

Mechanical Ventilation

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Objectives:

- To describe the role of the ventilator in determining respiratory mechanics
- To recommend disease-specific ventilator strategies aimed at reducing the adverse consequences of mechanical ventilation
- To review new information regarding ventilation in ARDS
- To address the role of noninvasive ventilation
- To discuss the complications of mechanical ventilation

Key words: ARDS; assist-control ventilation; COPD; inverse ratio ventilation; mechanical ventilation; noninvasive ventilation; pressure control; pressure support; status asthmaticus; tidal volume; synchronized intermittent mandatory ventilation

This lecture offers an approach in which the ventilator is used as a probe of the patient's respiratory system mechanical derangements, and then the ventilator settings are tailored to the patient's mechanical and gas exchange abnormalities. This facilitates early stabilization of the patient on the ventilator in such a way as to optimize carbon dioxide removal and oxygen delivery within the limits of abnormal neuromuscular function, lung mechanics, and gas exchange.

The fundamental purpose of mechanical ventilation is to assist in elimination of carbon dioxide and uptake of adequate oxygen while the patient is unable to do so or should not be allowed to do so. Such patients fall into two main groups: (1) those in whom full rest of the respiratory muscles is indicated (such as during shock; severe, acute pulmonary derangement; or deep sedation or anesthesia), and (2) those in whom some degree of respiratory muscle use is desired (*eg*, to strengthen or improve the coordination of the respiratory muscles; to assess the ability of the patient to sustain the work of breathing; or to begin spontaneous ventilation). It is important for the intensivist to be explicit about whether the respiratory muscles should be rested or exercised because the details of ventilation (mode, settings) usually follow logically from this fundamental point.

If full rest of the respiratory muscles is desired, it is incumbent on the physician to assure that this is indeed achieved. Although some patients are fully passive while being ventilated (those with deep sedation, some forms of coma, metabolic alkalosis, sleep-disordered breathing), most patients will make active respiratory efforts, even on assist-control ventilation (ACV), at times performing extraordinary amounts of work. Unintended patient effort can be difficult to recognize but, aside from obvious patient effort, may be signaled by an inspiratory fall in intrathoracic pressure (as noted on a central venous or pulmonary artery pressure tracing, or with an esophageal balloon) or by triggering of the ventilator. Recognizing patient effort has been greatly aided by the provision of real-time displays of flow and pressure waveforms, now commonly available on modern ventilators.

Choosing a ventilatory mode and settings appropriate for each individual patient depends not only on the physician's goals (rest vs exercise), but also on knowledge of the mechanical properties of the patient's respiratory system. Most ventilators now have the capability of displaying waveforms of pressure, flow, and volume vs time, as well as flow vs volume. Using waveforms, it is easiest to gather information regarding the patient-ventilator interaction when patients are ventilated with a volume-preset mode [ACV or synchronized intermittent mandatory ventilation (SIMV)]. Still, some useful information can be gleaned from waveforms during pressure-preset ventilation [pressure-support ventilation (PSV) and pressure-control ventilation (PCV)].

The first step is to seek signs of inspiratory effort in the pressure tracing. Respiratory muscle contraction does not cease in most patients receiving mechanical ventilation, even in the ACV mode. Thus, it is possible that a weak patient will remain weak, through continued breathing effort, despite the institution of mechanical support. In volume-preset modes, the signs of persistent effort include the presence of triggering, concavity during inspiration, and a variable peak airway opening pressure (P_{peak}). When the goal of ventilation is to rest the respiratory muscles, ventilator adjust-

ments, psychological measures, pharmacologic sedation, and therapeutic paralysis can be useful. Ventilator strategies to reduce the patient's work of breathing include increasing the minute ventilation to reduce P_{CO_2} (although this may run counter to other goals of ventilation, especially in patients with ARDS or severe obstruction), increasing the inspiratory flow rate, and changing the mode to pressure-preset ventilation (PSV or PCV).

The next step is to determine whether the patient has significant airflow obstruction. This can be inferred by inserting a brief end-inspiratory pause, then determining the difference between P_{peak} and plateau airway pressure (P_{plat}). Alternatively, one can examine the expiratory flow waveform, seeking low flow and prolonged expiration, signs that are present regardless of the mode of ventilation (ACV, SIMV, PSV, PCV). Bronchodilator therapy can be assessed by noting whether expiratory flow increases, the expiratory time (T_E) shortens, or there is a reduction in P_{peak} , P_{plat} , or auto-positive end-expiratory pressure (autoPEEP).

