa impede RV ejection (67), decreasing RV stroke volume (68) and inducing RV dilation, and passively impede venous return (52,54). If not relieved quickly, acute cor pulmonale rapidly develops (69). Furthermore, if RV dilation and RV pressure overload persist, RV free wall ischemia and infarction can develop (70). Importantly, rapid fluid challenges in the setting of acute cor pulmonale can precipitate profound cardiovascular collapse due to excessive RV dilation, RV ischemia, and compromised LV filling.

The pulmonary vascular resistance is alveolar PO2 (Pao2) decreases to below 60 mm Hg (71). This process of hypoxic pulmonary vasoconstriction is mediated, in part, by variations in the synthesis and release of nitric oxide by endothelial nitric oxide synthase localized on pulmonary vascular endothelial cells and in part by an NAD/NADH voltage-dependent calcium channel in the pulmonary vasculature. Hypoxic pulmonary vasoconstriction, by reducing pulmonary blood flow to hypoxic lung regions, minimizes shunt blood flow. However, if generalized alveolar hypoxia occurs, then pulmonary vasomotor tone increases, increasing pulmonary vascular resistance and impeding RV ejection (63). Importantly, at low lung volumes, alveoli spontaneously collapse as a result of loss of intrathoracic traction and closure of the terminal airways. This collapse causes both absorption atelectasis and alveolar hypoxia. Patients with acute hypoxic respiratory failure have small
lungs and are prone to spontaneous alveolar collapse (72,73). Therefore, pulmonary vascular resistance is often increased in patients with acute hypoxic respiratory failure due to small lung volumes and atelectasis (e.g., ARDS).

Mechanical ventilation may reduce pulmonary vasomotor tone, reducing pulmonary artery pressure and RV afterload by any one of many related processes. First, hypoxic pulmonary vasoconstriction can be inhibited if O2-enriched inspired gas increases PaO2 (74–77) or if the mechanical breaths and PEEP by recruiting collapsed alveolar units, increases PaO2 in those local alveoli (28,78–80). Second, mechanical ventilation often reverses respiratory acidosis by increasing alveolar ventilation, which itself stimulates pulmonary vasoconstriction (77). Finally, decreasing central sympathetic output by sedation during mechanical ventilation will also reduce vasomotor tone (81–83).

Increases in lung volume directly increase pulmonary vascular resistance by compressing the alveolar vessels (72,79,80). The actual mechanisms by which this occurs have not been completely resolved, but appear to reflect differential extra-aluminal pressure gradient–induced vascular compression. The pulmonary circulation can be conceptually viewed as existing in two distinct compartments based on the pressure outside the blood vessels, which will be either alveolar pressure (alveolar vessels) or extra-alveolar or ITP (extra-alveolar vessels) (79). The small pulmonary arterioles, venules, and alveolar capillaries sense alveolar pressure as their surrounding pressure while the large pulmonary arteries and veins, as well as the heart and intrathoracic great vessels of the systemic circulation, sense interstitial pressure or ITP as their surrounding pressure. Since alveolar pressure minus ITP is the transpulmonary pressure, and increasing lung volume requires transpulmonary pressure to rise, increases in lung volume augments the extraluminal pressure gradient from extra-alveolar vessels to alveolar vessels. Increases in lung volume progressively raise alveolar vessel resistance, becoming most noticeable above functional residual capacity (FRC) (75,84) (Fig. 127.1). Since the intraluminal pressure in the pulmonary arteries is generated by RV ejection relative to ITP, but the outside pressure of the alveolar vessels is alveolar pressure, if transpulmonary pressure exceeds intraluminal pulmonary arterial pressure, then the pulmonary vasculature will collapse where extra-alveolar vessels pass into alveolar lacs, reducing the vasculature cross-sectional area and increasing pulmonary vascular resistance. Hyperinflation can create significant pulmonary hypertension and may precipitate acute RV failure (acute cor pulmonale) (85) and RV ischemia (70), especially in patients prone to hyperinflation (e.g., chronic obstructive pulmonary disease [COPD]). Thus, PEEP may increase pulmonary vascular resistance if it induces lung overdistention (86). Similarly, if lung volumes are reduced, increasing lung volume back to baseline levels by the use of PEEP decreases pulmonary vascular resistance by reversing hypoxic pulmonary vasoconstriction (87).

**Ventricular Interdependence**

Although LV preload must eventually be altered by changes in RV output because the two ventricles are in series, changes in RV end-diastolic volume can also alter LV preload by altering LV diastolic compliance by the mechanism of ventricular interdependence (88). Ventricular interdependence functions through two separate processes. First, increasing RV end-diastolic volume will induce an intraventricular septal shift into the LV, decreasing LV diastolic compliance (89). Thus, for the same LV filling pressure, RV dilation will decrease LV end-diastolic volume and, therefore, cardiac output. Second, if pericardial restraint limits absolute biventricular filling, then RV dilation will increase Ppc without septal shift (2,90). This ventricular interaction is believed to be the major determinant of the phasic changes in arterial pulse pressure and stroke volume seen in tamponade, and is referred to as pulsatia paradoxus. Pulses paradoxus can be demonstrated during loaded spontaneous inspiration in normal subjects as an inspiration-associated decrease in pulse pressure. Pulse pressure is defined as systolic minus the diastolic blood pressure. If the pulse pressure change is greater than 10 mm Hg or 10% of the mean pulse pressure, then it is referred to as pulsus paradoxus (2). Maintaining a constant rate of venous return, either by volume resuscitation (91) or vasopressor infusion (27), will minimize hyperinflation-induced cardiac compression.

**Hyperinflation-induced Cardiac Compression**

As lung volumes increase, the heart is compressed between the expanding lungs (92), raising juxta-ventricular ITP. This compressive effect of the inflated lungs can be seen with either spontaneous (93) or positive pressure–induced hyperinflation (3,38,94–96). As described above, both Ppc and ITP are increased and no pericardial restraint exists. This decrease in apparent LV diastolic compliance (91) was previously misinterpreted as impaired LV contractility, because LV stroke work for a given LV end-diastolic pressure or pulmonary artery occlusion pressure is decreased (97,98). However, when such patients are fluid resuscitated to return LV end-diastolic volume to its original level, both LV stroke work and cardiac output also appear normal.
HEMODYNAMIC EFFECTS OF CHANGES IN INTRATHORACIC PRESSURE

The heart within the thorax is a pressure chamber within a pressure chamber. Therefore, changes in ITP must affect the pressure gradients for both systemic venous return to the RV and systemic outflow from the LV, independent of the heart itself. Increases in ITP, by increasing right atrial pressure and decreasing transmural LV systolic pressure, will reduce the pressure gradients for venous return and LV ejection, decreasing intrathoracic blood volume. Conversely, decreases in ITP will augment venous return and impede LV ejection, increasing intrathoracic blood volume. Everything else below follows from these two simple truths.

Venous Return

Blood flows back from the systemic venous reservoirs into the right atrium through low-pressure, low-resistance venous conduits (101). Right atrial pressure is the back pressure for venous return. Ventilation alters both right atrial pressure and venous reservoir pressure. It is these changes in right atrial and venous capacitance vessel pressure that induce most of the observed cardiovascular effects of ventilation. Pressure in the upstream venous reservoirs is called mean systemic pressure, and it is, itself, a function of blood volume, peripheral vasomotor tone, and the distribution of blood within the vasculature (102). Usually mean systemic pressure does not change rapidly during positive pressure ventilation, whereas right atrial pressure does owing to concomitant changes in ITP. Thus, variations in right atrial pressure represent the major factor determining the fluctuation in pressure gradient for systemic venous return during ventilation (103,104). The positive pressure inspiration increases in right atrial pressure decrease the pressure gradient for venous return, decreasing RV filling (68) and RV stroke volume (68,103,105–113). During normal spontaneous inspiration, the opposite occurs (2,26,68,69,107,110,114,115). The detrimental effect of positive pressure ventilation on cardiac output can be minimized by either fluid resuscitation to increase mean systemic pressure (27,105,116,117) or by keeping both mean ITP and swings in lung volume as low as possible. Accordingly, prolonging expiratory time, decreasing tidal volume, and avoiding PEEP all minimize this decrease in systemic venous return to the RV (4,24,103,107-111,118).

However, if positive pressure ventilation-induced increases in right atrial pressure always proportionally decreased venous return, then most mechanically ventilated patients would dysfunction the primary mechanism by which the decrease in venous return is minimized during positive pressure ventilation (121–125). However, laparotomy, by abolishing the inspiration-associated increases in intra-abdominal pressure, makes surgery patients especially sensitive to mechanical ventilation, requiring increased fluid resuscitation to sustain a constant cardiac output. This is one of the reasons why abdominal surgery patients often leave the operating room many liters positive.

Spontaneous inspiratory efforts usually increase venous return because of the combined decrease in right atrial pressure (2,26,108–110) and increase in intra-abdominal pressure (119,120). However, this augmentation of venous return is limited (126–128) because as ITP decreases below atmospheric pressure, central venous pressure also becomes subatmospheric, collapsing the great veins as they enter the thorax and creating a flow-limiting segment (101).

Ventricular Interdependence

Changes in RV volume induce reciprocal changes in LV diastolic compliance. Although decreasing RV volume during positive pressure inspiration increases LV diastolic compliance by decreasing RV filling, the hemodynamic impact of this effect is usually minimal (88,127–132) (Fig. 127.2). However, with spontaneous inspiration RV volumes increase, causing an immediate reduction in LV diastolic compliance. This process is the primary cause for the inspiration-associated decrease in LV stroke volume and pulse pressure (87,89,132,133). If the pulse pressure change is greater than 10 mm Hg or 10% of the mean pulse pressure, then it is referred to as pulsus paradoxus (2). Since spontaneous inspiratory

![Figure 127.2](image-url)
efforts can also occur during positive pressure ventilation, the use of ventilation-associated pulse pressure variation during positive pressure ventilation can reflect ventilator interdependence. Presently, positive pressure-induced changes in pulse pressure and LV stroke volume have been advocated to be a useful parameter of preload responsiveness (134). However, to assess volume responsiveness using pulse pressure variation, it is essential that no spontaneous inspiratory efforts be present.

**LV Afterload**

Changes in ITP can directly and indirectly alter LV afterload by altering both LV end-diastolic volume and ejection pressure. LV ejection pressure can be estimated as arterial pressure relative to ITP. Since baroreceptor mechanisms located in the extrathoracic carotid body maintain arterial pressure constant relative to atmosphere, if arterial pressure were to remain constant as ITP increased, then transmural LV pressure and thus LV afterload would decrease. Similarly, if transmural arterial pressure were to remain constant as ITP decreased, then LV wall tension would increase (133). Thus, under steady-state conditions increases in ITP decrease LV afterload and decreases in ITP increase LV afterload (136,137). The spontaneous inspiration–associated decrease in ITP-induced increase in LV afterload is one of the major mechanisms thought to be operative in the wean-induced LV schema described in the first part of this chapter since increased LV afterload must increase myocardial O$_2$ consumption (MVO$_2$). Thus, spontaneous ventilation not only increases global O$_2$ demand by its exercise component (3–5) but also increases MVO$_2$. Profoundly negative swings in ITP commonly occur during forced spontaneous inspiratory efforts in patients with bronchospasm and obstructive breathing. This condition may rapidly deteriorate into acute heart failure and pulmonary edema (63) as has been described for airway obstruction (asthma, upper airway obstruction, vocal cord paralysis). Stiff lungs (interstitial lung disease, pulmonary edema, and ALI) selectively increase LV afterload and may be the cause of their LV failure and pulmonary edema (1,49,6,64), especially if LV systolic function is already compromised (13,138). Clearly, weaning from mechanical ventilation is a selective LV stress test (135,139,140). Similarly, improved LV systolic function is observed in patients with severe LV failure placed on mechanical ventilation if the mechanical breaths abolish negative swings in ITP (140).

The observed improvement in LV function seen with positive-pressure ventilation in subjects with severe heart failure is self-limited because venous return also decreases limiting total blood flow. However, the effect of removing large negative swings in ITP on LV performance will also act to reduce LV afterload but will not be associated with a change in venous return because until ITP becomes positive, venous return remains constant. Thus, removing negative ITP swings in LV afterload will selectively reduce LV afterload in a fashion analogous to increasing ITP but without the effect on cardiac output (27,101,141–144). This concept has been validated to be a very important clinical approach for patients with obstructive sleep apnea. For example, the cardiovascular benefits of positive airway pressure in nonintubated patients can be seen with CPAP therapy (145,146). Even low levels of CPAP, if they inhibit airway obstruction, will be beneficial (147–149). Prolonged nighttime nasal CPAP can selectively improve respiratory muscle strength, as well as LV contractile function if the patients had pre-existent heart failure (150,151). These benefits are associated with reductions of serum catecholamine levels (152).

**Using Heart–Lung Interactions to Diagnose Cardiovascular Insufficiency**

Since the cardiovascular response to positive pressure breathing is determined by the baseline cardiovascular state, ventilation-associated changes in arterial pulse pressure and stroke volume should monitor dynamic changes in venous return and the responsiveness of the heart to these transient and cyclic changes in preload (153). Systolic pressure variations during positive pressure ventilation nicely describe both preload responsiveness if the systolic pressure decreases below an apneic baseline value and also predict heart failure with volume overload if the systolic pressure increases above apneic baseline values (154–157). However, it is often difficult to assess if the variation in systolic arterial pressure is primarily up or down in clinical settings. A more physiologic approach is to measure arterial pulse pressure and assess pulse pressure variation (134,158). This technique can be modified to assess stroke volume variation (139) and has profound clinical potential as newer monitoring devices allow for the bedside display of both pulse pressure and stroke volume variation. In subjects on controlled mechanical ventilation, a pulse pressure variation of >10% accurately predict preload responsiveness. This novel and exciting application of heart-lung interactions has been validated in many studies and is presently being assessed in prospective clinical trials. Assuming that this practical application of heart-lung interactions becomes commonplace, then a basic understanding of the principals described in this chapter will be an essential part of the training of acute care physicians.

