Mechanical Ventilation

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despite adequate ventilation), or both hypoxic and hypercapnic. In addition, respiratory failure can be caused by an inability to protect the airway.

Alterations in the normal physiology and anatomy of the respiratory system can lead to respiratory failure requiring mechanical ventilation. Anatomic alterations causing airway obstruction, such as tumors, edema, direct or indirect trauma, burns, or other such pathology, may result in respiratory failure. Central nervous system alterations caused by traumatic brain injury, intoxicants, and hemorrhagic or ischemic stroke can cause overt respiratory failure or loss of protective reflexes. Diseases of the peripheral nervous system may result in hypoventilation. Primary pulmonary diseases such as pneumonia can be manifested as ventilation-perfusion mismatch. Asthma and chronic obstructive pulmonary disease (COPD) can lead to hypercapnic respiratory failure. Cardiovascular disease may be accompanied by respiratory failure secondary to acute cardiogenic pulmonary edema, cardiac arrest, myocardial infarction, acute valvular insufficiency, cardiomyopathy, or arrhythmias. Finally, global states such as shock from any cause can lead to respiratory failure.

Presenting Signs and Symptoms

Patients requiring mechanical ventilation will typically be seen in extremis. Vital signs are paramount in the initial management. A rapid history and physical examination are also important. Patients may have a wide range of heart rate, respiratory rate (RR), and blood pressure. Pulse oximetry may be difficult to perform. Patients may complain of dyspnea, chest pain, anxiety, or generalized malaise. They may have altered mental status, tachypnea, hypopnea or apnea, diaphoresis, tachycardia, or bradycardia and occasionally arrive in full cardiac arrest. The history and physical examination should be focused on determining the need for mechanical ventilation and the cause of the respiratory failure.

Differential Diagnosis and Medical Decision Making

The decision to place someone on mechanical ventilation should be a clinical one performed at the bedside. Five basic questions can assist in determining the need for mechanical ventilation. First, is the patient failing to maintain an adequate
airway or protect the airway? Second, is adequate oxygenation being maintained? Third, is adequate ventilation being maintained? Fourth, is the patient’s expected clinical course such that intubation is indicated? “Yes” answers to any of these questions should prompt consideration for intubation. Finally, is the patient a candidate for noninvasive positive pressure ventilation (NPPV)? Selected patients may be given a trial of NPPV instead of intubation.

**TECHNIQUES AND METHODS OF MECHANICAL VENTILATION**

Mechanical ventilatory support may be provided through a noninvasive or invasive approach. Furthermore, each technique may be applied with a variety of ventilator modes. The key differences in ventilatory support are determined by the trigger, the limit, and the cycle. The trigger is the event that starts inspiration: either patient-initiated or machine-initiated respiratory effort. Limit refers to the airflow parameter that is regulated during inspiration: either airflow rate or airway pressure. The cycle terminates inspiration: either a set volume is delivered (volume-cycled ventilation [VCV]), a pressure is delivered for a set period (pressure-cycled ventilation [PCV]), or the patient ceases inspiratory effort (pressure support ventilation [PSV]).

The plethora of terms associated with mechanical ventilation can cause confusion and misunderstanding, especially because some terms are used interchangeably. Knowing a few simple terms can improve understanding and aid management. The ventilator can be set to reach either a target volume or a target pressure. Other terms used for this target are cycle and limit. Volume cycled, volume limited, and volume targeted all refer to the same thing. Similarly, pressure cycled, pressure limited, and pressure targeted also refer to the same mode. “Control” breaths are ventilator-initiated breaths. “Assist” breaths are patient-initiated breaths. Therefore, a ventilator that is set on volume-targeted (cycled, limited) assist/control (AC) mode has breaths that are initiated by the patient (assist breaths) and the ventilator (control breaths) and reaches a set volume target (cycle, limit).

**MODES OF INVASIVE MECHANICAL VENTILATION**

**CONTROL MODE**

Control mode ventilation (CMV) is used almost exclusively in anesthesia, but knowledge of this mode’s limitations aids in comprehension of other modes’ features (Fig. 3.1). In CMV, all breaths are triggered, limited, and cycled by the ventilator.