Finally, one should assure that the patient and ventilator are synchronized, *ie*, that each attempt by the patient to trigger the ventilator generates a breath. The most common situation in which the patient fails to trigger breaths occurs in severe obstruction when autoPEEP is present. This is recognized at the bedside when the patient makes obvious efforts that fail to produce a breath. Using waveforms, these ineffective efforts cause a temporary slowing of expiratory flow, sometimes halting it completely.

Modes of Mechanical Ventilation

Technologic innovations have provided a plethora of differing modes by which a patient can be mechanically ventilated.¹ Various modes have been developed with the hope of improving gas exchange, patient comfort, or speed of return to spontaneous ventilation. Aside from minor subtleties, however, nearly all modes allow full rest of the patient, on the one hand, or substantial exercise on the other. Thus, in the great majority of patients, choice of mode is merely a matter of patient or physician preference. Noninvasive ventilation should be considered before intubation and ventilation in many patients who are hemodynamically stable and do not require an artificial airway, especially those with acute-on-chronic respiratory failure,

postoperative respiratory failure, and cardiogenic pulmonary edema.

During volume-preset ventilation (and assuming a passive patient), P_{plat} is determined by the tidal volume (V_T) and the static compliance of the respiratory system (C_{rs}):

$$P_{plat} = V_T / C_{rs} + PEEP$$

where PEEP (positive end-expiratory pressure) also includes autoPEEP.

On the other hand, in pressure-preset modes, a fixed inspiratory pressure (P_{insp}) is applied to the respiratory system, whatever the resulting V_T . However, the V_T is predictable (again, assuming a passive patient) when the C_{rs} is known:

$$V_T = (P_{insp} - PEEP) \times C_{rs}$$

assuming time for equilibration between P_{insp} and alveolar pressure. Thus, a patient with static C_{rs} of 50 mL/cm H_2O ventilated on ACV at a V_T of 500 mL with no PEEP (or autoPEEP) will have a P_{plat} of about 10 cm H_2O , while the same patient ventilated on PCV at 10 cm H_2O will have a V_T of about 500 mL. Thus, while physicians' comfort level with volume-preset and pressure-preset modes may be very different, the modes can be similar as they are tied to each other through the patient's C_{rs} .

A potential advantage of pressure-preset ventilation is greater physician control over the P_{peak} (since $P_{peak} = P_{insp}$) and the peak alveolar pressure, which could lessen the incidence of ventilator-induced lung injury. However, this same reduction in volutrauma risk should be attainable during volume-preset ventilation if a V_T appropriate to the lung derangement is chosen. Indeed, the ARDSNet trial, which demonstrated a mortality reduction in the low- V_T group, used ACV and a V_T of 6 mL/kg. Nevertheless, pressure-preset modes make such a lung-protection strategy easier to carry out by dispensing with the need to repeatedly determine P_{plat} and periodically adjust the V_T . During use of pressure-preset modes, the patient also has greater control over inspiratory flow rate, and therefore potentially increased comfort. A disadvantage of pressure-preset modes is that changes in respiratory system mechanics (*eg*, increased airflow resistance or lung stiffness) or patient effort may decrease the minute ventilation, necessitating alarms for adequate ventilation. Also, the mechanics cannot be readily determined.

In the following descriptions, each mode is first illustrated for a passive patient, such as following muscle paralysis, then for the more com-

mon situation in which the patient plays an active role in ventilation. On some ventilators, V_T can be selected by the physician or respiratory therapist, while on others a minute ventilation and respiratory rate (f) are chosen, secondarily determining the V_T . Similarly, on some machines an inspiratory flow rate is selected, while on others flow depends on the ratio of inspiratory time (T_i) to total respiratory cycle time and f , or an inspiratory to expiratory (I:E) ratio and f .

Conventional Modes of Ventilation

Assist-Control Ventilation: Passive Patient—

The set parameters of the assist-control mode are the inspiratory flow rate, frequency (f), and V_T . The ventilator delivers f equal breaths per minute, each of V_T volume. V_T and flow determine the T_i , T_E , and the I:E ratio. P_{plat} is related to the V_T and the compliance of the respiratory system, while the difference between P_{peak} and P_{plat} includes contributions from flow and inspiratory resistance.