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**CHAPTER 128: ANATOMY OF MECHANICAL VENTILATION**

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**IMMEDIATE CONCERNS**

Ventilation/Perfusion (V\(_A\)/Q) in the Normal Lung

The lung’s primary function is to add oxygen (O\(_2\)) to, and remove carbon dioxide (CO\(_2\)) from, blood passing through the pulmonary capillary beds. For this to occur, the gas we breathe must be matched to the blood flowing through our lungs. Average minute alveolar ventilation (V\(_A\)) for a healthy adult is 4 liters (L) per minute, while resting cardiac output (Q) is 5 L/minute; therefore that optimal ventilation/perfusion matching (V\(_A\)/Q ratio) is 4 L/minute divided by 5 L/minute or 0.8. Perfect V\(_A\)/Q matching is unlikely because the distribution of gas and blood flow varies across the lung fields for several reasons. Both gases and blood have mass and are therefore gravity dependent (Fig. 128.1); as a result, both increase as we progress from the apex to the base of the lung. Gravity’s effect on blood flow is, however, predominant; it has been estimated that in an upright subject, six times as much blood passes through each lung base compared to its apex, whereas only two times as much air reaches each lung base. These different gradients dictate that the V\(_A\)/Q ratio rises progressively from the bottom to the top of the lungs.

Furthermore, the lungs are composed of millions of alveoli, connected to each other and eventually to the trachea by a labyrinth of pathways and interconnections (pores of Kohn). Few connections are consistent in either length or diameter; this effect conspires to further disrupt the distribution of inhaled gases. Even the healthiest athletes exhibit areas of shunt and dead space (Fig. 128.2). About 30% of the air a healthy adult breathes each minute is wasted as dead space ventilation (V\(_D\)) and to 1% of the cardiac output passes through the lungs without undergoing gas exchange (shunt). Any pathophysiological stimulus that acutely increases or decreases ventilation or cardiac output is likely to have a pronounced impact on V\(_A\)/Q ratios and in turn, on oxygenation and CO\(_2\) removal.

**Positive Pressure Breathing**

Positive pressure mechanical ventilation, unlike normal breathing, increases transpulmonary pressure, reduces venous return, as much air reaches each lung base. These different gradients as much air reaches each lung base. These different gradients dictate that the V\(_A\)/Q ratio rises progressively from the bottom to the top of the lungs.

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and ultimately lowers cardiac output. Positive pressure breathing also preferentially forces gas into areas of the lung with the lowest airway resistance ($R_{aw}$) and highest compliance ($C_{RS}$). It is not uncommon for ventilated patients to require airway pressures ($P_{aw}$) of 30 cm H$_2$O or more; yet, normal systolic pulmonary arterial pressures seldom exceed 20 to 25 mm Hg. It follows that during positive pressure inflations, if intraluminal alveolar pressure exceeds the hydrostatic pressure, blood flow and gas exchange cease—until alveolar pressure falls below hydrostatic levels again during exhalation. Given these factors, it is easy to understand how mechanical ventilation often disrupts $V_{A}/Q$, and why up to 60% or more of each positive pressure breath is wasted as $V_D$. Allowing patients to breathe spontaneously between mechanical breaths significantly reduces mean transpulmonary and transluminal pressures and improves venous return and cardiac output, which in turn improves $V_{A}/Q$.

**$V_{A}/Q$ Inequalities in Respiratory Failure**

It is not necessary that alveoli be completely deprived of $V_A$ or $Q$ for life-threatening symptoms to exist. When significant areas of a patient’s lungs receive too much or too little $V_A$ or $Q$, these regions exhibit abnormally high or low $V_{A}/Q$ ratios, referred to as relative shunt and relative dead space (Fig. 128.3).

- **Relative shunt** and $V_D$ are extremely common in the intensive care unit (ICU) setting, and disrupt $CO_2$ removal and oxygenation just as quickly as comparable but smaller areas of absolute $V_D$ or shunt.

**Conditions Affecting Lung Structure**

Along with conditions that affect only $V_A$ or $Q$, several disorders actually damage lung structure. Furthermore, failure to properly manage the ventilator, in some situations, may play a role in determining the ultimate severity and progression of the lung disease (1–7). Critical care personnel called on to manage ventilators for these patients, whether in a primary or consulting role, must possess a thorough understanding of the pathophysiology and treatment of acute respiratory failure.

Reducions in the arterial partial pressure of oxygen ($PaO_2$) and carbon dioxide ($PaCO_2$) are characteristic of the early stages of acute respiratory distress syndrome (ARDS).
Widespread, but not uniform, alveolar destabilization and collapse (atelectasis) are hallmarks of ARDS. If ARDS is not aggressively managed in its early stages, pulmonary consolidation (secondary to atelectasis) develops and may lead to a fibro-prolific phase; the chances for recovery are significantly reduced if the disease progresses to this point (3). Hypoxemia results from both relative and absolute shunting caused by complete or partial alveolar collapse and the continued perfusion of these lung regions.

**Ventilator Therapy**

With respect to therapy, a shifting emphasis in the role of mechanical ventilation has occurred. Positive pressure ventilation was clearly responsible for the decrease in mortality following the poliomyelitis epidemic. Yet, a similar reduction in mortality has been slow to respond following the widespread application of mechanical ventilatory support to ARDS or to acute exacerbations of chronic obstructive pulmonary disease (COPD). Although this is in part due to the multisystem dysfunction that frequently accompanies such problems, it now appears that the inappropriate use of mechanical ventilation has played a significant role. Poliomyelitis, Guillain-Barré syndrome, and other neuromuscular disease states produce respiratory insufficiency because of mechanical and neural failure to control diaphragmatic driven ventilation. In the absence of complications such as aspiration of gastric contents, pulmonary parenchymal function remains intact. By contrast, ARDS represents a failure of gas exchange that is related almost entirely to parenchymal injury. In particular, the musculoskeletal inability of the diaphragm to relax properly has led to widespread atelectasis and hypoxemia; these conditions respond poorly to mechanical ventilation alone. The law of Laplace states that pressure inside a spherical structure is directly proportional to tension in that structure's wall and inversely proportional to its radius. Normally, alveolar surface forces, at alveolar-capillary membranes, are essentially identical. Laplace's law dictates that a loss of surfactant means a greater pressure is required to keep smaller alveoli open (Fig. 128.4). When this occurs, smaller alveoli empty into larger ones, eventually collapsing. A plot of the lung's pressure-volume relationship during ARDS helps to better visualize this phenomenon (Fig. 128.5). Without adequate surfactant, significant portions of the lungs collapse at end-exhalation. During inhalation, as pressure is applied to the airways (x-axis, Fig. 128.5), nothing initially happens. However, when the applied pressure reaches sufficient magnitude, in this instance 14 cm H₂O, some of the collapsed alveoli start to open and gas begins entering the lungs. This “opening” pressure is commonly referred to as the lower inflection point (Fig. 128.5) and provides the theoretical underpinnings for the use of PEEP, that is, an ARDS-related surfactant deficiency predisposes to alveolar collapse unless counteracted by force. Clinically, the easiest way to accomplish this goal is by maintaining PEEP.

Reduced levels of surfactant, such as occur during ARDS, lead to widespread atelectasis and hypoxemia; these conditions respond poorly to mechanical ventilation alone. The law of Laplace states that pressure inside a spherical structure is directly proportional to tension in that structure's wall and inversely proportional to its radius. Normally, alveolar surface forces, at alveolar-capillary membranes, are essentially identical. Laplace's law dictates that a loss of surfactant means a greater pressure is required to keep smaller alveoli open (Fig. 128.4). When this occurs, smaller alveoli empty into larger ones, eventually collapsing. A plot of the lung's pressure-volume relationship during ARDS helps to better visualize this phenomenon (Fig. 128.5). Without adequate surfactant, significant portions of the lungs collapse at end-exhalation. During inhalation, as pressure is applied to the airways (x-axis, Fig. 128.5), nothing initially happens. However, when the applied pressure reaches sufficient magnitude, in this instance 14 cm H₂O, some of the collapsed alveoli start to open and gas begins entering the lungs. This “opening” pressure is commonly referred to as the lower inflection point (Fig. 128.5) and provides the theoretical underpinnings for the use of PEEP, that is, an ARDS-related surfactant deficiency predisposes to alveolar collapse unless counteracted by force. Clinically, the easiest way to accomplish this goal is by maintaining PEEP.

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**Positive End-expiratory Pressure (PEEP)**

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Section XIII: Respiratory Disorders

A. Without Surfactant

B. With Surfactant

\[ P = \frac{2T}{r} \]

FIGURE 128.4. Laplace's law and its effect on alveoli. Laplace's law states that pressure (P) inside a sphere is directly proportional to the tension in the walls (T) and inversely proportional to the sphere’s radius (r). A: Without surfactant. Wall or surface tension in both large and small alveoli is about the same. As a result, a greater pressure develops in the smaller alveolus, which then proceeds to empty into adjacent larger alveoli. B: With surfactant. The surface tension-reducing properties of surfactant increase as individual surfactant molecules get closer together. This property counteracts Laplace’s law and reduces the tendency for small alveoli to empty into nearby larger alveoli.

FIGURE 128.5. Inflation and deflation characteristics in the surfactant-deficient lung. Surfactant-deficient alveoli generally remain open throughout exhalation (open circles); at end-exhalation, unstable alveoli empty into adjacent larger alveoli and collapse, significantly reducing functional residual capacity (FRC). Furthermore, once collapsed, alveoli tend to stay collapsed until a relatively high pressure is applied. In this idealized example, airway pressure is steadily increased to inflate the lungs (solid circles). Note that airway pressure reaches 14 cm H\(_2\)O before any measurable volume enters the lungs; at this point (lower inflection point), collapsed alveoli begin to open, the lungs begin to accept volume, and the pressure-volume curve changes slope upward. Alveoli continue to open, those already open expand, and airway pressure continues to rise until the average patent alveoli begin to approach their maximum volume. At this point, the pressure-volume curve flattens; in other words, from this point on, larger changes in pressure will be required to produce a complementary change in volume. This slope change is referred to as the upper inflection point. Although the pressure-volume curve may be difficult to measure at the bedside, avoiding ventilator-induced lung injury likely requires that all mechanical ventilation occur between the upper and lower inflection points, that is, patient airway pressure should not be allowed above the upper inflection point or below the lower inflection point.
preferably somewhat above the lower inflection point (12). Since the therapeutic objective is to prevent alveolar collapse—that is, to keep the alveoli open—the approach is often referred to as the open lung approach (13,14).

Combining mechanical ventilation and PEEP usually decreases shunt and improves oxygenation (15–18), often significantly; nevertheless, ARDS mortality rates have to improve. The reasons for this are complex, but theories for these failures are starting to emerge (19). It now appears that, to reduce ARDS-associated morbidity and mortality, we must avoid the risks associated with both low-volume (4–7) and high-volume (1–3) lung injury. To this end, all tidal ventilation must occur between the lower and upper inflection points (19). It sounds easy, but bedside determination of inflection points is difficult; nevertheless, it is worth the effort.

To date, a universally agreed on ventilatory approach, or mode, for managing critically ill patients has failed to emerge. Considering the wide variety of conditions ameliorated by mechanical ventilation, and the extreme range of severity between patients with the same problem, a single, always best approach may not exist. Clinicians, therefore, must understand and recognize the potential and limitations of their favored approaches.

**VENTILATOR CLASSIFICATION**

**Positive versus Negative Pressure**

Today, virtually all ventilators function by providing some variant of positive pressure. Yet, during the polio epidemic, “iron lungs” or negative pressure ventilators were in common use. Negative pressure devices require that the patient’s body be tightly enclosed within a tube or box while the head remains outside. Once the patient is sealed inside, a pump or bellows evacuates gas from inside the box; this creates a negative pressure around the patient’s thorax, making atmospheric pressure positive in relation to alveolar pressure. As a result, gas flows from the mouth to alveoli, trying to equalize the pressure difference. Since this process is nearly identical to normal breathing, negative pressure ventilators tend to provide better $V_t/Q$ ratios (20) and produce less interference with cardiac output (21) than positive pressure counterparts. Nevertheless, these devices quickly lost favor for several compelling reasons: (a) iron lungs are very large and difficult to move; (b) maintaining an airtight seal around the patient’s neck, without irritation, is nearly impossible; (c) personnel responsible for providing patient monitoring and routine care could not easily access important areas of their patient’s body.

**Controlled versus Assisted Breaths**

Although modern ICU ventilators offer many different operational modes, from the patient’s standpoint, only two breath types remain: controlled (mechanical or mandated) and assisted/spontaneous. Controlled breaths, used during controlled mechanical ventilation (CMV), are completely defined by the attending clinician. Controlled breaths are always delivered on schedule and without regard for the patient. For this reason, clinicians favoring CMV must hyperventilate (to suppress respiration), heavily sedate, or even paralyze their patients to avoid patient-ventilator interface complications. From another perspective, CMV strategies should replace 100% of a patient’s work of breathing (WOB). Patients allowed to breathe spontaneously during CMV frequently end up out of phase with the ventilator—that is, attempting to breathe when the ventilator is not in the inspiratory phase. Also known as dyssynchrony, out of phase breathing during CMV produces very high patient WOB. Nevertheless, assisted/spontaneous breathing strategies involve a work-sharing approach between patient and ventilator (23). Theoretically speaking, a work-sharing approach makes perfect sense; ideally, the ventilator functions to “unload” the WOB the patient cannot tolerate. Critically ill patients face an above-normal workload, primarily from their pulmonary disease process, and secondarily, from their artificial breathing apparatus, including the endotracheal tube (ET), breathing circuitry, humidifiers, and the ventilator (24,25). Unfortunately, there is a fatal flaw in the ventilator-patient work-sharing concept: until recently (26) we have not been able to find a reliable, readily available, easy-to-perform, and noninvasive methodology for determining just how much WOB our patients can actually tolerate (27–29), and this determination is absolutely crucial. If the ventilator off-loads too much work, the patient’s respiratory muscles are predisposed to atrophy. If the ventilator provides insufficient support, fatigue is likely. Either scenario can add unnecessary days, or even weeks, to the period of time patients require ventilatory support. Fatigued or weak patients make poor candidates for weaning and attempts at liberation from the ventilator; moreover, the risk for developing ventilator-associated pneumonia (VAP) correlates directly to the time spent on ventilatory support (30). For some, these concepts imply that hyperventilation, sedation, and paralysis predispose to atrophy, and that CMV should be used with extreme caution. Research suggests that the diaphragm, which evolved to contract without interruption from birth until death, begins to lose contractility shortly after initiating CMV (31); the loss of contractility is time dependent and continues to worsen as mechanical ventilation is prolonged (31).