![Fig. 3.1 Control mode. Tidal volume, respiratory rate, inspiratory flow rate, FiO₂, and positive end-expiratory pressure are controlled. In this mode there is no synchronization with the patient’s respiratory effort.](image-url)
In volume-targeted mode, the physician selects a tidal volume (Vt), RR, inspiratory flow rate (IFR), fraction of inspired oxygen (FiO2), and positive end-expiratory pressure (PEEP). The machine then delivers positive pressure and applies as much pressure as required to deliver the set Vt at the set IFR. (In pressure-targeted mode the physician sets the pressure high, RR, FiO2, and pressure low or PEEP.) Note that patients can set their own flow rate in pressure-targeted modes. The machine then delivers positive pressure and applies as much pressure as required to reach the set pressure high. The Vt values generated are a function of respiratory system compliance. The patient is not able to initiate or terminate a breath. If inspiratory effort is initiated before the machine is triggered to deliver a breath, airflow would not occur regardless of the patient’s inspiratory effort. If exhalation is incomplete and the time for the machine to deliver a breath has occurred, the ventilator would provide as much pressure as necessary to cause inhalation. Imagine forcibly exhaling, or coughing, when the ventilator begins to deliver a breath. This lack of synchrony would cause distress and risk structural lung or airway injury. For these reasons, CMV is never used except for apneic, paralyzed, or anesthetized patients.

ASSIST/CONTROL MODE

AC mode usually provides the greatest level of ventilatory assistance (Fig. 3.2). In volume-targeted ventilation, the physician sets Vt, RR, IFR, FiO2, and PEEP. (In pressure-targeted mode, the physician sets the pressure high, RR, FiO2, and pressure low or PEEP.) In contrast to all other modes, the trigger that initiates inspiration depends on the patient’s inspiratory effort. When either occurs, the machine delivers the set Vt (in volume-targeted mode) or pressure high (in pressure-targeted mode). The machine follows a time algorithm that synchronizes the machine with patient-initiated breaths. If the patient is breathing at or above the set RR, all breaths are initiated by the patient. If the patient breathes below the set RR, machine-initiated breaths are interspersed among the patient’s breaths. Work of breathing (WOB) is primarily the effort that the patient exerts to cause airway pressure to drop to the threshold that triggers onset of the ventilator. (Manipulating the sensitivity of the ventilator sets this threshold.) Furthermore, WOB may be performed to a variable degree during inspiration, depending on how much the respiratory muscles are activated. WOB with the volume-targeted AC mode may be extreme in two situations: when the Vt drawn by the patient is greater than the set Vt and when the patient inspires at a rate that exceeds the set IFR (see later).

In the majority of situations, AC mode is used as described earlier and is termed volume-targeted or volume-cycled ventilation. As an alternative, some ventilators allow pressure-targeted (cycled) ventilation (PCV, not to be confused with PSV, described later) (Fig. 3.3). Instead of IFR, the limit during PCV is a set airway pressure. Instead of Vt, the cycle during PCV is a set inspiratory time (Ti). On some ventilator models, RR and the inspiratory-to-expiratory (I:E) ratio are set, and Ti is calculated from these settings. On other models, Ti is available as a setting. Because Vt is not set, the Vt delivered varies slightly from breath to breath, depending on lung compliance, airway resistance, and patient effort. PCV may offer a slight advantage over VCV in clinical scenarios that require control of the I:E ratio, but a body of literature investigating this concept does not exist. Historically, PCV was commonly used in neonates and infants, although modern ventilators that precisely measure small Vt are currently favored. PCV may be the only mode available on some portable and transport ventilators.

Synchronized Intermittent Mandatory Ventilation and Pressure Support

Synchronized intermittent mandatory ventilation (SIMV) is probably the most commonly misunderstood mode of mechanical ventilation (Fig. 3.4). The physician sets Vt, RR, IFR, FiO2, and PEEP, as in AC mode. In contrast to AC mode, however, the trigger that initiates inspiration depends on the...
patient’s RR relative to the set RR. When the patient breathes at or below the set RR, the trigger can be either elapsed time or the patient’s respiratory effort. In this case, WOB is equivalent to AC. When the patient breathes above the set RR, the ventilator is not triggered to assist in making spontaneous breaths in excess of the set RR. The work associated with such breaths may be quite high because the patient must generate enough negative force to pull air through the ventilator and overcome the resistance to airflow caused by the ventilator circuit tubing and the endotracheal tube (ETT), in addition to the WOB required as a result of the underlying disease process.