Active Patient—The patient has the ability to trigger extra breaths by exerting an inspiratory effort exceeding the preset trigger sensitivity, each at the set V_T and flow, and to thereby change T_i , T_E , and I:E ratio, and to potentially create or increase autoPEEP. Typically, each patient will display a preferred rate for a given V_T and will trigger all breaths when the controlled ventilator frequency is set a few breaths/min below the patient's rate; in this way, the control rate serves as an adequate support should the patient stop initiating breaths. When high inspiratory effort continues during the ventilator-delivered breath, the patient may trigger a second, superimposed ("stacked") breath (rarely a third as well). Patient effort can be increased (if the goal is to exercise the patient) by increasing the magnitude of the trigger or by lowering V_T (which increases the rate of assisting). Lowering f at the same V_T generally has no effect on work of breathing when the patient is initiating all breaths.

Synchronized Intermittent Mandatory Ventilation: In the passive patient, SIMV cannot be distinguished from controlled ventilation in the ACV mode. Ventilation is determined by the mandatory f and V_T . However, if the patient is not truly passive, he may perform respiratory work during the mandatory breaths. More to the point of the SIMV mode, he can trigger additional breaths by lowering the airway opening pressure below the trigger

threshold. If this triggering effort comes in a brief, defined interval before the next mandatory breath is due, the ventilator will deliver the mandatory breath ahead of schedule in order to synchronize with the patient's inspiratory effort. If a breath is initiated outside of the synchronization window, V_T , flow, and I:E ratio are determined by patient effort and respiratory system mechanics, not by ventilator settings. The spontaneous breaths tend to be of small volume and are highly variable from breath to breath. The SIMV mode is often used to gradually augment the patient's work of breathing by lowering the mandatory breath f (or V_T), driving the patient to breathe more rapidly in order to maintain adequate ventilation, but this approach appears to prolong weaning.^{2,3} Although this mode continues to be used widely, there is little rationale for it and SIMV is falling out of favor.

Pressure-Control Ventilation: In the passive patient, ventilation is determined by f , the inspiratory pressure increment ($P_{insp} - PEEP$), I:E ratio, and the time constant of the patient's respiratory system. In patients without severe obstruction (*ie*, time constant not elevated) given a sufficiently long T_i , there is equilibration between the ventilator-determined P_{insp} and alveolar pressure (P_{alv}) so that inspiratory flow ceases. In this situation, V_T is highly predictable, based on P_{insp} ($= P_{alv}$), and the mechanical properties of the respiratory system (C_{rs}). In the presence of severe obstruction or if T_i is too short to allow equilibration between ventilator and alveoli, V_T will fall below that predicted based on P_{insp} and C_{rs} .

The active patient can trigger additional breaths by reducing the airway opening pressure (P_{ao}) below the triggering threshold, raising the I:E ratio. The inspiratory reduction in pleural pressure combines with the ventilator P_{insp} to augment the transpulmonary pressure and the V_T . Because T_i is generally set by the physician, care must be taken to discern the patient's neural T_i (from the waveforms display) and adjust the ventilator accordingly; otherwise, additional sedation might be necessary.

Pressure-Support Ventilation: The patient must trigger the ventilator in order to activate this mode, so pressure support is not applied to passive patients. Ventilation is determined by P_{insp} , patient-determined f , and patient effort. Once a breath is triggered, the ventilator attempts to maintain P_{ao} at the physician-determined P_{insp} , using whatever flow is necessary to achieve this. Eventually flow

begins to fall as a result of either cessation of the patient's inspiratory effort or increasing elastic recoil of the respiratory system as V_T rises. The ventilator will maintain a constant P_{insp} until inspiratory flow falls an arbitrary amount (eg, to 20% of initial flow) or below an absolute flow rate. The patient's work of breathing can be increased by lowering P_{insp} or making the trigger less sensitive, and can inadvertently increase if respiratory system mechanics change, despite no change in ventilator settings. Respiratory system mechanical parameters cannot be determined readily on this mode because the ventilator and patient contributions to V_T and flow are not represented by P_{ao} ; accordingly, these important measurements of P_{plat} , $P_{peak} - P_{plat}$, and autoPEEP are measured during a brief, daily switch from PSV to volume-preset ventilation. A potential advantage of PSV is improved patient comfort and, for patients with very high drive, reduced work of breathing compared with volume-preset modes.

Mixed Modes: Some ventilators allow combinations of modes, most commonly SIMV plus PSV. There is little reason to use such a hybrid mode, although some physicians use the SIMV as a means to add sighs to PSV, an option not otherwise generally available. Because SIMV plus PSV guarantees some backup minute ventilation (which PSV does not), this mode combination may have value in occasional patients at high risk for abrupt deterioration in central drive.