**Ventilator Breaths—Defining Characteristics**

The idea of trying to classify each ventilator type, to better understand how specific ventilators interact with and affect physiology, remains as common a goal today as ever. Yet, today’s ventilators include so many modes and options, they are nearly impossible to classify. For a time, some tried to classify ventilator modes (32), but even this strategy is no longer reliable because many ventilators now incorporate dual-mode capabilities—the ability to switch modes within an individual ventilator breath. Instead of trying to classify ventilators, modes, or even sub-modes, it may be easier to develop and use a standardized set of terms and describe the breath types in use. This is possible since, regardless of ventilator or breath type, all ventilator breaths are delivered in four distinct phases or variables (Fig. 128.6).

**Phase or Control Variables**

Each ventilator breath must begin for some reason and at some specific moment in time. The physical change that initiates a breath is known as the trigger variable (labeled A, Fig. 128.6). Once a breath is triggered “on,” the ventilator must somehow
FIGURE 128.6. Pressure, flow, and volume curves for a mechanical ventilator breath. (A) The trigger variable is the physical characteristic used by the ventilator to initiate a mechanical inflation of the lungs. In this case, pressure falls before the breath starts; the represented ventilator breath is likely pressure triggered. (B) Control variable. For patient safety, the ventilator must precisely control an important aspect involved with inflating the patient’s lungs. For this breath, flow is held constant; the ventilator is described as flow controlled. (C) Cycle variable. Each mechanical breath must end such that the lungs are properly filled and then allow the patient to exhale. The physical characteristic that determines appropriate lung filling is the cycle variable. This example shows that the breath ending after the control variable (flow) has continued for a specific interval of time; this ventilator is time cycled. (D) Expiratory variable. Modern ventilators either control pressure during exhalation or do not. In this example, pressure returns to ambient (0 cm H₂O), so this ventilator has no operative expiratory variable.

Trigger Variable

Ventilators are triggered “on” by time, pressure, flow, or volume. Today’s ventilators, however, often depend on multiple trigger variables used sequentially. For example, with a trigger sensitivity set for 2 (actually, –2 cm H₂O), breath rate set to ten breaths per minute (bpm) and in the CMV mode, this ventilator has at least two trigger variables. First, breaths may be pressure-triggered when the ventilator is in the expiratory phase and baseline airway pressure is reduced by 2 cm H₂O or more. If, however, the patient makes no attempt to breathe, the CMV rate timer count reaches zero and a time-triggered breath results—exactly 6 seconds (s) after the last ventilator breath. This mode is known as assist-control (A/C), because patients can assist as often as they like, but if they stop breathing (assisting), the ventilator reverts to the predefined CMV rate. Another way ventilators employ multiple trigger variables is by using two sensors; for instance pressure and flow; triggering occurs in response to the variable’s threshold (pressure or flow) that is breached first.

Control Variable

Once a breath is triggered “on,” patient safety cannot be ensured unless the delivery of gas into the lungs is precisely controlled. Of the four potential physical changes (Table 128.1), only pressure and flow are used to any extent. Flow is emphasized to underscore an engineering issue: volume-controlled...
ventilators are actually flow controlled. There are several reasons: First, the integral of flow, with respect to time, is volume. Therefore, precisely controlling inspiratory flow ($V_I$) for a preset inspiratory time ($T_I$) produces an exact tidal volume ($V_T$) based on the following equation:

$$V_I \times T_I = V_T$$  \[1\]

Algebraically speaking, the operator can preset only two of these variables, in this case $V_I$ and $T_I$, the third variable ($V_T$) cannot be preset because it is a consequence of $V_I$ and $T_I$. Given a choice, however, most clinicians prefer to preset the $V_I$ and $V_T$; in this situation, $T_I$ becomes the consequential or resultant variable. In an effort to obviate this preference, most of today’s ventilators use an algebraic variant of equation 1:

$$V_I/T_I = V_T$$  \[2\]

This design allows operators to preset $V_I$ and $V_T$. Furthermore, since the operator actually predetermines $V_T$—referring to the ventilator as a volume controller, or as volume-controlled ventilation (VCV)—this is perfectly acceptable; this nomenclature will be used henceforth.

Volume-controlled strategies differ markedly from pressure-controlled ventilation (PCV). When we opt for VCV, our priorities are clear: we wish to prescribe (preset) $V_T$, $V_I$, and flow pattern. If we want these parameters reliably delivered, then airway pressure ($P_{aw}$) must not be restricted. When $P_{aw}$ is allowed to vary, $V_T$, $V_I$, and flow pattern are delivered, regardless of the patient’s pulmonary mechanics (Fig. 128.7). High peak inflation pressures (PIP) are a concern, so ventilators allow clinicians to preset a maximum safe level of $P_{aw}$; this setting, referred to as a high-pressure limit, functions as a cycle variable—that is, ending inspiration (or diverting gas flow) the moment $P_{aw}$ violates the established threshold. Keep in mind, though, cycling out a high-pressure limit truncates breath delivery, negates volume control, and reduces $V_T$.

Pressure-controlled strategies allow us to preset a desired $P_{aw}$ and $T_I$; conversely, $V_I$, $V_T$, and flow waveform cannot be predetermined. Pressure-controlled breaths always generate an exponentially decelerating flow pattern; the individual’s $C_{rs}$ and $R_{aw}$ determine the magnitude of $V_I$ and $V_T$ (Fig. 128.8) (33,34). Inasmuch as PCV does not control $V_T$, and preset pressure never varies, clinicians must ensure that PCV is carefully monitored; always carefully set low/high $V_T$ alarms (if available), as well as low/high minute ventilation alarms.

**Cycle Variable**

All four physical changes are commonly used for cycling ventilator breaths. Pressure cycling is common during intermittent positive pressure breathing (IPPB); flow cycling predominates during pressure support ventilation (PSV); and either time or volume is common during VCV. Without question, time is the most commonly used cycle variable, particularly if one remembers that with today’s VCVs, cycling occurs when $T_I$ lapses, not in response to volume.

**Expiratory Variable**

The expiratory phase is the least varied of the four; attempts at manipulating the expiratory phase variable have met with little success. Varying flow resistance (retard), negative end-expiratory pressure (NEEP), and PEEP have all been thoroughly tried and discarded. Compelling evidence, however, substantiates using continuous positive airway pressure (CPAP) and PEEP to restore or increase functional residual capacity (FRC) (35–37), reduce shunt and improve oxygenation (15–18), and reduce WOB (38).

**Classifying Breaths**

The four phase variables provide us with a method for classifying ventilator breaths that is easy to use and understand.
It makes sense to classify by breath behavior because today’s ventilators offer so many breath types. For instance, the breath depicted in Figure 128.6 is pressure-triggered, volume-controlled, and time or volume cycled; there is no exhalation phase variable. In Figures 128.7 and 128.8, the breaths are time triggered, volume controlled, and time or volume cycled, and time triggered, pressure controlled, and time cycled, respectively. Some ventilator modes like intermittent mandatory ventilation (IMV) or synchronized IMV (SIMV) allow two different breath types. Mandated breaths might be time triggered, volume controlled, and time cycled, whereas between scheduled breaths, spontaneous breaths might be pressure triggered, pressure controlled, and pressure cycled.

VENTILATOR DESIGN

Modern ICU ventilators are expensive and seemingly complex; yet, although the electronics may be complicated, the basic ventilator component configuration is simple and has changed very little over the last 20 years. In its simplest form, a mechanical ventilator requires only a few essential components (Fig. 128.9).

Power Sources

Pneumatics

Ventilators must have power. Most patients require at least some oxygen; this makes the energy stored within compressed oxygen a reliable and convenient power source. Gas-powered ventilators are called pneumatic. The powering gas source can be oxygen or compressed air, as long as the gas source is free of contaminants and debris and is dry. Hospital oxygen supplies virtually never pose contamination or water concerns; whether in bulk form or in cylinders, oxygen is certified clean and pure (99.99%). Compressed air sources are, however, a completely different matter. Compressors aspirate air from the environment; if aspirated air is contaminated, so too will be the compressed air. There have been instances of hospitals locating compressor intakes too close to parking lots and, on occasion, compressing exhaust gases along with air. Also, environmental air contains water vapor, some of which condenses and becomes liquid during the compression process; any and all water must be removed or it can cause serious damage to ventilators and other pneumatic equipment. Finally, most compressors use rapidly moving pistons or rotors to compress the air; operating at such high speed requires lubrication. Compressed air, for human consumption, should never involve using an oil-lubricated device. Small oil particles are compressed along with the air and can cause serious lung injury if inhaled.

Despite potential drawbacks associated with using compressed air sources, pneumatic ventilators offer several advantages, particularly when used for transport. For instance, pneumatic ventilators are always ready to go; they never require time-consuming recharging as battery-powered units do. Moreover, pneumatic ventilators use no expensive batteries, power supplies, or electric cables that can fail or must be periodically replaced. Furthermore, batteries often contain...
Chapter 128: Anatomy of Mechanical Ventilation

FIGURE 128.8. Response of pressure-controlled ventilation (PCV) to a sudden change in respiratory system compliance (C_{RS}). A: Compliance = 50 mL/cm H₂O. An airway pressure, flow, and tidal volume curve for a patient with this C_{RS} and receiving PCV. B: Compliance = 10 mL/cm H₂O. An airway pressure, flow, and tidal volume curve for the same patient as shown in panel A, except the patient’s C_{RS} is acutely reduced. Note pressure is essentially unaffected, but flow and volume are dramatically reduced as the far stiffer lungs respond to the same airway pressure with less flow and volume.

FIGURE 128.9. Schema of a basic ventilator. This schematic includes all of the major components necessary for ventilator operation. The logic component provides timing signals responsible for the inspiratory and expiratory phases. Ventilator logic must also synchronize the onset of each breath by the closing of the exhalation valve. Ventilator logic may be provided by fluidics, analog electronics, digital electronics (microprocessors), or pneumatics. All ventilators, regardless of simplicity, are either electrically powered (with or without battery backup) or pneumatically powered. None so far are powered by both modes.
extremely toxic components—lead, cadmium, lithium, and so forth—and must be properly disposed of or recycled, often at hospital expense. Pneumatic ventilators are also exceptionally robust; many pneumatic components will operate through many millions of actuations without failure. They are also reasonably priced and easy to maintain.

**Electric Power**

Electricity is cheap, reliable, and, in most countries, virtually ubiquitous. As a result, electricity powers most ventilators. Electrically powered units use alternating current (AC), AC converted to direct current (DC), battery, or some combination (Fig. 128.9). Unfortunately, ventilators are either pneumatically or electrically powered, never both. As a result, if power outages are likely, clinicians must consider their alternatives carefully: ventilators with battery backup are great, but will only operate for, at best, a few hours. Few if any of us have considered how ventilator-dependent patients would be ventilated in the event of an extended loss of electricity. This point is not simply a theoretical one, as just such a scenario occurred following Hurricane Katrina (39).

**Conventional Ventilator Logic**

All ventilators require some sort of logic to coordinate the timing of inhalation (I) and exhalation (E), as well as actuating the flow/volume delivery mechanism and the exhalation valve (Fig. 128.9).

Traditionally, ventilator logic involved pneumatics, standard electronics, fluidics, or some combination of these. To initiate and maintain inhalation, logic signals simultaneously activate both the flow/volume delivery system and the exhalation valve. At the same time, ventilator logic is responsible for timing or controlling inhalation and for monitoring breath delivery; the ventilator’s logic must be prepared to cycle the breath “off” if the high Paw limit is breached or when cycling criteria is met.

**Microprocessor-controlled Logic**

The first microprocessor-controlled ventilator was introduced in the early 1980s. Today, microprocessor-controlled logic dominates virtually every category of mechanical ventilation. Given that a microprocessor, or central processing unit (CPU), has virtually no influence on ventilator performance per se, it is not unreasonable to wonder why microprocessor ventilators are so popular. The answer is, in a nutshell, that they actuated. It is this memory that cues the CPU as to how often and when to act.

**Logical Expressions**

Microprocessors can also evaluate logical expressions or operate on the results of a relational question; logical operations follow the rules of Boolean algebra. For example, NOT true is false and NOT false is true. The AND function operates on two relational questions and requires that they both be true for the result to be true. That is, true AND true is true, but true AND false is false. The OR function also operates on two relational questions but requires only one of the questions to be true for the result to be true; true OR false is true, but false OR false is false. As an example, a CPU might evaluate the following two questions: Is exhaled V̇ < than inhaled V̇, divided by 2? AND is the operator setting for V̇ unchanged? If answered true AND true, then the ventilator might be instructed to warn clinicians of a low exhaled V̇ from there, the patient’s breathing circuit and ET cuff could then be quickly checked for leaks. With these simple building blocks, powerful algorithms can be devised that monitor all aspects of ventilator operations and make today’s mechanical ventilators safer than ever before.

**Computer Memory**

A CPU, no matter how powerful or fast, cannot function without memory. How could a CPU answer the relational question, Is x (Paw) greater than y (airway pressure limit)? if it couldn’t remember the value of y? Additionally, how would a CPU know when, or how often, to answer relational questions?

In our example, the value for x (Paw) varies continuously as a function of time, whereas the value for y (airway pressure limit) may remain constant; on the other hand, y will most likely vary from one patient to the next. Somehow, the CPU must be able to update the values for x and y as often as they change. This requires easily erasable memory, known as random-access memory (RAM). There is a caveat, however—easily erased means volatile and volatile means easily lost. For instance, valuable data might be lost the instant power is lost. As a result, ventilator CPUs cannot operate safely without battery backup to maintain critical data stored in RAM; without a patient’s exact data safely stored, the ventilator could malfunction, even if power was lost for an instant. Data stored in RAM is bidirectional; this means the CPU can store (write) information into RAM and read it later. Memory is limited, so the CPU must use its memory over and over again. Suppose the area used for storing a patient’s pressure limit (y) is already “occupied” and the operator changes the pressure limit; the CPU simply “writes over”—thereby erasing, the pre-existing pressure limit value.