This limitation of SIMV can be diminished by the addition of PSV (Fig. 3.5). PSV causes inspiratory positive pressure to be applied during patient-initiated breaths that exceed the set RR. The patient initiates and terminates inspiration, thereby determining VT. Once the patient triggers pressure support, it is maintained until the machine detects cessation of patient effort, as indicated by a fall in inspiratory airflow. VT, IFR, and T1 are not controlled but instead are determined by patient effort. The WOB performed during PSV involves triggering the ventilator to deliver the pressure and maintaining inspiratory effort throughout inhalation. Contrast this with machine-assisted ventilation in AC or SIMV, where WOB involves triggering the ventilator but lung inflation continues regardless of the patient’s inspiratory effort. WOB during PSV also depends on the set level of pressure support. Insufficient pressure support is associated with high WOB, which leads to a small VT and a high RR. Adequate pressure support reduces WOB and improves VT and RR. Many experts view RR as the best index of the adequacy of the level of pressure support. It should be adjusted to maintain an acceptable RR of less than 30 but preferably less than 24 breaths per minute.

SIMV can be used in pressure-targeted ventilation. Essentially, the ventilator is set to reach a target pressure for each of the ventilator-initiated breaths and potentially a different target for patient-initiated breaths. Another way to consider pressure-targeted SIMV is as PSV with a set rate.

**CONTINUOUS POSITIVE AIRWAY PRESSURE**

CPAP alone is not a true form of assisted mechanical ventilation because inspiration is not assisted by increasing airway pressure. Pressure greater than ambient atmospheric pressure is supplied, but it is held constant throughout the respiratory cycle. During inhalation, the gradient between the airway and intrathoracic pressure is higher than it would be if breathing ambient air. Conversely, the gradient is lower during exhalation. As a result, inhalation requires slightly less effort than normal breathing does, and the airways are held open during exhalation to allow better expiratory airflow. As in SIMV, PSV may be added to CPAP. CPAP with PSV is a form of assisted ventilation because inspiratory pressure is augmented, and this is more appropriately referred to as simply PSV. As discussed earlier, the patient initiates and terminates each breath; therefore, WOB is performed as the patient initiates each breath and maintains inspiratory effort throughout inhalation.

**OTHER MODES**

The most recent innovations in ventilator modes are those that combine volume and pressure targets, which are referred to as dual modes. Pressure-regulated volume control, autoflow, volume ventilation plus, adaptive support ventilation, variable pressure control, and variable pressure support are all dual modes that adjust pressure or volume targets from breath to breath to reach the goals desired. Volume-ensured PSV and pressure augmentation alter parameters within the breath to reach goals. Unfortunately, few studies have compared these latest modes with one another or with conventional modes.

Finally, modes that have rarely been used in the ED setting and are beyond the scope of this chapter include high-frequency ventilation, airway pressure release ventilation, bilevel ventilation, proportional assist ventilation plus, and proportional pressure support.
MONITORING DYNAMIC PRESSURE DURING INVASIVE VENTILATION

Mechanical ventilation can cause damage to the lungs on a macroscopic and microscopic level. The direct cause of lung injury is believed to be a combination of overdistention of the alveoli and repetitive alveolar opening and closing with shear of the alveolar wall. The concept of ventilator-induced lung injury (VILI) has evolved to encompass all forms of injury at the organ and alveolar level, including pneumothorax, pneumomediastinum, bronchial rupture, diffuse alveolar damage, and acute respiratory distress syndrome (ARDS). Pressure is measured at the ventilator end of the circuit (the proximal part of the airway), and this measurement is used as an index of the pressure within the lung.

PEAK INSPIRATORY AIRWAY PRESSURE

Peak inspiratory airway pressure (P_{peak}) is the highest pressure that is generated during inflation of the lung. Because pressure decreases incrementally along the path at each point of resistance, the pressure delivered at the alveolar level may be significantly less than the measured P_{peak}, particularly when resistance to airflow is high. Therefore, P_{peak} is not an ideal surrogate measurement for alveolar pressure and does not correlate with VILI.