Triggered Sensitivity: In the assist-control, SIMV, and pressure-support modes, the patient must lower the P_{ao} below a preset threshold in order to "trigger" the ventilator. In most situations, this is straightforward: The more negative the sensitivity, the greater the effort demanded of the patient. This can be used intentionally to increase the work of breathing when the goal is to strengthen the inspiratory muscles. When autoPEEP is present, however, the patient must lower P_{alv} by the autoPEEP amount in order to have any impact on P_{ao} , then further by the trigger amount to initiate a breath. This can dramatically increase the required effort for breath initiation.

Flow-triggering systems ("flow-by") have been used to further reduce the work of triggering the ventilator. In contrast to the usual approach in which the patient must open a demand valve in order to receive ventilatory assistance, continuous-flow systems maintain a continuous high flow, then

further augment flow when the patient initiates a breath. These systems can reduce the work of breathing slightly below that present when using conventional demand valves, but do not solve the problem of triggering when autoPEEP is present.

Unconventional Ventilatory Modes

Inverse-Ratio Ventilation: Inverse-ratio ventilation (IRV) is defined as a mode in which the I:E ratio is > 1 . There are two general ways to apply IRV: pressure-controlled IRV (PC-IRV), in which a preset airway pressure is delivered for a fixed period of time at an I:E ratio > 1 , or volume-controlled IRV (VC-IRV), in which a V_T is delivered at a slow (or decelerating) inspiratory flow rate (or an end-inspiratory pause is inserted) to yield an I:E > 1 . For PC-IRV, the physician must specify the inspiratory airway pressure, f , and I:E ratio, while V_T and flow profile are determined by respiratory system impedance as discussed for PCV above. Commonly, the initial P_{insp} is 20 to 40 cm H_2O (or 10 to 30 cm H_2O above the PEEP), f is 20/min, and the I:E is 2:1 to 4:1. For VC-IRV, the operator selects a V_T , f , flow (typically a low value), flow profile, and, possibly, an end-inspiratory pause. The chosen values result in an I:E $> 1:1$ and as high as 5:1.

Compared with conventional modes of ventilation, lung oxygen exchange is often improved with IRV, owing to increased mean alveolar pressure and volume consequent to the longer time above functional residual capacity, or due to creation of autoPEEP. It is remotely possible that IRV causes better ventilation of lung units with long time constants, but these are so short in normal lungs (and shorter in acute hypoxemic respiratory failure) that such redistribution is unlikely to occur with slower flow, and could not reduce shunt even if it did. Because autoPEEP is a common consequence of IRV, serial determination of its magnitude is essential for safe use of this mode. Both PC-IRV and VC-IRV generally require heavy sedation with or without muscle paralysis.

Airway Pressure Release Ventilation: Airway pressure release ventilation consists of continuous positive airway pressure (CPAP) which is intermittently released to allow a brief expiratory interval. Conceptually, this mode is pressure-controlled IRV during which the patient is allowed to initiate spontaneous breaths. A potential advantage of airway pressure release ventilation is that mean

alveolar pressure is lower than it would be during positive-pressure ventilation from the same amount of CPAP, possibly reducing the risks of barotrauma and hemodynamic compromise. Whether this mode provides any benefit over modern low- V_T ventilation remains to be shown.

Dual-Control Ventilation (Pressure-Regulated Volume-Control, Volume-Assured Pressure-Support): Some modes attempt to derive the benefits of volume- and pressure-preset modes at the same time. For example, pressure-regulated volume-control mode applies a time-limited pressure and adjusts the pressure of subsequent breaths, as needed, to assure a set V_T . The purported advantages of this mode are prevention of overdistention (by limiting pressure) and high initial inspiratory flows, while guaranteeing a V_T . A down side is that as patient effort increases, the ventilator *reduces* support. In the volume-assured pressure-support mode, volume is monitored during each pressure-support breath and, if a preset V_T is not achieved, additional volume is given at constant flow to augment that breath. The primary down side of this mode, compared with PSV, is that the extra volume may cause overdistention (and volutrauma).

The greatest problem with such newer modes is that they are very complex, the algorithm describing their function is not usually understood by practitioners, and they change during a breath, or from breath to breath, depending on patient effort, sometimes in ways that can provoke unanticipated effects.