Instructions how to use data stored in RAM and the sequence and timing of all functions carried out by the CPU reside in a different type of memory on known as read-only memory (ROM). This form of memory is nonvolatile and not easily altered. It is this memory that cues the CPU as to how often and when to evaluate the relational and logical operations. In fact, the entire sequence, or code, responsible for every conceivable ventilator function is stored in ROM. The use of ROM comes
TABLE 128.2
ADVANTAGES OF MICROPROCESSOR-CONTROLLED VENTILATORS

<table>
<thead>
<tr>
<th>General Versatility</th>
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<tbody>
<tr>
<td>■ Can provide virtually any desired mode of positive-pressure ventilation</td>
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<tr>
<td>■ Can provide a wide variety of inspiratory flow waveforms</td>
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<tr>
<td>■ Offers choice of cycling or trigger variable in many modes</td>
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<tr>
<td>■ Can be easily reprogrammed to meet changing trends in mechanical ventilation</td>
</tr>
<tr>
<td>■ Can ventilate adults, pediatric, and neonatal patients</td>
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<tr>
<td>■ Can provide adaptive modes of ventilation such as volume-targeted, pressure ventilation or proportional assist ventilation (PAV)</td>
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<tr>
<th>Monitoring</th>
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<tr>
<td>■ Provides real-time monitoring and alarms for various ventilatory parameters</td>
</tr>
<tr>
<td>■ Can measure and display lung-thorax compliance ($C_LT$), airway resistance ($R_{aw}$), plateau pressure, minute exhaled ventilation, auto-PEEP</td>
</tr>
<tr>
<td>■ Provides on-board computer memory that saves, for later retrieval, ventilation data for trend analysis</td>
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<th>Computer Correction and Safety</th>
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<tr>
<td>■ Can automatically correct for many internal variations that might affect prescribed tidal volume ($V_T$) or target pressure</td>
</tr>
<tr>
<td>■ Can automatically maintain the set inspiratory flow rate, waveform, and $V_T$, even when the patient's impedance ($C_LT$, $R_{aw}$) decreases or increases</td>
</tr>
<tr>
<td>■ Measures and displays tidal and minute volumes corrected to BTPS</td>
</tr>
<tr>
<td>■ Monitors all critical computer and patient parameters, declares an inoperative condition, terminates ventilation, opens the safety valve (to allow spontaneous breathing from the room), or begins a backup mode of ventilation any time a dangerous situation is detected</td>
</tr>
<tr>
<td>■ Saves and stores, for later retrieval and analysis, all errors, including patient alarms or other important issues detected during operation</td>
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<th>Display and Communications Capability</th>
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<tr>
<td>■ Can process and display any monitored data and important patient ventilation parameters</td>
</tr>
<tr>
<td>■ Can communicate and interface with remote monitors</td>
</tr>
<tr>
<td>■ Can communicate with separate microcomputers (personal computers) for monitoring and storage of data</td>
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<th>Repairs and Maintenance</th>
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<tr>
<td>■ Provides troubleshooting programs or extensive testing programs that pinpoint problems, facilitate repairs, and minimize downtime</td>
</tr>
<tr>
<td>■ Contains few moving parts; maintenance may involve only routine filter changes</td>
</tr>
<tr>
<td>■ Provides modular design and easily removable printed circuit boards that facilitate repair</td>
</tr>
</tbody>
</table>

PEEP, positive-end-expiratory pressure; BTPS, body temperature and pressure saturated.

with a caveat too: ROM is nonvolatile, but it is not impervious. Even the slightest change in a critical instruction could harm a patient. As a result, ventilator CPUs must have powerful “watchdog” systems that constantly evaluate every aspect of their behavior. Watchdogs always err on the side of safety—should they detect anything out of the ordinary, they immediately terminate CPU operation, protect the patient, and alert the operator of the malfunction—often referred to as a vent-inop. Once a bizarre or unusual behavior has been detected, manufacturers ensure that CPU integrity is verified before the ventilator will function again. Unfortunately, too often vent-inop conditions require technical assistance from a biomedical engineer or factory representative. To obviate such problems, engineers have tried two, or even three, CPUs, which are programmed to constantly evaluate each other. This strategy eliminates the need for watchdogs but, in the case of only two CPUs, does not eliminate the problem; when one CPU detects a problem, who decides which CPU is still functioning properly? With a three-CPU design, there is always a “referee”; the aberrant CPU, once detected, can be shut off leaving two CPUs to continue safely operating the ventilator until it can be safely replaced.

## Ventilator Control Systems

### Open Loop Control

Open loop ventilator designs (Fig. 128.10) are economical and straightforward but functionally limited. Ventilators using open loops offer VCV or PCV; they seldom provide both. Open loop systems are also not fault tolerant. For instance, suppose over time and with prolonged usage, a ventilator’s flow valve...
gradually drifts out of calibration. Now, consider that the signal designed to produce 0.75 L/s yields only 0.60 L/s, and \( V_T \) is preset to 0.75 L. This patient will receive a \( V_T \) no greater than 0.6 L, and the ventilator, even if CPU controlled, would have no way of detecting this problem.

**Closed Loop Control**

Closed loop, or feedback, designs (Fig. 128.11) are far more complex and expensive than open loop designs. In return, they deliver exceptional accuracy and automatically correct for many common failures and variances. Using the same preset \( V_T = 0.75 \) L and \( V_I = 0.75 \) L/s, a closed loop ventilator delivers the requested \( V_T \) even if the flow valve is no longer calibrated, thereby protecting the patient. Given the example above, ventilator logic opens the flow valve, expecting 0.75 L/s; yet, a flow sensor, located just downstream from the flow valve, measures the actual flow (0.6 L/s) and sends an electric signal—proportional to measured flow—to the comparator (Fig. 128.11). The comparator functions to analyze (electrically) the difference between the measured flow and actuating signals; if the signals are identical, nothing happens; if the signals vary, the comparator provides an output signal proportional in magnitude to the difference. The comparator’s output adds to, or removes from, the existing signal actuating the valve—in this case, the combined signals open the valve to produce a higher flow. Comparators function nearly instantaneously, so the flow valve’s output can be corrected repeatedly, as often as the valve’s response time and the programmed \( T_I \) allow. A response time of 10 ms allows 100 corrections in a \( T_I \) of 1 second, if necessary.

Closed loop feedback also corrects the ventilator outputs when affected by changing pulmonary impedance, different breathing circuits, and high-resistance humidifiers. Closed loop designs that incorporate flow and pressure sensors do not require separate valves for VCV and PCV. In this instance, a flow valve is either opened and a prescribed \( V_T \) delivered, or the valve is opened to provide an initial high flow, and a closed loop pressure algorithm maintains any desired target pressure by manipulating the valve’s output flow based on the target pressure.

Closed loop designs require accurate onboard flow and pressure sensors as well as sophisticated control algorithms; these are relatively costly and can be damaged by rough handling. For these reasons, transport ventilators often incorporate open loop designs.

**Microprocessors and Closed Loop Control**

The first CPU-powered ventilators were too slow to perform all of the tasks involved in operating the ventilator and provide the corrected signals required for closed loop control. To maintain accuracy and speed, the first generation of CPU-powered ventilators combined digital logic with analog, closed loop control systems. In contrast, today’s CPUs perform billions of operations per second, leaving adequate time for the CPU to provide “corrected” signals necessary for closed loop control (Fig. 128.12).

Microprocessors operate using digital (D) signals, but most of our real-world hardware (valves, sensors, transducers) require analog (A) signals. It follows that for a CPU to actuate a valve, a digital signal from the CPU must first be converted to analog; this takes place in a D-to-A signal processing chip (Fig. 128.12). Analog information, such as measured flow signals, must be similarly converted A to D before the CPU can
FIGURE 128.11. Schema of closed loop ventilator control. Ventilator reliability and accuracy is vastly improved by measuring the actual output (flow), comparing the measured to desired, and correcting the actuating signal by the difference. At the onset of each breath, a signal from the ventilator’s logic actuates (opens) the output valve. The resultant output (flow) is measured immediately by a flow sensor positioned downstream. The flow sensor converts measured gas flow into an analog electric signal, which is routed to one side of an electronic comparator. The actuating signal (actual) from the logic element is fed into the other side of the comparator, where it is compared to the measured signal. If the two signals differ, the comparator adds (or subtracts) an amount of electricity proportional to the signal difference to (or from) the actuating signal. The entire loop requires only about 10 msec to complete; that means the actual signal could theoretically be corrected as many as 100 times in a typical mechanical breath lasting just 1 sec. Normally, however, it requires only a few iterations before the measured and desired signals are identical.

CONVENTIONAL MECHANICAL VENTILATORY TECHNIQUES

Compliance and Resistance—The End-inspiratory Plateau

Operational Principles

The terms postinflation hold, end-inspiratory pause, and end-inspiratory plateau (EIP) all refer to the same ventilator routine; the instant VT delivery is complete, the ventilator stops gas flow but does not allow the patient to exhale until a specified period of time, the EIP, elapses (Fig. 128.13). The EIP is considered part of TI because the VT volume remains in the lungs and the patient does not exhale until the EIP is complete; ventilators often allow plateaus as long as 2 seconds. Although this may not seem excessive, when combined with the existing TI, EIPs are often long enough to adversely impact hemodynamics and are poorly tolerated by spontaneously breathing patients.

Clinical Applications

An end-inspiratory plateau has been advocated as a method to improve the distribution of inhaled gases, thereby decreasing Vd/Vt and PaCO₂ (40). Theoretically, this makes sense; if inhalation was long enough, gas redistribution into slow-filling spaces would improve overall distribution (41). Gas redistribution during EIP is thought to result secondary to collateral ventilation and Pendelluft flow.

Collateral ventilation occurs when gas enters the alveoli from adjacent alveoli through channels in the alveolar walls (pores of Kohn) or through cross-communications between bronchioles (Lambert canals). Pendelluft flow occurs when, during EIP, volume from fast-filling spaces redistributes into
slow-filling spaces. Such gas flow is caused by regional pressure gradients that arise as a consequence of maldistribution secondary to positive pressure inflation.

The EIP is seldom used to improve distribution, but rather to determine $C_{RS}$ and $R_{aw}$. During the plateau time, as gas flow ceases, the flow-resistive component of PIP disappears. The remaining pressure—the plateau pressure—also reflects the static elastic recoil pressure (ERP) of the lungs. Exhaled $V_t$, PIP, and ERP are used in determining the patient’s $C_{RS}$ and $R_{aw}$ (Table 128.3). These measurements are often performed routinely to assess the patient’s progress or to gauge the response to bronchodilators.

**Inspiratory Flow Waveforms**

Before the advent of microprocessor-powered ventilators, different ventilator brands delivered gas flow using a wide variety of methodologies: pistons, injectors, bellows, solenoids, and so forth. Each flow-generating technique produced a different inspiratory flow pattern: square or constant (Fig. 128.14A), sinusoidal, decelerating (Fig. 128.14B), accelerating. Clinicians immediately began to wonder which waveform was best, or, could matching specific waveforms with specific pulmonary conditions make a difference? To this day, these questions remain essentially unresolved. Some tried various waveforms and found little or no difference in the distribution of ventilation (42). Other studies, modeling multiple lung compartments with different $R_{aw}$, showed improved distribution with the decelerating waveform compared to others (43,44). Clinical reports confirmed the utility of a decelerating pattern (45–47). In one investigation, $V_t$, $T_i$, I:E ratio, and ventilator rate were held constant. Compared to the constant-flow pattern, the decelerating waveform significantly reduced patient PIP, PaCO$_2$, $V_{D}/V_{T}$ ratio, and alveolar-to-arterial oxygen pressure gradient.

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**FIGURE 128.12.** Schema of a microprocessor-controlled closed loop control system. First-generation microprocessor-controlled ventilators combined digital (D) signals converted to analog (A) with an analog closed loop system. This approach was necessary because digital control of closed loop feedback added several time-consuming steps: corrected signals had to be converted D to A before they could operate ventilator valves, and the measured signal had to be converted A to D before the microprocessor could compare it to desired and determine an appropriate correction. Unfortunately, microprocessors available at the time were simply not fast enough to adequately monitor lung inflation and provide corrected closed loop signals. Today’s microprocessors easily perform billions of operations per second, and most second- or third-generation microprocessor-controlled ventilators provide closed loop control using only digital signal processing.
Chapter 128: Anatomy of Mechanical Ventilation

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FIGURE 128.13. Compliance and resistance determination in the ventilator patient. Respiratory system compliance (CRS) and airway resistance (Raw) determination by end-inspiratory pause (EIP) requires volume-controlled mode, square flow pattern, inspiratory flow rate (V̇I) of exactly 1 liter (L)/second (s) (or 60 L/minute), and an EIP of ≥0.25 second. In this example, the peak inflation pressure (PIP) reaches 25 cm H₂O the instant flow ceases. After the preset tidal volume (VT) is delivered, the EIP begins, that is, gas flow from the ventilator ceases and the patient is not permitted to exhale. During the EIP, pressure equilibrates between the lungs and breathing circuit and elastic recoil pressure (ERP) of the lungs can be measured at the airway opening. The greater the difference between a patient's PIP and ERP, the greater the Raw. Compliance is computed by dividing VT (0.65 L) by measured ERP minus the baseline pressure (0 cm H₂O), and is given in units of L/cm H₂O or mL/cm H₂O. Resistance is computed as PIP minus ERP divided by V̇I (must be 1 L/s) and is stated in units of cm H₂O/L/s.