PLATEAU PRESSURE

Plateau pressure (P_{plat}) is the end-inspiratory airway pressure and is measured just after airflow has ceased. Because this is a static measurement (absence of airflow), resistance of the circuit and airways does not play a role. Therefore, P_{plat} is a logical surrogate measurement for mean alveolar pressure. Its primary limitation is that compliance is not equal in all regions of the lung. The degree of alveolar distention in healthy regions of the lung may be significantly greater than that in heavily diseased lung regions at the same P_{plat}. In a healthy adult undergoing mechanical ventilation with normal lung compliance, P_{plat} is low, usually in the range of 5 to 15 cm H_{2}O. Patients with alveolar disease (pneumonia, cardiogenic pulmonary edema, acute lung injury [ALI], and ARDS) have poor lung compliance, and P_{plat} is typically much higher in these states. Measures to maintain P_{plat} below the currently recommended limit of 30 cm H_{2}O are discussed later.

INTRINSIC POSITIVE END-EXPIRATORY PRESSURE

PEEP indicates that the airway pressure measured at the end of exhalation is above ambient air pressure. When PEEP is set by the clinician and applied by the ventilator, it is termed extrinsic PEEP (PEEP_{x}). In contrast, intrinsic PEEP (PEEP_{i}) arises when exhalation is incomplete because of either intrathoracic airway obstruction, early airway closure during exhalation, or inadequate exhalation time. The common end point is trapping of air in the lung at the end of exhalation, which ultimately leads to increased intrathoracic pressure. PEEP_{i} can cause problems through several mechanisms. First, because exhalation is incomplete, air is progressively being trapped in the lungs, thereby leading to early airway closure and dynamic hyperinflation with an associated risk for VILI. Second, PEEP_{i} leads to difficulty triggering the ventilator and increased WOB, as discussed previously. Third, PEEP_{i} can cause patient-ventilator dyssynchrony when the patient continues active contraction of the respiratory muscles at end exhalation as the ventilator is triggered. Lung inflation may begin while the patient is attempting to complete exhalation. Finally, increased intrathoracic pressure can impede venous return to the heart and consequently lead to hemodynamic instability. Simultaneously, impaired venous return may compromise pulmonary blood flow, increase physiologic dead space, and result in worsening hypercapnia. Control of PEEP_{i} is discussed later.

MODES OF NONINFRINGEMENT MECHANICAL VENTILATION

The cause of the respiratory failure is the best predictor of whether a patient will respond to noninvasive techniques. The literature supports the application of NPPV for certain conditions—COPD, asthma, congestive heart failure (CHF), pneumonia, trauma, cancer, and neuromuscular disease—as well as for pediatric patients.

Noninvasive ventilators are more portable because of the use of a smaller air compressor/blower, but they cannot develop pressures as high as larger critical care ventilators can. A noninvasive ventilator can provide up to 20 to 40 cm H_{2}O of air pressure, as compared with critical care ventilators capable of delivering greater than 100 cm H_{2}O of air pressure. Newer noninvasive ventilators can be set for volume- or pressure-targeted mode, AC or SIMV, and even proportional assist.

SPONTANEOUS AND SPONTANEOUS/TIMED MODES

In spontaneous mode, airway pressure cycles between inspiratory positive airway pressure (IPAP) and expiratory positive airway pressure (EPAP). This mode is commonly referred to as biphasic (or bilevel) positive airway pressure, but other proprietary names refer to the same mode. The trigger to switch from EPAP to IPAP is the patient’s inspiratory effort. A variety of ventilator models use one or several of the following to indicate patient effort: a drop in airway pressure, measured inspired volume (usually 5 to 6 mL), or an increase in airflow rate. The limit during inspiration is the set level of IPAP. The inspiratory phase cycles off when the machine senses cessation of patient effort, as indicated by a decrease in inspiratory flow below a set threshold (typically 60% of the peak IFR) or attainment of maximum inspiratory time (usually 3 seconds). The latter is a safety mechanism to prevent lung hyperinflation as a result of ventilator “runaway,” but it was not available on early generations of noninvasive ventilators. VT may vary from breath to breath, dependent primarily on the magnitude and duration of patient effort, but also on lung compliance. WOB is predominantly related to initiating and maintaining airflow throughout the inspiratory phase. Additional WOB may occur if the patient actively contracts the expiratory muscles.