Proportional-Assist Ventilation: Proportional-assist ventilation is intended only for spontaneously breathing patients. The goal of this novel mode is to attempt to normalize the relationship between patient effort and the resulting ventilatory consequences.⁴ The ventilator adjusts P_{insp} in proportion to patient effort both throughout any given breath and from breath to breath. This allows the patient to modulate his breathing pattern and total ventilation. This is implemented by monitoring instantaneous flow and volume (V) of gas from the ventilator to the patient and varying the P_{insp} as follows:

$$P_{insp} = f_1 \times V + f_2 \times \text{flow}$$

where f_1 and f_2 are selectable functions of volume (elastic assist) and flow (resistive assist), values for which can be estimated from the patient's respiratory mechanics. Potential advantages of this method are greater patient comfort, lower P_{peak} , and enhancement of the patient's reflex and behavioral respiratory control mechanisms.

High-Frequency Ventilation: Several modes of ventilation have in common the use of V_T smaller than the dead space volume.⁵ Gas exchange does not occur through convection as during conventional ventilation, but through bulk flow, Taylor diffusion, molecular diffusion, nonconvective mixing, and possibly other mechanisms. These modes include high-frequency oscillatory ventilation and high-frequency jet ventilation. Theoretical benefits of high-frequency ventilation (HFV) include lower risk of barotrauma as a result of smaller tidal excursions, improved gas exchange through a more uniform distribution of ventilation, and improved healing of bronchopleural fistulas. A substantial risk is that dynamic hyperinflation is the rule and alveolar pressure is greatly underestimated by monitoring pressure at the airway opening. Controlled trials of HFV have failed to demonstrate any clinically relevant benefit, and complications (especially barotrauma and tracheal injury) are seen frequently. Nevertheless, HFV is the natural extension of lowering the V_T as a means to prevent volutrauma, and there is renewed interest in this old technique.

Noninvasive Ventilation

Mechanical ventilation for acute respiratory failure carries a high morbidity and mortality due, in part, to violation of the glottis by the endotracheal tube. In patients with acute-on-chronic respiratory failure, numerous studies have demonstrated that noninvasive ventilation (NIV) effectively relieves symptoms, improves gas exchange, reduces the work of breathing, lessens complications, shortens the ICU length of stay, and improves survival.^{6,7}

Both nasal and oronasal masks have been used successfully. Nasal masks are especially difficult to use in edentulous patients who are unable to control mouth leak. Careful attention to mask leaks and adjusting air flow and pressure-support levels are important considerations. Inflatable cuffs, nasal bridge protection, and the availability of a range of mask sizes to ensure proper fit can minimize mask complications. I find it useful to initiate ventilation by briefly holding the mask (already connected to the ventilator) onto the patient's face, rather than first strapping the mask on and then initiating ventilatory assistance. Sedative medications are occasionally appropriate and can improve tolerance of NIV, but carry some risk of respiratory depression and aspiration.

Patient-ventilator asynchrony (PVA) describes a patient's breathing efforts that are not coupled to machine output. During NIV, two mechanisms of PVA are common. The first is failure of the patient to lower sufficiently the proximal airway pressure (mask pressure) due to the presence of autoPEEP. As during invasive ventilation, counterbalancing the autoPEEP with externally applied PEEP provides a means by which to lower the work of triggering. The second common mechanism for PVA is failure of the ventilator to detect end inspiration because the patient's subsiding effort is cloaked by a mask leak. Most pressure-support ventilators terminate inspiration when inspiratory flow falls to a preset threshold, often at an arbitrary low value of flow or at a fixed percent of the peak inspiratory flow. Mask leaks prevent the flow from falling to this threshold, so the ventilator fails to switch off the inspiratory pressure even while the patient is making active expiratory efforts. This serves to increase patient discomfort and the work of breathing. Using other methods for terminating inspiration, such as time-cycled pressure-support or volume assist-control, can minimize this problem.

Either conventional ICU ventilators or one of many portable bilevel pressure-targeted ventilators, initially designed for home ventilation, can be used. Limitations of portable pressure-targeted ventilators include the lack of waveform displays, the inability to deliver a high fraction of inspired oxygen (F_{IO_2}) [greater than about 40%; some new machines allow an F_{IO_2} as high as 1.0], and the potential for rebreathing of exhaled gas. Whether volume-preset ventilation (such as assist-control) or pressure-preset ventilation is superior for NIV remains debated. Both modes have been used successfully, but direct comparisons between modes are few.