TABLE 128.3

MEASUREMENT OF COMPLIANCE AND AIRWAY RESISTANCE

<table>
<thead>
<tr>
<th>Definition:</th>
<th>End-inspiratory pause may be used to differentiate dynamic (Cdyn) (L/cm H₂O) from static lung-thorax compliance (Cst) (L/cm H₂O) and to determine airway resistance (Raw) (cm H₂O/L/s).</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Dynamic Cdyn = VT/IPP – baseline airway pressure</td>
<td>Where: VT = exhaled tidal volume (L) IPP = peak inflation pressure (cm H₂O) Baseline airway pressure = atmospheric pressure, continuous positive airway pressure (CPAP) (cm H₂O), or positive end-expiratory pressure (PEEP) (cm H₂O)  e.g., Cdyn = 0.65 L/(25 cm H₂O – 0 cm H₂O) = 0.026 L/cm H₂O</td>
</tr>
<tr>
<td>2. Static Cst = VT/ERP – baseline airway pressure</td>
<td>Where: ERP = static elastic recoil pressure of the respiratory system (cm H₂O)  e.g., Cst = 0.65 L/(17 cm H₂O – 0 cm H₂O) = 0.038 L/cm H₂O</td>
</tr>
<tr>
<td>3. Raw = (PIP – ERP) / V̇I</td>
<td>Where: V̇I = inspiratory flow rate* (L/s)  e.g., Raw = (25 cm H₂O – 17 cm H₂O) × 1 L/s = 8 cm H₂O/L/Ls</td>
</tr>
</tbody>
</table>

*The selected inspiratory flow pattern must be constant (square).
FIGURE 128.14. Differences in peak inflation pressure (PIP) when using a square or decelerating inspiratory flow (V_{I}) waveform. A: An airway pressure (P_{aw}), flow, and volume curve for a typical volume-controlled breath delivered using a square V_{I} waveform. Following breath delivery, an end-inspiratory plateau (EIP) terminates gas flow and allows pressure to equilibrate between the lungs and airway opening; at equilibration, P_{aw} reflects the elastic recoil pressure (ERP) of the respiratory system; that is, of the lungs and thorax combined. Note that the PIP of this breath is nearly 25 cm H\textsubscript{2}O. B: An airway pressure, flow, and volume curve for the same breath, delivered to the same patient, except using a decelerating V_{I} waveform in this instance. Again, following breath delivery, an EIP allows determination of ERP. Note that the PIP of this breath is 8 cm H\textsubscript{2}O lower than for the square waveform; nevertheless, the measured ERP (18 cm H\textsubscript{2}O) is exactly the same as that using the square flow pattern. This occurs because a decelerating flow pattern reduces gas flow to near zero (as during an EIP) before the breath cycles “off” and the EIP begins.

P(\text{A}−\text{a})O_{2} (46). However, mean P_{aw} was significantly greater, predisposing to adverse hemodynamic effects.

In addition to the potential to improve distribution, decelerating waveforms significantly reduce PIP, especially when contrasted to square (Fig. 128.14) or accelerating patterns. Some clinicians opt for a decelerating pattern, believing the lower PIPs may help protect their patients from ventilator-induced lung injury (VILI). This logic is flawed; the pulmonary edema and lung injury, often seen during mechanical ventilation, are now believed to be the consequence of excessive volume (volutrauma) rather than excessive pressure (barotrauma) (48,49). Furthermore, the main determinant of volutrauma appears to be end-inspiratory lung volume (the overall lung distention) rather than the FRC (which depends on PEEP) (19,37). Based on this information, reducing PIP by waveform selection offers no advantage; patients supported with VCV receive the same V_{T}, and therefore overall lung expansion, regardless of waveform (Fig. 128.14).

Inspiratory flow waveforms impact yet another aspect of mechanical ventilation: patient-ventilator synchrony. During any form of patient-triggered mechanical ventilation, the spontaneous inspiratory effort may extend well into mechanical inflation. If, at any point, spontaneous flow demand exceeds the preset V_{I}, flow starvation results. Flow starvation distorts pressure patterns and exaggerates WOB. Decelerating flow patterns often provide initial V_{I} spontaneous sufficient to meet patient demand; if at any point, however, V_{I} is reduced below that the patient is demanding, flow starvation follows. Often, flow starvation of this nature can be managed by simply switching from a decelerating to a constant waveform to maintain a higher V_{I} throughout the breath (Fig. 128.15).

Controlled Ventilation

Operational Principles

Mechanical ventilation is indicated when spontaneous ventilation is inadequate or absent. Physiologically, this means the patient is incapable of maintaining acceptable PaCO\textsubscript{2} and arterial pH levels. CMV delivers an operator-selected breathing rate, V_{T}, peak V_{I}, and flow pattern; CMV operates completely independent of patient efforts to breathe (Fig. 128.16A). When patients attempt to breathe during CMV, the result can be
violent patient-ventilator dysynchrony. Consequently, patients supported by CMV often require hyperventilation to blunt the normal stimulus to breathe, heavy sedation, or even pharmacologic paralysis.

Clinical Applications
Indications for CMV and CMV with PEEP (Fig. 128.16B) include apnea, ARDS, central nervous system depression, drug overdose, or neuromuscular dysfunction. For this subset of patients, an accidental disconnection from the ventilator or a ventilator failure is life threatening. Thus, CMV requires vigilant monitoring and carefully set disconnect and failure-to-cycle alarms.

Patient-triggered Ventilation
Operational Principles
There are two basic forms of patient-triggered breaths: mechanical and spontaneous. Patient-triggered mechanical breaths (Fig. 128.16C) are nearly identical to CMV breaths in that the VT, peak VI, and flow pattern are all operator selected; the only difference is assisted mechanical ventilation (AMV) which requires that the patient trigger each and every breath. Thus, when supported by AMV, patients must not experience an acute apneic episode; otherwise, all ventilation ceases. Concern for this possibility explains why so few physicians opted to use AMV before ventilators came equipped with backup ventilator modes. For AMV, a backup mode might allow the operator to select desired CMV settings which the ventilator defaults to and uses in the event of apnea.

Clinical Applications
Patient-triggered ventilation is considered a vital link between CMV and extubation. In theory, it allows the patient to breathe spontaneously in preparation for removal of the ventilator. Spontaneous breathing is never consistent, however, meaning AMV is extraordinarily difficult to optimize to a patient’s efforts. If the preset VT, VT, or both are too high, patient WOB falls to essentially zero; if they are insufficient or the patient becomes dysynchronous, WOB skyrockets.
The use of dual-trigger variables allowed clinicians to safely use AMV well before the incorporation of backup modes. Patient-triggered support with a time-triggered CMV backup was coined assist-controlled ventilation (A/C) (Fig. 128.16D). When using A/C, the operator sets a minimum acceptable breathing rate using the CMV rate control, and adjusts trigger sensitivity (usually pressure). As with AMV, the patient triggers breaths, as often as desired, by breaching the trigger threshold. If the patient stops breathing, however, or the spontaneous breathing rate drops below the preset minimum, time-triggered CMV intercedes until a clinician investigates or spontaneous breathing rate is restored. The simple concept of providing a continuous flow, regardless of which variable triggered the breath, the CMV rate timing clock is restarted. At this point, if a spontaneous effort triggers a breath before the CMV timer lapses, the breath is pressure triggered and the CMV clock restarted. If the patient fails to breathe or cannot spontaneously trigger, the CMV clock will run down and the next breath will be time triggered. Using the A/C strategy, patients may breathe as rapidly as they desire, but never at a rate lower than the CMV mechanical rate setting. As the ventilator patient dysynchrony is very common during CMV, AMV, and A/C modes of ventilation. These modes all require preset VT and flow waveforms while patient breathing patterns frequently vary. When patient flow demand exceeds that provided by the ventilator, the WOB imposed on the patient may become excessive (32). Recent trends toward smaller mechanical VTs have exacerbated the issue (53). Patients allowed to breathe spontaneously while receiving low-VT lung-protective ventilation will likely suffer from both flow and volume starvation (Fig. 128.17). The additional WOB can be enormous. Clinicians facing this situation are left with few palatable options: increasing VT and PEEP predispose to ventilator-induced lung injury; yet, continued sedation or paralysis will undoubtedly complicate or prolong the weaning process.

Operational Principles

With spontaneous breathing rates often >100 breaths per minute, infants with hyaline membrane disease continue to be the best early efforts at patient-ventilator synchronisation. The simple concept of providing a continuous flow, from which these babies could breathe spontaneously between

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**CMV Backup**

Assisted mechanical ventilation and CMV, used alone or in conjunction, predispose to hyperinflation, ventilator-induced V/Q abnormalities, and excessive WOB. These untoward effects are related to anxiety-driven ventilator-patient dysynchrony and maldistribution of ventilation, respectively. A disproportionate amount of the VT is delivered anteriorly to non-dependent lung regions with decreased perfusion when patients are in the supine position (50). Conversely, spontaneous breathing tends to promote better V/Q distribution. Some studies have demonstrated that VT increases during CMV and AMV, with or without PEEP (51,52). Downs and Mitchell (51) reported that increases in VT were related to the rate of mechanical breathing, regardless of the ventilatory pattern, mode, and whether or not PEEP was used.

As mentioned, ventilator-patient dysynchrony is very common during CMV, AMV, and A/C modes of ventilation. These modes all require preset VT and flow waveforms while patient breathing patterns frequently vary. When patient flow demand exceeds that provided by the ventilator, the WOB imposed on the patient may become excessive (22). Recent trends toward smaller mechanical VTs have exacerbated the issue (53). Patients allowed to breathe spontaneously while receiving low-VT lung-protective ventilation will likely suffer from both flow and volume starvation (Fig. 128.17); the additional WOB can be enormous. Clinicians facing this situation are left with few palatable options: increasing VT and PEEP predispose to ventilator-induced lung injury; yet, continued sedation or paralysis will undoubtedly complicate or prolong the weaning process.

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**Intermittent Mandatory Ventilation**

With spontaneous breathing rates often >100 breaths per minute, infants with hyaline membrane disease continue to be the best early efforts at patient-ventilator synchronisation. The simple concept of providing a continuous flow, from which these babies could breathe spontaneously between
mandated mechanical breaths (54–56), resulted in a new ventilatory mode referred to as intermittent mandatory ventilation (IMV). After proving its utility on neonates, IMV was later advocated for adults, especially those difficult to wean from mechanical ventilation (57). Neonatal IMV systems provided a continuous flow of gas throughout the respiratory cycle; tidal ventilation was accomplished by simply closing the exhalation valve and diverting flow into the lungs (54,55).

When applying IMV, the operator preselects the desired ventilator rate, tidal volume ($V_T$), and flow pattern; in this aspect, it does not differ from CMV. With IMV, however, the patient breathes as often as desired between sequential positive pressure breaths. In theory, a designed and adjusted IMV system provides an unrestricted gas flow equal to or greater than the patient’s peak $V_I$ demand; these conditions minimize WOB. Early IMV systems were “homemade” (58,59), leading some to opine that reported failures due to poor system design, not were to IMV, per se (60).

**Clinical Applications**

Most clinicians set an IMV rate sufficient to complement the patient’s own spontaneous breathing and still maintain an acceptable alveolar ventilation, $P_{aCO_2}$, and pH. Mandated breathing rates of 4 to 6 breaths per minute were popular because they provided an adequate $V_A$ in the event of apnea. When patients with pre-existing COPD were managed with IMV and compared to others managed with patient-triggered ventilation, IMV offered better control of $P_{aCO_2}$ and pH (61,62). When IMV is combined with CPAP, the cardiopulmonary effects are improved compared to CMV or A/C modes of ventilation; as a result, IMV makes it possible to maintain higher mean expiratory positive pressures with fewer deleterious effects on venous return and cardiac output (63,64).

**Potential Problems**

IMV and CMV share many similarities, such as preset $V_T$, $V_I$, and flow waveform. Furthermore, with both modes, all mandated breaths are time triggered, unresponsive to any patient effort. During IMV, however, the patient breathes freely, as desired. It follows that, occasionally, a time-triggered breath might be delivered at or near end-inhalation for a spontaneous breath (breath-stacking); if the stacked volumes were large enough, they would predispose to elevated PIP, mean $P_{aCO_2}$, and cardiovascular embarrassment; without any compelling evidence validating this hypothesis, many clinicians nevertheless completely avoided IMV.

The concept of providing an unimpeded continuous gas flow equal to or exceeding the patient’s peak $V_I$ introduced an unexpected consequence to developing safe and effective IMV systems: finding a humidifier capable of the task. Poulton and Downs (65) studied the issue and reported that with the exception of the Bird humidifier, the others tested either imposed significant flow resistance—which would certainly affect patient WOB—or failed to provide sufficient humidity at the high flow rates IMV often required.

Breathing spontaneously requires patient WOB; the amount of work required, however, depends on a number of physiologic and external apparatus variables such as $C_{RS}$, $R_{aw}$,
effort required to trigger breaths (if needed), available flow to that demanded, ET size, exhalation valve performance, and the rate and duration of the pressure drop experienced during inhalation. Downs (60) and others (66) suggested that properly adjusted continuous-flow IMV systems minimized the apparatus portion of WOB; several studies comparing continuous-flow to demand-flow valves corroborated this theory (67–69). Nevertheless, continuous-flow IMV systems were never popular—they were bulky, noisy, and difficult to adequately humidify; required frequent readjustment; and wasted massive amounts of gas. Clinicians therefore prevailed on manufacturers to refine and improve their demand valves or demand-flow systems. Gradually, demand-flow system performance improved; by 1985, Katz et al. (70) reported comparing seven demand-flow CPAP systems against a continuous-flow system (at 60 L/minute) and reported that some demand-flow systems performed as well or better than the continuous-flow system.

**Synchronized Intermittent Mandatory Ventilation**

**Operational Principles**

Clinical concerns about the potential for breath-stacking stimulated the development of synchronized intermittent mandatory ventilation (SIMV), which allowed patient-triggered mandated breaths. That is, the A/C mode of ventilation, SIMV used two trigger variables, usually pressure and time; if the patient failed to trigger, an IMV breath was time triggered when the rate clock reached zero. Operators established SIMV by setting patient rate, \( V_L \), \( V_T \), \( V_E \), \( P_f \), or cardiovascular effects. These benefits were not all easily substantiated. Shapiro et al. (71) reported that mean intrapleural pressure was substantially lower with SIMV than with IMV in normal volunteers. Hasten et al. (72) compared SIMV and IMV in 25 critically ill patients; they found that although PIP was higher, blood pressure, cardiac output, stroke index, central venous pressure, and pulmonary artery pressure did not differ significantly. In a similar study, Heenan et al. (73) studied anesthetized, near-drowned dogs ventilated with IMV or SIMV. Again, no differences were noted with respect to cardiac output, stroke volume, intrapleural pressure, and intrapulmonary shunt. Mean airway pressure and PIP were significantly elevated with IMV, and some breath-stacking occurred, but the authors noted no adverse effects from these differences. Based on these data, SIMV seems to offer little clinical advantage compared to IMV with CPAP. There is a logical explanation for these findings, however: spontaneously breathing critically ill patients seldom inspire large spontaneous \( V_T \) from CPAP systems. In fact, the high-rate/low-aw \( V_T \) breathing pattern is extremely common; indeed, a high breathing frequency (t) to \( V_T \) or \( P_f \); ratios correlates well with extubation success (74). So, IMV breath-stacking, when it occurs, is unlikely to result in dangerously high spontaneous \( V_T \), PIP, or cardiovascular interference.