Spontaneous mode is dependent on the patient’s effort to trigger inhalation. Respiratory acidosis will develop in a patient breathing at a slow, inadequate rate. To prevent this adverse consequence, spontaneous/timed mode allows the machine to be triggered either by patient effort or after an elapsed time interval that is calculated from a set minimum
RR. If the patient does not initiate inspiration during the set interval, IPAP is triggered. For machine-initiated breaths, the machine cycles back to EPAP based on a set inspiratory time. For patient-initiated breaths, the ventilator cycles as it would in spontaneous mode.

Pragmatically, NPPV (noninvasive) and PSV (invasive) are similar but have a few noteworthy differences. First, the trigger for PSV is a drop in airway pressure sensed by the ventilator. Some ventilators monitor airflow in the inspiratory and expiratory limbs of the ventilator circuit and will be triggered if airflow in the inspiratory limb is greater than airflow in the expiratory limb. The sensitivity of the trigger can be adjusted on a conventional ventilator by setting the magnitude of the change in pressure required for triggering. This is contrasted with NPPV, in which sensitivity is continuously and automatically adjusted by the noninvasive ventilator based on the amount of air leak and is not able to be adjusted by the physician. Second, because PSV is supplied by a critical care ventilator, leaks are not tolerated or compensated. Because airflow through a leak may be misinterpreted in this mode as patient inspiratory effort, a leak may lead to early triggering before exhalation is complete. Leaks may also cause failure to cycle off in synchrony with cessation of patient effort. These phenomena are less likely to occur when using a noninvasive ventilator. Finally, the nomenclature used for airway pressure is different. Pressure during the expiratory phase is termed PEEP, analogous to the EPAP of NPPV. Pressure during the inspiratory phase is termed peak inspiratory pressure, analogous to the IPAP of spontaneous mode. The distinction is that in PSV the numerical value for pressure support is the equivalent of the difference between IPAP and EPAP.

INITIATION OF NONINVASIVE POSITIVE PRESSURE VENTILATION

The process of initiating a trial of noninvasive ventilatory support consists of four basic steps. First, the patient must be willing to accept face mask ventilation. Because the patient should remain awake and cooperative during ventilation, the process should be explained before the mask is applied. Initially, an FiO₂ of 100% with 3 to 5 cm H₂O of CPAP is provided. Acceptance may improve if the patient holds the mask against the face. The mask is secured with straps once the patient demonstrates acceptance.

Next, after explaining that the pressure will change, ventilation is switched to NPPV with an EPAP of 3 to 5 cm H₂O and an IPAP of 8 to 10 cm H₂O. IPAP is titrated in 2- to 3-cm H₂O increments until exhaled Vₜ (measured by the ventilator) is in the range of 6 to 9 mL/kg IBW. Further adjustment of IPAP should be directed toward obtaining an RR of less than 30. Another option is to start with high IPAP (20 to 25 cm H₂O) and titrate down based on patient comfort. Of note, no studies have compared a low-to-high IPAP versus a high-to-low IPAP approach.

EPAP is then adjusted to the lowest level that allows synchrony between the patient and ventilator. Understanding this process requires review of the components of WOB related to triggering the ventilator. The patient activates the inspiratory muscles to decrease intrathoracic pressure. As intrathoracic pressure falls below airway pressure, transpulmonary pressure becomes positive, airflow begins, and the ventilator is triggered. In a normal patient, the inspiratory muscle force required to lower intrathoracic pressure to a level that triggers the ventilator is not great. In a patient with high PEEP, (also known as auto-PEEP), intrathoracic pressure is high at end exhalation. The inspiratory muscle force required to lower intrathoracic pressure below airway pressure is significantly greater. Thus the WOB that is performed to trigger the ventilator is proportional to the amount of PEEP, that is present.

While delivering NPPV, it is impossible to measure PEEP without invasive means. Instead, to detect PEEP, signs of difficulty triggering the ventilator or signs of expiratory airflow obstruction should be sought. On physical examination, recruitment of the accessory muscles of inspiration suggests that PEEP, is a problem. A useful technique is palpation of the sternocleidomastoid muscle while simultaneously watching the ventilator flow graphs or listening for the ventilator to trigger. When the muscle is felt to contract before the ventilator triggers, PEEP, may be the culprit. Observation of active abdominal muscle recruitment during exhalation indicates airflow obstruction as a cause of elevated PEEP. When elevated PEEP, is suspected, EPAP should be increased in increments of 2 to 3 cm H₂O until the problem is controlled. The maximum safe level of EPAP that should be used during NPPV has not been determined in an evidence-based manner. Typical initial settings range from 0 to 5 cm H₂O; maximum settings described in the methods sections of various trials range from 12.5 to 15 cm H₂O. It is prudent to measure the heart rate and blood pressure and perform pulse oximetry after each increase in EPAP because high levels may compromise cardiac output. As EPAP is increased, corresponding increasing increments in IPAP are required to maintain a differential between EPAP and IPAP that ensures adequate VT.