I believe the following points will minimize the chances that NIV will fail:

1. Develop an individual and institutional commitment to NIV.
2. Select patients carefully, excluding those with hemodynamic instability, inadequate airway protective reflexes, or little prospect of improvement within the next several days.
3. Have available a selection of oronasal and nasal masks to increase the probability of a good fit.
4. Use the pressure-support mode, beginning with modest settings, such as PEEP = 3 cm H_2O , PSV = 5 cm H_2O , and the most sensitive trigger, periodi-

cally removing the mask to allow the patient to sense its effect.

5. Education, reassurance, and modest sedation (when required) may improve tolerance to the mask and ventilator.
6. Increase the PEEP to ease the work of triggering with a goal of (typically) 4 to 8 cm H_2O ; raise the level of PSV until the patient is subjectively improved, the V_T is sufficient, and the rate begins to fall, with goal of 10 to 15 cm H_2O .
7. Detect and correct mask leaks by repositioning, achieving a better fit, changing the type of mask, removing nasogastric tubes (gastric decompression is not recommended during NIV), or adjusting the ventilator to reduce peak airway pressure.
8. Pay particular attention in the first hour to patient-ventilator synchrony, using waveform displays as a guide.

Management of the Patient

Initial Ventilator Settings

Initial ventilator settings depend on the goals of ventilation (*eg*, full respiratory muscle rest vs partial exercise), the patient's respiratory system mechanics, and minute ventilation needs. Although each critically ill patient presents myriad challenges, it is possible to identify five subsets of ventilated patients (1) the patient with normal lung mechanics and gas exchange; (2) the patient with severe airflow obstruction; (3) the patient with acute-on-chronic respiratory failure; (4) the patient with acute hypoxemic respiratory failure, and (5) the patient with restrictive lung or chest wall disease.⁸

In all patients, the initial F_{IO_2} should usually be 0.5 to 1.0 to assure adequate oxygenation, although it can usually be lowered within minutes when guided by pulse oximetry and, in the appropriate setting, applying PEEP. In the first minutes following institution of mechanical ventilation, the physician should remain alert for several common problems. These include, most notably, airway malposition, aspiration, and hypotension. Positive-pressure ventilation may reduce venous return and so cardiac output, especially in patients with a low mean systemic pressure (*eg*, hypovolemia, venodilating drugs, decreased sympathetic tone from sedating drugs, neuromuscular disease) or a very high ventilation-related pleural pressure (*eg*, chest wall restriction, large amounts of

PEEP, or obstruction causing autoPEEP). If hypotension occurs, intravascular volume should be rapidly expanded while steps are taken to lower the pleural pressure (smaller V_T , less minute ventilation).

The Patient With Normal Respiratory Mechanics and Gas Exchange

Patients with normal lung mechanics and gas exchange can require mechanical ventilation for several reasons: (1) because of loss of central drive to breathe (eg, drug overdose or structural injury to the brainstem); (2) because of neuromuscular weakness (eg, high cervical cord injury, acute idiopathic myelitis, myasthenia gravis); (3) as an adjunctive therapy in the treatment of shock; or (4) in order to achieve hyperventilation (eg, in the treatment of elevated intracranial pressure following head trauma). Following intubation, initial ventilator orders should be an F_{IO_2} of 0.5 to 1.0, V_T of 8 to 15 mL/kg, rate of 8 to 12, and inspiratory flow rate of 40 to 60 L/min. Alternatively, if the patient has sufficient drive and is not profoundly weak, PSV can be used. The level of pressure support is adjusted (usually to the range of 10 to 20 cm H_2O above PEEP) to bring the respiratory rate down into the low 20s, usually corresponding to a V_T of about 400 mL. If gas exchange is entirely normal, the F_{IO_2} can likely be lowered further based on pulse oximetry or arterial blood gas determinations.

Soon after the initiation of ventilation, airway pressure and flow waveforms should be inspected for evidence of patient-ventilator dyssynchrony or undesired patient effort. If the goal of ventilation is full rest, the patient's drive can often be suppressed by increasing the inspiratory flow rate, frequency, or V_T ; of course, the latter two changes may induce respiratory alkalemia. If such adjustments do not diminish breathing effort (despite normal blood gases) to an undetectable level, sedation may be necessary. If this does not abolish inspiratory efforts and full rest is essential (as in shock), muscle paralysis should be considered. Measures to prevent atelectasis should include sighs (6 to 12/h at 1.5 to 2 times the V_T) or small amounts of PEEP (5 to 7.5 cm H_2O).