Modern ventilators all incorporate SIMV, and in the United States, SIMV with pressure support ventilation (PSV) is now the preferred ventilatory mode (75). When combined with PSV, synchronization is an absolute must. Unlike assisted or unassisted spontaneous breaths taken from a CPAP system, PSV breaths are frequently large—as large as or larger than the mandated \( V_T \). During SIMV with PSV, if a mandatory breath stacks on to relatively large PSV breath, a very large and dangerous \( V_T \) would result.

**Potential Problems**

The two trigger variables used with SIMV cannot be programmed to function as flawlessly as during A/C ventilation. For instance, if the SIMV rate is 8 breaths per minute, but the patient is breathing spontaneously at a rate varying between 30 and 40 breaths per minute, exactly which of the 30 to 40 breaths should be selected for synchronization? If every fifth breath is selected, and the breath rate is 40 breaths per minute, the patient gets exactly 8 mandated breaths per minute; but what happens if the spontaneous rate suddenly drops to 30 breaths per minute? To overcome this dilemma, ventilator logic divides 60 seconds by the preset SIMV rate—in this case 60/8 = 7.5 seconds—and then opens a new timing window every 7.5 seconds. The ventilator is programmed to synchronize to the first patient effort in each timing window. If the patient makes no effort, the breath is delivered at the end of the window; this results in the desired SIMV rate—at least most of the time. Problems still occur, however. During apnea, a bizarre pattern often results. Since the patient fails to make an effort, a mandated breath occurs at the very end of a timing window; the next successive time window opens immediately, even before the just-delivered mandated breath can be exhaled. Exhalation therefore proceeds well into the next timing window. If at the end of this exhalation, \( P_{aw} \) falls a few cm H2O below baseline pressure as often happens, the ventilator often mistakes this pressure drop for the first patient effort in the timing window and—delivers a second successive SIMV breath, a phenomenon often referred to as autotriggering. The strange behavior does not end there. Since the ventilator has already “synchronized” to the first breath in the present timing window, and because the patient is apneic, it will not trigger again until the next successive timing window expires. A breathing pattern consisting of two consecutive mandated breaths, followed by 15 seconds without a mandated breath, followed by two consecutive mandated breaths repeats over and over (Fig. 128.18). Despite the bizarre appearance, the patient actually receives the eight mandated breaths per minute, just not in the pattern expected. Autotriggering is easily rectified by increasing trigger sensitivity to a point just below the pressure drop noted at end-exhalation. There is a caveat to this solution, however. Increasing trigger sensitivity makes triggering more difficult. Therefore, the operator must be sure to readjust the sensitivity as soon as spontaneous breathing activity resumes.

**Pressure Support Ventilation**

**Operational Principles**

Pressure support ventilation likely developed as a method to counteract the additional WOB imposed by easily, poorly designed demand valves and demand-flow systems (28).

As demand systems improved, the WOB imposed by the breathing apparatus approached zero, still, patients often
Chapter 128: Anatomy of Mechanical Ventilation

**Time-Triggered Breath at End of Timing Window**

**Trigger Sensitivity**

**Timing Window 1**

**Pressure (cm H₂O)**

**Time (s)**

**A. Too Sensitive**

**B. Increased Sensitivity**

FIGURE 128.18. Synchronized intermittent mandatory ventilation (SIMV) and autotriggering during apnea. **A: Too sensitive**. An airway pressure curve demonstrating how trigger sensitivity set for normal breathing may be too sensitive during apnea. SIMV modes are programmed to synchronize to the first patient effort in each timing window. When no effort occurs, as during apnea, the ventilator must time trigger at the end of the timing window. If sensitivity is set too low (1–2 cm H₂O), exhalation from the previous breath may appear as the first effort in the next timing window and autotrigger a breath. This scenario results in a bizarre breathing pattern: two breaths in succession, followed by a very long pause and two breaths in succession; the pattern then repeats. Interestingly, this pattern delivers the preset breathing rate, but not in the sequence expected. **B: Increased sensitivity**. An airway pressure curve of the same apneic patient, except that trigger sensitivity is increased to a pressure lower than that occurring during exhalation; this eliminates autotriggering and results in a more uniform pattern of ventilation.

Pressure support has replaced CPAP as the spontaneous breathing mode used during SIMV. PSV helps to reduce WOB by partially unloading the respiratory muscles. Approaches to the use of PSV vary; some advocate just enough PSV to zero-out any additional WOB imposed by the breathing apparatus and ET (25,27,29,78), whereas others try to neutralize both the imposed WOB and some of the physiologic WOB—enough to provide comfort and avoid fatigue. With backup modes for safety, some use PSV as a stand-alone (79,80).

**Potential Problems**

Despite its promise, PSV may have created more problems than it solved. If set too low, the patient continues to struggle and may fatigue; set too high, the patient does essentially...
no work and is predisposed to disuse atrophy of the respiratory muscles. Contounding matters are two unresolved problems: first, no one has developed a noninvasive, easy-to-use, reliable method for determining the proper PSV level; and second, patient demands vary considerably, and a single PSV level cannot possibly meet every conceivable patient demand level. Studies suggest that if PSV levels are managed around the clock, patients spend significantly less time receiving ventilatory support (81).

**Pressure-controlled Ventilation**

**Operational Principles**

As outlined in the classification section, in today's ICU, virtually all mechanical breaths are either volume or pressure controlled. Pressure-controlled breaths can be used for CMV, A/C, mandated SIMV breaths, as well as for pressure-controlled inverse-ratio ventilation (PC-IRV). Operators set the desired ventilator rate, \( T_i \), target pressure (above baseline), and, for A/C or SIMV, trigger sensitivity. After a PCV breath is triggered, flow and pressure rise rapidly to the preset pressure; on reaching pressure, flow decelerates as needed to maintain that pressure until the preset \( T_e \) elapses. For patient-triggered modes, the same rise time control used for PSV adjusts the initial flow rate and pressure rise in PCV; on occasion, an appropriate adjustment of the rise time control significantly enhances patient comfort. For those acutely ill with ARDS, PC-IRV is sometimes applied. Using PC-IRV, however, requires sedation and sometimes neuromuscular blockade; awake and alert patients seldom tolerate the extended \( T_e \) intervals—with \( T_e \) ratios up to 4:1—used during PC-IRV without "fighting" the ventilator.

**Clinical Applications**

Clinicians opt for PCV instead of VCV for three reasons or some combination thereof: (i) to provide higher initial and average \( V_t \) for patients breathing spontaneously during mechani-cal breaths; (ii) to control PIP and limit the possibility of VILI; and/or (iii) to use PC-IRV. Although several studies report improvements in oxygenation at lower PIP using PC-IRV with infants (82,83), its use in adults (84,85) remains limited. Clearly, PC-IRV elevates mean \( P_{aw} \), which in turn raises FRC. Propos-ents of PC-IRV, however, claimed similar improvements in oxygenation to those seen using VCV but at much lower CPAP or PEEP levels. Using PCV as a method to control PIP and minimize the risk of VILI is an interesting approach. However, if the culprit in VILI is the end-inspiratory lung volume and not the PIP, then the approach is terribly flawed. That is, during PCV, the patient is free to interact with the ventilator. If the patient inhales deeply during any pressure-controlled breath, the resul-tant spontaneous \( V_t \) may be quite large. Thus, while PCV may control \( P_{aw} \) precisely, it fails to limit \( V_t \) to a safe level. Along the same lines, using PCV to provide higher peak and average \( V_t \) makes sense only if the attending clinician is comfortable with the potential for a large range in spontaneous \( V_t \).

**Potential Problems**

The defining characteristics of PCV are high peak \( V_t \) decelerating flow waveform, and no control over \( V_t \). At one extreme, PCV continues to control pressure even if the ET occludes and \( V_t \) drops to zero; at the other extreme, a vigorous patient effort often produces very large spontaneous \( V_t \), especially when compared to the \( V_t \) delivered with no effort. Clinicians using PCV must carefully set high and low \( V_t \) and minute ven-tilation alarms to avoid these problems, as well as keep a close eye on ventilator graphics, if available. Often, these problems are visually apparent well before adverse responses.

For a time, PC-IRV was popular; this was, in part, due to claims that the mode increased oxygenation and reduced PIP without using high PEEP or CPAP. In retrospect, these au-thors were probably mistaken. Two editorials questioned these claims by suggesting that the benefits seen with PC-IRV were due to PEEP—that is, undetected auto-PEEP (86,87). Auto-PEEP results from incomplete exhalation; gas volume trapped in the lungs from the proceeding breath exerts an elastic press-sure similar to PEEP or CPAP—thus the name auto-PEEP. Gas trapping results for two primary reasons: incomplete time for exhalation or premature airway collapse. Inverse ratios, as used in PC-IRV, reduce exhalation time, often significantly; hence PC-IRV predisposes to air trapping or auto-PEEP. Furthermore, during normal exhalation, auto-PEEP exists only in the lungs, not in the breathing circuit, making it difficult to detect; for this reason, some have even called it occult-PEEP (88). Auto-PEEP can, however, be estimated at the bedside, if one suspects its existence (88). A patient's total PEEP must take into consider-ation auto-PEEP:

\[
\text{total PEEP} = \text{applied PEEP} + \text{auto-PEEP}
\]

Once this concept was understood, clinicians began routinely monitoring for auto-PEEP. Of note, interest in PC-IRV waned almost concomitantly, providing strong circumstantial ev-dence that auto-PEEP was indeed responsible, in part, for PC-IRV's initial success in adults. Today's mechanical ventilators measure auto-PEEP “on request,” assuming the patient makes no effort to breathe during the measurement.

**SPONTANEOUS BREATHING**

**CPAP and Spontaneous PEEP**

**Operational Principles**

Given the improvements in oxygenation, \( V_t/Q, C_H \), and WOB attributed to using PEEP or CPAP during mechanical ventilation (14–16,68,89), it seems logical to use them as stand-alone modes. With CPAP, both inspiratory and expiratory pressures remain positive. As a practical matter, the inspiratory pressures are lower than expiratory during CPAP. This occurs for three reasons: (i) during inspiration, the removal of gas from the continuous flow by the patient causes pressure to fall; (ii) trig-gering a demand CPAP system generally requires that pressure fall; (iii) the CPAP system cannot meet the patient's inspiratory flow demands. By definition, spontaneous PEEP requires that the patient reduce \( P_{aw} \) to zero or less during the inspiratory phase; \( P_{aw} \) returns to the PEEP level during exhalation. Spontaneous PEEP differs from CPAP in that the large pressure fluctua-tions during inspiration impose a significantly greater WOB on the patient (90) (Fig. 128.19). When tolerated, however, the same large pressure swings improve venous return and cardiac output (91). Conversely, CPAP reduces patient WOB, but at the expense of cardiac output.
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Figure 128.19. Work imposed by spontaneous continuous positive airway pressure (CPAP) or positive end-expiratory pressure (PEEP) breathing systems. The inspiratory work of breathing (WOB\textsubscript{I}) encountered by patients breathing spontaneously is affected by the type of breathing system. Additional workloads imposed on spontaneously breathing patients are determined by measuring the area under the curve obtained by plotting airway pressure (P\textsubscript{aw}) versus volume (V\textsubscript{T}) loops taken at the proximal airway (i.e., at the endotracheal tube opening). End-expiratory pressure is 10 cm H\textsubscript{2}O, while the simulated patient effort is identical for both systems. The additional WOB\textsubscript{I} (cross-hatched areas) is computed from the area within the P\textsubscript{aw}/V\textsubscript{T} loop while P\textsubscript{aw} remains below the starting point (10 cm H\textsubscript{2}O). With the CPAP system, P\textsubscript{aw} initially falls to 7 cm H\textsubscript{2}O (during triggering) but quickly returns to 9 cm H\textsubscript{2}O for the remainder of inspiration; with the spontaneous PEEP system, the same patient effort initially reduces P\textsubscript{aw} to −2 cm H\textsubscript{2}O, but it quickly returns to 0 cm H\textsubscript{2}O. The computed WOB\textsubscript{I} for the CPAP system is approximately 0.1 joules (J) and for the PEEP system 0.48 J, an increase of 480%. The expiratory work of breathing (WOB\textsubscript{E}) is generally a nonfactor; the energy required to exhale is provided by the energy released as patients' highly elastic lungs, which were stretched during inflation, recoil to their resting position (like releasing a just-inflated party balloon).

Clinical Applications

The rationale for applying spontaneous PEEP or CPAP is the same, whether they are used with or without mechanical ventilation: both techniques improve oxygenation, V\textsubscript{A}/Q, C\textsubscript{rs}, FRC, and WOB. Furthermore, CPAP/PEEP prevents airway collapse during exhalation and reduces the potential for low-volume lung injury.

Potential Problems

Both spontaneous PEEP and CPAP impose an additional workload on the patient's respiratory muscles. Of the two, there is no argument that spontaneous PEEP imposes a far greater workload. However, even a "perfect" CPAP system—one that allows little or no pressure deflection during inspiration—still does not...
eliminate the additional WOB imposed by the patient’s ET. If an ET is too small, kinks, or becomes partially occluded, the patient's WOB may be intolerable, even with the best CPAP system.

Consider the following example:

Suppose a patient’s ET imposes a flow resistance of 10 cm H₂O/L eccentric, CPAP is set at 10 cm H₂O, which drops to 8 cm H₂O at mid-inspiration, and peak VT measures 60 L/minute; pressure at the carinal end of the ET at mid-inspiration is as follows:

\[ \text{pressure (at the carina)} = \text{CPAP} = (\text{resistance at VT} + \text{pressure drop}) = 10 - (10 + 2) = -2 \text{cm H}_2\text{O} \]

What is defined as CPAP in the breathing circuit is actually spontaneous PEEP at the carinal end of the ET. In this situation, maintaining CPAP at the carinal end of the ET requires an airway pressure of ≤20 cm H₂O. In this situation, CPAP APRV achieves an elevated baseline pressure target, which, in turn, reduces venous return and cardiac output; with APRV, however, a higher breathing rate actually lowers the mean Pao₂. As a result, APRV has far less influence on cardiovascular parameters than a comparable level of CPAP.