Patients With Severe Airflow Obstruction

Severe obstruction is seen most commonly in patients with status asthmaticus, but also rarely in those with inhalation injury or central airway lesions,

such as tumor or foreign body, that are not bypassed with the endotracheal tube. Some of these patients may benefit from NIV, but most will require invasive ventilation. These patients are usually extremely anxious and distressed. Deep sedation should be provided in such instances, supplemented in some patients by therapeutic paralysis, although the use of paralytic drugs occasionally causes long-lasting weakness. These interventions help to reduce oxygen consumption (and hence carbon dioxide production), to lower airway pressures, and to reduce the risk of self-extubation.

Because the gas exchange abnormalities of airflow obstruction are largely limited to ventilation-perfusion mismatch, an F_{IO_2} of 0.5 suffices in the vast majority of patients. Ventilation should be initiated using the ACV mode (or SIMV), the V_T should be small (5 to 7 mL/kg), and the respiratory rate 12 to 15 breaths/min. A peak flow of 60 L/min is recommended and higher flow rates do little to increase T_E . For example, if the V_T is 500, the respiratory rate 15, and the flow is 60 L/min, the T_E is 3.5 s. Raising flow (dramatically) to 120 L/min increases the expiratory time to only 3.75 s, a trivial improvement. In contrast, a small reduction in respiratory rate to 14/min increases the T_E to 3.8 s. This example serves to emphasize not only the relative lack of benefit of raising the flow rate but also the importance of minimizing minute ventilation when the goal is to reduce autoPEEP. Some patients who remain agitated during ACV can be made more comfortable by using PSV (or PCV) with a total inspiratory pressure of around 30 cm H_2O . Finally, if the patient is triggering the ventilator, some PEEP should be added to reduce the work of triggering.⁹ Although this occasionally compounds the dynamic hyperinflation, potentially compromising cardiac output, usually autoPEEP increases little as long as PEEP is not set higher than about 85% of the autoPEEP. The goals are (1) to minimize alveolar overdistention ($P_{plat} < 30$) and (2) to minimize dynamic hyperinflation (autoPEEP < 15 cm H_2O or end-inspiratory lung volume < 20 mL/kg), a strategy that largely prevents barotrauma.^{10,11} Reducing minute ventilation to achieve these goals generally causes the P_{CO_2} to rise above 40 mm Hg, often to 70 mm Hg or higher. Although this requires sedation, such permissive hypercapnia is tolerated quite well, except in patients with increased intracranial pressure, and perhaps in those with ventricular dysfunction or critical pulmonary hypertension.¹²

Patients With Acute-on-Chronic Respiratory Failure

Acute-on-chronic respiratory failure is a term used to describe the exacerbations of chronic ventilatory failure, often requiring ICU admission, usually occurring in patients with COPD. Unlike patients with status asthmaticus, patients in this population tend to have relatively smaller increases in inspiratory resistance, their expiratory flow limitation arising largely from loss of elastic recoil. As a consequence, in the patient with COPD and minimally reversible airway disease, peak airway pressures on the ventilator tend not to be extraordinarily high, yet autoPEEP and its consequences are common. At the time of intubation, hypoperfusion is common, as manifested by tachycardia and relative hypotension, and typically responds to briefly ceasing ventilation combined with fluid loading.

Because the majority of these patients are ventilated after days to weeks of progressive deterioration, the goal is to rest the patient (and respiratory muscles) for 36 to 72 h. Also, because the patient typically has an underlying compensated respiratory acidosis, excessive ventilation risks severe respiratory alkalosis and, over time, bicarbonate wasting by the kidney. Many such patients can be ventilated effectively with NIV, as described above. For those who require intubation, the goals of rest and appropriate hypoventilation can usually be achieved with initial ventilator settings of a V_T of 5 to 7 mL/kg and a respiratory rate of 20 to 24 breaths/min, with either an SIMV or an ACV mode set on minimal sensitivity. Because gas exchange abnormalities are primarily those of ventilation-perfusion mismatch, supplemental oxygen in the range of an F_{IO_2} of 0.4 should achieve better than 90% saturation of arterial hemoglobin.

The majority of patients with COPD will appear exhausted at the time when mechanical support is instituted and will sleep with minimal sedation. To the extent that muscle fatigue has played a role in a patient's functional decline, rest and sleep are desirable. Two to 3 days of such rest presumably will restore biochemical and functional changes associated with muscle fatigue, but 24 h is probably not sufficient. Small numbers of patients are difficult to rest on the ventilator, continuing to demonstrate a high work of breathing. Examination of airway pressure and flow waveforms can be very helpful in identifying this extra work, and

in suggesting strategies for improving the ventilator settings. In many patients, this is the result of autoPEEP-induced triggering difficulty. Adding extrinsic PEEP to nearly counterbalance the autoPEEP dramatically improves the patient's comfort.