During the process of initiating APRV, the operator presets the desired CPAP level, number of releases/minute (or breath rate), release time, and release pressure. Although it is possible to set VT during APRV, it is a difficult trial-and-error process. VT is limited by the CPAP level, release pressure, release time, and patient Cao₂. For example, suppose a patient’s CPAP level is set at 10 cm H₂O, Cao₂ is 50 mL/cm H₂O, and the release pressure is set to 0 cm H₂O. The maximum VT for this patient is (assuming complete exhalation) as follows:

\[ \text{VT} = \text{change in pressure} \times \text{Cao₂} = (10 - 0) \text{cm H}_2\text{O} \times 50 \text{mL/cm H}_2\text{O} = 500 \text{mL} \]  

[4]

Release volume or VT also varies with release time; we get the maximum VT only if release time is long enough for the lungs to completely empty. On many occasions, however, oxygenation deteriorates when using a release pressure of zero. When this happens, raising the release pressure will restore oxygenation (97), but unless the CPAP level is raised concomitantly, VT will fall. When switching from conventional mechanical ventilation to APRV, some suggest an adequate VT results by setting CPAP at 1.5 to 2 times that required during conventional ventilation (98,99).

Clinical Applications

Proponents believe APRV should minimize barotrauma because PIP never surpasses the CPAP level and maximum lung volume never exceeds the restored FRC. In addition, APRV lowers physiologic V̇O₂ and improves oxygenation (97,100,101). If these assertions are proven accurate, APRV might be used in place of conventional ventilation for virtually any and all patients.

A variant of APRV, called intermittent CPAP, has recently been used during general anesthesia. Bratke et al. (102) studied surgical patients, exposing them to alternating trials of APRV and CMV. During APRV, patients required less ventilation to produce a PaCO₂ comparable to that during CMV; this finding represents a significant reduction in V̇O₂ and an improved efficiency of ventilation. They also reported that, compared to CMV, APRV improves the accuracy of using the end-tidal partial pressure of CO₂ (ṖETCO₂) as a monitor of PaCO₂.

Potential Problems

There is a potential flaw in the potential for APRV to minimize barotrauma. That is, APRV as described and studied does not necessarily eliminate the risk for low-volume lung injury (4–7). If avoiding VILI requires that all tidal ventilation take place between the lower and upper inflection points, as some now believe (19), then APRV will fail to protect patients, especially if release pressures of 0 to 6 cm H₂O are used as reported (97,100,101). On the other hand, given that the volumetric distance between the lower and upper inflection point is often small and that APRV maintains the same PaCO₂ levels as CMV with less ventilation, APRV may well offer the best lung-protective ventilatory strategy available. This assumes, of course, that the CPAP level is set to a pressure below the upper inflection point and the release level to a pressure above the lower inflection point (19).

### SPECIAL TECHNIQUES

#### Pressure-targeted Volume Ventilation

### Operational Principles

For years, the Food and Drug Administration (FDA) resisted approving any sort of “smart” ventilator setting. Pressure-targeted volume ventilation, pressure-regulated volume control (PRVC), or volume ventilation plus represent a distinct departure from that stance; this mode is far more automated than any before it. To initiate the mode, the operator presets a ventilator rate, VT, Ti, and trigger sensitivity (if patient triggering is desired). On connecting the patient, the ventilator performs up to three test breaths. Test breaths are, as a rule, square flow pattern, volume-controlled breaths with a short end-inspiratory pause. Assuming one of the test breaths is not disturbed by patient attempts to breathe, the ventilator determines the patient’s Cao₂ (Fig. 128.13 and Table 128.3). Once VT and Cao₂ are known, the ventilator rearranges and...
solves equation 4:

\[ \text{Pressure change (target)} = V_t / C_{RS} \]

Having determined the target pressure, the ventilator switches from the test breath protocol into what amounts to a smart PCV. For safety reasons, the ventilator begins PRVC using a pressure substantially below that calculated (above). The ventilator “watches” the exhaled \( V_t \) and gradually, over the next five to ten mechanical breaths, ramps up its pressure until the exhaled \( V_t \) equals that requested. At this point, the operator must thoughtfully set the ventilator’s high \( P_{aw} \) limit based on the current target pressure and the maximum \( P_{aw} \) deemed safe for the patient. This setting is crucial because PRVC is not limited to the initial or starting target pressure. In fact, PRVC changes target pressure as often as required, within limits, to maintain an exhaled \( V_t \) equal to that requested. If, for instance, patient \( C_{RS} \) suddenly deteriorates, \( V_t \) also falls in direct proportion and concomitantly. The ventilator immediately detects the reduced exhaled \( V_t \) and increases target pressure at a rate of 2 or 3 cm H\(_2\)O per breath. Target pressure continues to increase with each breath until either exhaled \( V_t \) is restored or target pressure reaches a value no greater than the high \( P_{aw} \) limit minus 2 to 3 cm H\(_2\)O. The same but opposite response occurs should \( C_{RS} \) improve and \( V_t \) increase; that is, target pressure is reduced by 2 to 3 cm H\(_2\)O until the exhaled \( V_t \) is re-established, but target pressure can go no lower than baseline pressure (PEEP or CPAP level, if applicable) plus 3 cm H\(_2\)O.

**Clinical Applications**

In theory, PRVC combines the best aspects of both VCV and PCV: consistent \( V_t \), delivered at lower \( P_{aw} \) with high initial peak \( V_t \). Some clinicians will consider using this mode for its ability to deliver the same \( V_t \) at lower PIP. There is no doubt this strategy works to lower PIP, but the same \( V_t \) produces the same ERP, regardless of how that volume is forced into the lungs (Fig. 128.14). This explains why, in two carefully controlled studies comparing PRVC to VCV, the researchers found significantly lower PIPs but were unable to detect any difference in outcome [103,104].

Others might consider using PRVC for its ability to provide high initial peak \( V_t \), reduce WOB, and better synchronize with spontaneously active patients. Kallet et al. [53] looked at this issue during lung-protective ventilation, and concluded that PCV and PRVC offer no advantage in reducing WOB when compared to VCV with a high preset \( V_t \).

**Potential Problems**

Conceptually, PRVC appears inherently safer than traditional PCV because it automatically maintains the preset \( V_t \). Nevertheless, if the operator does not carefully set the high \( P_{aw} \) limit, PRVC may produce unexpected and dangerous changes. For instance, imagine a patient supported by PRVC with a \( V_t \) of 500 mL and a target pressure of 25 cm H\(_2\)O; the operator sets a high \( P_{aw} \) limit of 50 cm H\(_2\)O. Suppose this patient develops an acute tussive pneumothorax and the affected lung collapses. The patient’s apparent \( C_{RS} \) would suddenly be reduced by one half or more. If the mode were traditional PCV, \( V_t \) would be reduced to the same extent, thereby leaving the contralateral lung inflated by essentially the same \( V_t \) as prior to the pneumothorax. In contrast, \( V_t \) would also be initially reduced with PRVC, but almost immediately, PRVC would begin increasing the target pressure at 3 cm H\(_2\)O per breath in an effort to re-establish the preset exhaled volume. If the desired \( V_t \) was re-established at 47 cm H\(_2\)O (high \( P_{aw} \) limit minus 3 cm H\(_2\)O), then the entire initial \( V_t \) for both lungs is forced into the remaining good lung, risking hyperinflation and damage to this lung as well.

**Proportional Assist Ventilation (PAV)**

**Operational Principles**

As explained, PAV unloads potentially fatiguing workloads from spontaneously breathing patients. Unfortunately, it is difficult to determine the needed level of PSV, and PCV does not accommodate changes in patient breathing pattern. As a consequence, PAV either oversupports or understrengthens the patient most of the time. Ideally, we need a variable support mode that automatically raises or lowers its response to maintain the same level of support, regardless of patient effort. Younes proposed such a mode, which he named proportional assist ventilation (PAV) [105,106]. This mode relies on what is referred to as the equation of motion of the respiratory system, which states:

\[ P_{aw} = V_t / C_{RS} + V_t / R_{aw} \]  

If \( C_{RS} \) and \( R_{aw} \) are known and the ventilator measures \( V_t \) and \( V_t \) instantaneously, as it provides them, the work required—measured as pressure—can easily be computed, regardless of effort level; recall that work is defined as the integral of \( P_{aw} \) with respect to time. If the ventilator also knows the ET size and flow resistance, stored in ROM, the ventilator can compute nearly the total WOB, although this approach cannot determine the work needed to inflate the chest wall. Estimating patient WOB in real time, throughout each and every breath, allows the ventilator to unload any quantifiable amount or percentage of that WOB; in fact, most PAV systems are preset to unload a specific percentage of the total WOB provided by the ventilator. For instance, if PAV was preset for 50%, the ventilator would measure the work being performed and provide exactly half of it. By measuring \( V_t \) and \( V_{aw} \), many times within each breath, PAV provides 50% of the work generated, regardless of how much or little effort the patient expends.

Assuming PAV works as theorized, it should meet a patient’s varying needs by proportionally altering its response, a feature PSV cannot match. Nevertheless, PAV brings us no closer to a quantifiable and reliable method for determining an appropriate level of support; that is, we do not know what percentage of the total WOB the patient can tolerate. Manufacturers recommend starting PAV at a high percentage and gradually tapering down as the patient improves; the statement simply states the obvious and applies equally well to PSV.

**Clinical Applications**

If PAV proves effective, clinicians should use it in any instance when they would previously have used PSV. So far, PAV remains unproven and controversial. Giannouli et al. [107] compared different levels of PSV and PAV; they reported that PAV seemed more synchronous, but the differences had no effect on gas exchange or spontaneous breathing rate. Hart et al. [108] compared PAV to PSV in patients with neuromuscular and chest wall deformity and concluded that both modes produced similar improvements. Finally, Passam et al. [109] compared different levels of PSV and PAV on breathing pattern, WOB, and gas exchange in mechanically ventilated, hypercapnic COPD.
patients. They concluded that in COPD, although both PAV and PSV produced similar improvements in blood gases, higher levels of PSV often resulted in spontaneous efforts that failed to trigger breaths, whereas under similar circumstances, PAV developed the "runaway" phenomenon. Runaway occurs when, during PAV, the ventilator begins to trigger on and cycle off at rates much higher than the patient's actual spontaneous breathing frequency. Finally, another group reported that PAV was not superior to PSV in unloading the respiratory muscles following artificially increased ventilatory demand (110). These failures have caused some to question whether PAV represents any improvement over PSV (111,112).

**Potential Problems**

Based on existing research, patients respond very differently to PAV than PSV; yet, the end results so far appear similar. To date, we have over 20 years of clinical experience using PSV. It makes little sense switching to PAV without a compelling reason to do so, especially if PAV is going to affect patients differently and in ways we may not yet understand. One of those differences involves the previously mentioned runaway, which results when the pressure provided by PAV exceeds the patient's elastance (inverse of $C_{RS}$) and $R_{aw}$, and persists into the exhalation phase. The physiologic problems that might result during runaway PAV remain to be thoroughly understood and unexplained.

**TRANSPORT VENTILATION**

**Automatic or Manual Ventilation**

Ventilation during transport is still often supported using a self-inflating bag (e.g., the Ambu device), a flow-inflating (Maple-som) system, or oxygen-powered breathing devices (Elder demand valve or similar device). This is indeed surprising in an era dominated by evidence-based medicine. The use of bags and valves, even in the most skilled hands, results in significant breath-to-breath differences in $V_T$, respiratory rate, minute ventilation, PIP and, in some cases, inspired fraction of oxygen ($F_{O2}$). These differences, acting alone or in combination, may disrupt arterial blood gases at the end of even a short intrahospital transport (113–116). Furthermore, bags and valves may not deliver an effective level of PEEP/CPAP and can make spontaneous breathing difficult. A portable ventilator with the proper capabilities is clearly a better choice.

**TABLE 128.4**

**DESIRABLE CHARACTERISTICS FOR TRANSPORT VENTILATORS**

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Requirement</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adult/pediatric use (&gt;5 kg or 11 lb)</td>
<td>Transportable, rugged (&lt;15 lb) and easily stored</td>
</tr>
<tr>
<td>Transportable, rugged (&lt;15 lb) and easily stored</td>
<td>Power source (pneumatic/electric with battery backup)</td>
</tr>
<tr>
<td>Battery life a minimum of 3 hrs</td>
<td>Nonproprietary, single use, universal breathing circuit</td>
</tr>
<tr>
<td>Nonproprietary, single use, universal breathing circuit</td>
<td>NBC filter-compliant (for use in nuclear, biologic, or chemically hazardous environments)</td>
</tr>
<tr>
<td>$V_T$ (50–3,000 mL)</td>
<td>Rate (spontaneous—100 breaths/min)</td>
</tr>
<tr>
<td>$F_{O2}$ (adjustable at least between 0.6 and 1.0, preferably between 0.21 and 1.0)</td>
<td>PEEP/CPAP (0–20 cm $H_2O$)</td>
</tr>
<tr>
<td>Trigger sensitivity (&lt;5 cm $H_2O$, regardless of CPAP level)</td>
<td>Inspiratory time (adjustable to 3.0 sec)</td>
</tr>
<tr>
<td>Inspiratory time (adjustable to 3.0 sec)</td>
<td>Breath types: Volume control (VC) and/or pressure control (PC), spontaneous continuous positive airway pressure (CPAP)</td>
</tr>
<tr>
<td>Alarms (audible and visual) should include:</td>
<td>Positive airway pressure (CPAP)</td>
</tr>
<tr>
<td>Low pressure/disconnect</td>
<td>PEEP, positive end-expiratory pressure.</td>
</tr>
<tr>
<td>High pressure (3–75 cm $H_2O$)</td>
<td>Loss of power (pneumatic or electric)</td>
</tr>
<tr>
<td>Loss of power (pneumatic or electric)</td>
<td>Battery low (if applicable)</td>
</tr>
<tr>
<td>Battery low (if applicable)</td>
<td>Vent-inop</td>
</tr>
<tr>
<td>Alarm silence (≥60 sec)</td>
<td></td>
</tr>
</tbody>
</table>

PEEP, positive end-expiratory pressure.
emergency ventilators that are powered either electrically or pneumatically. It seems only reasonable that disaster preparedness teams consider having at least a few pneumatic transport ventilators available; at present, with certain exceptions, this does not seem to be the case.

**Spontaneous Breathing during Transport**

Disaster preparedness for mass casualty scenarios or avian flu outbreaks has taken the country by storm. To meet the demand, many companies are either modifying existing ventilators for emergency use or are bringing new products to market. Currently, dozens of brands of transport and emergency ventilators are available; this makes the selection process truly difficult at best.

Perhaps a transport ventilator's spontaneous breathing capabilities provide us with a useful metric. Clearly, the act of spontaneous breathing reduces Vp, (112,113,117) True spontaneous breathing, however, dramatically improves virtually every parameter associated with Vp. Putensen et al. (117) compared APRV, which allows unrestricted spontaneous breathing, to PSV, which provides pressure assistance during each spontaneous breath. The APRV group demonstrated improved Vp, Q matching, venous return, right ventricular end-diastolic filling, stroke index, PaO2, O2 delivery, and mixed venous oxygen content when compared to PSV. In addition, APRV reduced Vo2, intrapulmonary shunting, pulmonary vascular resistance, and O2 extraction when compared to PSV (117). Given the compelling strength of the data, it seems strange that so few clinicians consider the idea of spontaneous breathing during transport. Perhaps this oversight developed because so few transport ventilators allow effortless spontaneous breathing. Some might argue that patients do not really need to breathe spontaneously during a 10-minute transport. On the other hand, heavy sedation, paralysis, and CMV really do not appear to be in the best interest of potential avian flu victims.

The vast majority of true emergency or transport ventilators simply do not facilitate spontaneous breathing. Making matters worse, many use optional PEEP valves that attach to the exhalation valve, and demand valves that trigger at subambient pressure; these greatly magnify patient WOB as compared to CPAP, often requiring a Herculean effort to trigger and maintain flow during spontaneous breaths (Fig. 128.19). To circumvent these issues, clinicians take one of several approaches: heavily sedate or even paralyze patients they plan to transport, thus allowing the use of CMV; or moderately sedate patients, allowing them to use the A/C mode of ventilation with minimal patient interaction.

For those wishing to avoid sedatives and paralytic agents, the transport ventilator needed is one that allows unimpeded, extremely low WOB spontaneous breathing. The pNeuton ventilator (Airon Corporation) is pneumatic and claims spontaneous WOB comparable to that of an ICU ventilator; unfortunately, this claim has not been scientifically validated. Without scientific data, testing ventilators with actual breathing will often expose a system's strengths and weaknesses in terms of its spontaneous breathing claims. Considering the potential benefit to patients allowed to spontaneously breathe during transport, it is surprising there are so few scientific studies thoroughly examining WOB as it applies to transport ventilators. Furthermore, the studies done so far are now dated; that is, they do not include many of today's newer and more popular models. For example, one study found the LTV 1000 (Pulmonetics) consistently produced WOB values comparable to those of critical care ventilators (117). The LTV 1000 has two distinct drawbacks as a transport ventilator. First, it will not tolerate the many "crash landings" most transport ventilators must endure. Second, the LTV 1000 uses a very precise, high-speed turbine compressor. When used in a dehumidified, heated, and air-conditioned environment like a hospital, the LTV 1000 will likely work for many hours without incident. On the other hand, if the LTV 1000 is ever briefly exposed to the heat and humidity often found outdoors and is then moved back indoors, a potential exists for condensation to develop on the turbine's bearings. If this occurs, the lifespan of the turbine can be dramatically shortened.

### SIDE EFFECTS AND COMPLICATIONS

#### Spontaneous Breathing

An understanding of the effects of any form of positive airway pressure requires a working knowledge of the physiology that drives spontaneous breathing. Normal spontaneous inspiration at ambient pressure involves contraction of the diaphragm. As the diaphragm contracts, a pressure gradient develops between the pleural space and the mouth; in response, air rushes from the mouth into the lungs. During expiration, the diaphragm relaxes, the gradient reverses, and the gas leaves the lungs.

#### Hemodynamics

Return of venous blood to the heart is dependent on the pressure gradient between the peripheral vasculature and the right atrium. If mean pressure rises or right atrial pressure (RAP) falls, venous return increases. Conversely, a fall in mean pressure or a rise in RAP decreases venous return. Because output of the right ventricle is dependent on the venous return factors that alter mean pressure and RAP also affect cardiac output (119).

#### Intrathoracic Pressure Changes

A decrease in intrathoracic pressure during inspiration is associated with a similar decrease in RAP which enhances venous return. During exhalation, intrapleural pressure increases and venous return falls; these fluctuations are familiar to anyone who has viewed a recording of central venous pressure in a spontaneously breathing patient.

#### Ventricular Interdependence

Because the right and left ventricles are surrounded by the pericardium, volume changes in one chamber affect the other. An increase in right ventricular volume during inspiration pushes the interventricular septum toward the left (posteriorly), thereby increasing left ventricular pressure; this in turn reduces...
left ventricular filling and changes the spatial configuration and compliance of the left ventricle.

### Left Ventricular Afterload

The decrease in intrathoracic pressure is also transmitted to the left ventricle. At the peak of spontaneous inspiration (lowest intrathoracic pressure), the left ventricular end-diastolic pressure is reduced correspondingly. In contrast, the pressure that must be developed by the ventricle to perfuse the systemic vessels outside the thoracic cavity remains the same. Because the ventricle is initiating contraction from a lower baseline pressure, however, the gradient of pressure that must be generated is increased.

The increment in necessary wall tension represents an increase of the left ventricular afterload and may be tolerated poorly by patients with ischemic heart disease and compromised ventricular function. Spontaneous breathing with PEEP, which requires greater decrements in airway pressure and pulmonary pressures for gas to flow, predisposes to this chain of events. Conversely, a properly functioning CPAP system, which requires minimal deflections in airway and intrapleural pressures, minimizes such changes.

### Pulmonary Vascular Changes

Expansion of the lungs also affects hemodynamic function. Alveolar vessels are compressed and elongated while pressure and alveolar volume increase. Extra-alveolar vessels, however, are opened by traction of lung inflation, with a consequent decrease in their resistance. When the alveolar vessels are engorged, inspiration decreases net pulmonary blood volume, and pulmonary venous return to the left ventricle may rise. Conversely, when alveolar vessels contain less blood, spontaneous inspiration decreases pulmonary vascular resistance and abnormal loading of the right ventricle. As end-expiratory pressure is raised, FRC increases back toward normal, and underventilated alveoli re-expand; this releases some of the hypoxic vasoconstriction, and unloading of the right ventricle may occur.

### Spontaneous PEEP and CPAP

Common hemodynamic alterations seen during spontaneous PEEP or CPAP involve five major areas:

1. **Decreased venous return.** Most studies confirm increased intrathoracic pressures, secondary to the increased mean airway pressures used by these modalities. This reduces the mean pressure/RAP gradient, which in turn reduces venous return. The heart has less blood to pump and output falls.

2. **Decreased right ventricular function.** Acute respiratory failure and PEEP/CPAP may, in some circumstances, increase pulmonary vascular resistance. Conceivably, the right ventricle might fail under these conditions; yet, this is probably not a major direct cause of positive-pressure-induced cardiovascular insufficiency.

3. **Decreased left ventricular function.** Left ventricular dysfunction can result from increased mean airway pressures, but the changes are most likely the result of right ventricular dilatation and encroachment. Some investigators question this hypothesis, however. Prewitt and Wood (120) suggest that in selected instances (for example, high PEEP), 50% of the reduction in cardiac output results from left ventricular failure. Clearly, the mechanisms are complex and incompletely understood.

4. **Neural and humoral depression.** In canine cross-circulation studies, PEEP applied to one dog’s lungs resulted in a reduction in cardiac output in both animals (121). The nature and composition of the depressant substance or substances is unknown.

5. **Reduction of endocardial blood flow.** It has been suggested that an increased mean airway pressure may act to impede coronary arterial blood flow (122). Such a decrease has been demonstrated experimentally during the use of PEEP.

Most studies show that spontaneous PEEP does not affect circulatory function adversely, as long as patients are not hypovolemic. These observations, oddly, are diametrically opposed to those expected during normal breathing. This phenomenon is best explained by the fact that PEEP is not generally used on normal lungs; when it is, FRC is raised above normal, often significantly. With more volume in the lungs, even during exhalation, venous return may be compromised. When either PEEP or CPAP is used therapeutically, however, patients invariably have reduced compliance and a significantly reduced FRC.Collapsed and underventilated alveoli produce hypoxic vasoconstriction in the affected areas; this often markedly increases pulmonary vascular resistance and abnormal loading of the right ventricle. As end-expiratory pressure is raised, FRC increases back toward normal, and underventilated alveoli re-expand; this releases some of the hypoxic vasoconstriction, and unloading of the right ventricle may occur.

Large intrapleural pressure fluctuations are not always beneficial, and could easily overfill the right ventricle, resulting in an elevated left ventricular afterload. It is quite possible a worst case scenario could involve a patient with severe coronary artery disease and left ventricular failure, managed using a high level of spontaneous PEEP. The resultant increase in the left ventricular afterload on an already failing myocardium would predispose to a dramatic and potentially dangerous fall in systemic cardiac output, as well as acute pulmonary edema. Substituting a properly functioning spontaneous CPAP system with a minimal trigger sensitivity would, all other things being equal, unload the left ventricle by decreasing the preloading of the right ventricle.

### Mechanical Ventilation

#### Hemodynamics

**Falling Pressures.** As a rule, any form of positive pressure breathing will reduce venous return and decrease right
ventricular preload; with normal lungs, the effect is pronounced. Even modest increases in airway pressure will elevate the FRC well above normal; an increased intrapleural pressure from the overexpanded lungs presses the pericardium and compresses the heart. These conditions make interpreting filling pressures a challenge, as the central venous pressure (CVP) and pulmonary artery occlusion pressure may be elevated while ventilator stroke volumes are actually decreased.

**Ventricular Function.** As stated, the elevated airway pressures associated with positive pressure breathing increase the right ventricular afterload, and, in turn, decrease venous inflow to the right heart. At the same time, transmural aortic pressure, left ventricular afterload, and end-systolic left ventricular volume fall. The reduced output volume is easily restored by intravascular volume expansion—enough to counteract some or all of airway pressure on venous return. If, after re-expansion of intravascular volume, the source of elevated airway pressure—i.e., ventilator or PEEP/CPAP system—is suddenly removed, a venous return surge that may be well beyond the initial baseline predisposes the patient to acute pulmonary edema (123); this complication is especially likely if the patient’s left ventricle is compromised.

Robotham et al. (124) summarized the known and postulated effects of positive pressure inflation of the lungs on cardiopulmonary function; most of these have already been reviewed. They concluded that mechanical ventilation may act as a (relatively) noninvasive cardiac assist device and deserves further evaluation as such.

**IMV/SIMV/CPAP.** The hemodynamic effects of spontaneous breathing, with or without CPAP, and those secondary to positive pressure ventilation become complicated when combined, as during IMV/SIMV with CPAP. The cumulative effect likely depends on the relative contributions of spontaneous versus mandated breaths, as well as the absolute values of the inspiratory and expiratory pressures, P~i~ baseline cardiovascular status, intravascular volume, and so forth. Ventilator performance also plays an important role, especially during spontaneous breathing.

A difficult-to-trigger CPAP system, or a poorly designed and slow-to-respond demand-flow valve, often requires major inspiratory efforts by the patient. These factors were the primary reason many early trials of IMV failed; that is, patients were simply fatigued to the point of failure by work imposed by the system—not by the technique.

**Barotrauma and Ventilator-Induced Lung Injury**

For many years, the concept of pulmonary barotrauma was limited to extra-alveolar air leaks. It is now abundantly clear that human lungs can be damaged internally by the ventilator with or without air leaks; this type of damage is known as ventilator-associated lung injury (VALI) and is reviewed in depth elsewhere (see Chapter 138).

**Extra-alveolar Air Leaks.** All forms of mechanical ventilation, whether by virtue of positive or negative pressure, rhythmically drive air in and out of the lungs. As outlined, mechanical ventilation disrupts normal hemodynamics, alters V~i~/Q ratios, and occasionally damages the lungs. Pulmonary barotrauma (PBT) represents the classic form of VILI. Pulmonary barotrauma includes pneumothorax, pneumomediastinum, pneumopericardium, pneumoperitoneum, pneumoretroperitoneum, subcutaneous air, and air embolization—either venous or arterial.

Interestingly, none of these conditions actually describe lung injury. Rather, each of these represents a form of extra-alveolar air; each occurs after the lung fabric is torn and an air leak follows. Air leaks occur following a tear in the fabric of lung parenchyma. If the tear involves the visceral pleura, a pneumothorax often results as the air quickly moves into the pleural space, which the pressure is negative relative to alveolar pressure most of the time. Tension pneumothorax is the most threatening variety of this problem and can obliterate cardiac output if not immediately decompressed. When an air leak occurs away from the pleura, the repeated stretching associated with positive pressure ventilation facilitates the dissection of air along the perivascular sheaths that surround the airways. Eventually, the dissecting air reaches the pulmonary hilum, where it may invade the subcutaneous tissues of the neck or enter the mediastinum and beyond. Rarely, the rent exposes a vessel large enough for air to enter, and with sufficient air pressure changes (positive or negative), air may enter the circulation. Scuba divers who ascend too rapidly or hold their breath while surfacing can easily rupture their lungs. Because divers normally surface with their heads up, the air bubbles rise and may reach the brain. Cerebral air emboli can be fatal, particularly when the diver cannot be quickly recompressed in a hyperbaric chamber.

A common misconception—that PEEP and CPAP increase the incidence of barotrauma—persists even today. Yet, no increase in the incidence of barotrauma occurs when positive pressure ventilation and CPAP are compared with positive pressure alone (125,126).

**SUMMARY**

Mechanical ventilators continue to evolve rapidly in complexity, design, and function. It is quite likely this brisk pace will persist for many years. As outlined, it also seems likely that controversies regarding how and when to use ventilators safely and effectively will not be conclusively resolved anytime soon. This set of circumstances means that those of us responsible for prescribing, operating, monitoring, or repairing ventilators must rise to the challenge of maintaining an up-to-date knowledge base. This is no easy task but one that will result in significant benefits for all parties involved.

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