Patients With Acute Hypoxemic Respiratory Failure

Acute hypoxemic respiratory failure is caused by alveolar filling with blood, pus, or edema, the end results of which are impaired lung mechanics and gas exchange. The gas exchange impairment results from intrapulmonary shunt that is largely refractory to oxygen therapy. In ARDS, the significantly reduced functional residual capacity arising from alveolar flooding and collapse leaves many fewer alveoli to accept the V_T , making the lung appear stiff and dramatically increasing the work of breathing. The ARDS lung should be viewed as a small lung, however, rather than a stiff lung. In line with this current conception of ARDS, it is now clearly established that excessive distention of the ARDS lung compounds lung injury and may induce systemic inflammation.^{13,14} Ventilatory strategies have evolved markedly in the past decade, changing clinical practice and generating tremendous excitement.

The goals of ventilation are to reduce shunt, avoid toxic concentrations of oxygen, and choose ventilator settings that do not amplify lung damage. The initial F_{IO_2} should be 1.0 in view of the typically extreme hypoxemia. PEEP is indicated in patients with diffuse lung lesions, but may not be helpful in patients with focal infiltrates, such as lobar pneumonia. In patients with ARDS, PEEP should be instituted immediately, then rapidly adjusted to the lowest PEEP necessary to produce an arterial saturation of 90% on an F_{IO_2} no higher than 0.6 ("least-PEEP approach"). An alternative approach is to set the PEEP at a value 2 cm H_2O higher than the lower inflection point of the inflation pressure-volume curve ("open-lung approach"), but this approach has not been validated, is rather complex, and is not recommended. Recruitment maneuvers have not been shown to be useful or necessary. The V_T should be 6 mL/kg on ACV; a higher V_T is associated with higher mortality. Presumably, PCV could be used as well, but the parameters that assure lung-protective ventilation are not known. In either mode, the respiratory rate should be set

at 24 to 36/min. An occasional consequence of lung-protective ventilation is hypercapnia. This approach of preferring hypercapnia to alveolar overdistention, termed "permissive hypercapnia," is very well tolerated.

The Patient With Restriction of the Lungs or Chest Wall

A small V_T (5 to 7 mL/kg) and rapid rate (18 to 24/min) are especially important in order to minimize the hemodynamic consequences of positive-pressure ventilation and to reduce the likelihood of barotrauma. The F_{IO_2} is usually determined by the degree of alveolar filling or collapse, if any. When the restrictive abnormality involves the chest wall (including the abdomen), the large ventilation-induced rise in pleural pressure has the potential to compromise cardiac output. This in turn will lower the mixed venous PO_2 and, in the setting of ventilation/perfusion mismatch or shunt, the PaO_2 as well. If the physician responds to this falling PaO_2 by augmenting PEEP or increasing the minute ventilation, further circulatory compromise ensues. A potentially catastrophic cycle of worsening gas exchange, increasing ventilator settings, and progressive shock is begun. This circumstance must be recognized because the treatment is to reduce dead space (eg, by lowering minute ventilation or correcting hypovolemia).

The Airway During Split-Lung Ventilation

The lungs may be separated for purposes of differential ventilation by two major means: (1) blocking the bronchus of a lobe or whole lung while ventilating with a standard endotracheal tube, or (2) passing a double-lumen tube (DLT). A number of different devices have been used to obstruct a bronchus, but experience is largest with the Fogarty embolectomy catheter. DLTs carry the advantages of allowing each lung to be ventilated, collapsed, re-expanded, or inspected independently.

Split-lung ventilation is only rarely useful in the critical care unit, but occasionally its benefits are dramatic. Large bronchopleural fistulas severely compromise ventilation and may not respond to HFV. A DLT will maintain ventilation of the healthy lung while facilitating closure of the bronchopleural fistula. During massive hemoptysis, lung separation may be lifesaving by minimizing blood aspiration,

maintaining airway patency, and tamponading the bleeding site while awaiting definitive therapy. Finally, patients with focal causes of acute hypoxemic respiratory failure, such as lobar pneumonia or acute total atelectasis, may benefit from differential ventilation and application of PEEP.

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