Finally, FiO₂ is adjusted to maintain adequate O₂ saturation. In many clinical situations, continuous pulse oximetry alone is adequate for this purpose. Arterial blood gas determinations are not routinely required but may be helpful in select patients to assess improvement in respiratory acidosis.

SPECIFIC DISEASE PROCESSES

CONTROLLING AIRWAY PRESSURE—LUNG-PROTECTIVE VENTILATOR STRATEGIES

Causes of difficulty with mechanical ventilation fall into four general categories:

- High airway pressure during lung inflation
- High PEEP, because of obstructive airways disease
- Patient-ventilator dyssynchrony
- Equipment failure

Acute Respiratory Distress, Acute Lung Injury, and Pulmonary Edema

Elevated plateau pressure is encountered in patients with poor lung compliance as a result of parenchymal lung disease (e.g., pulmonary edema, either cardiogenic or noncardiogenic) or obstructive airways disease with air trapping. The goal is to support the respiratory system while avoiding iatrogenic injury.

Initial studies compared a conventional ventilation strategy (Vₜ of 10 to 15 mL/kg IBW with a goal of obtaining normal PaO₂ and PaCO₂) with a lung-protective ventilation strategy.
Obstructive Airways Disease

Exacerbation of obstructive airways disease requiring mechanical ventilation is often associated with air trapping and dynamic hyperinflation of the lungs. High $P_{\text{peak}}$ arises as a result of inspiratory airflow resistance, a phenomenon more common in patients with severe asthma than in those with COPD. High $P_{\text{plat}}$ is caused by lung overdistention and consequent diminished compliance. Patients with both high $P_{\text{peak}}$ and $P_{\text{plat}}$ comprise a group of high-risk patients with both obstruction and overdistention who are at high risk for complications, including pneumothorax, tension pneumothorax, pneumomediastinum, dysrhythmias, and hemodynamic collapse. No prospective trials comparing ventilation strategies in such patients have been conducted. It is common practice to use a strategy of permissive hypercapnia to eliminate PEEP, and avoid high $P_{\text{plat}}$. This strategy makes use of low VT, low RR, and high IFR to shorten the inspiratory time and prolong the expiratory time. Although this strategy often leads to hypercapnia, it is considered safer to allow respiratory acidosis to develop than to ventilate at excessive airway pressure. A lower limit of acceptable pH has not been established, but general recommendations have been to allow pH values as low as 7.15 to 7.2. Permissive hypercapnia is required more often in the management of status asthmaticus than in the management of COPD. Evidence in support comes from retrospective studies. Current recommendations include VT less than 8 mL/kg IBW, an initial RR of 8 to 10 cycles/min, and $F_{\text{I,O}_2}$ adjusted to obtain a $Pa_{\text{O}_2}$ of approximately 60 mm Hg or a $Pa_{\text{CO}_2}$ of 85% to 88%. The concept of permissive hypercapnia and controlled hypoventilation in the management of acute asthma exacerbation has been widely accepted.\(^{15}\) Some reports suggest that $P_{\text{peak}}$ and $P_{\text{plat}}$ are not adequate indicators of pulmonary hyperinflation\(^{16}\) and recommend that expiratory volumes be measured in these patients.\(^{17}\) This latter technique has not gained widespread acceptance, however.
**BOX 3.1 Complications of Mechanical Ventilation**

- Pneumothorax
- Auto-PEEP, dynamic hyperinflation, breath stacking
- Decreased cardiac output and blood pressure
- Vocal cord damage
- Tracheal stenosis
- Unplanned extubations
- Ventilator-associated pneumonia

**PEEP, Positive end-expiratory pressure.**

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**COMPlications**

**INVASIVE MECHANICAL VENTILATION**

In the ED, complications of mechanical ventilation (Box 3.1) can begin during the preintubation period. Induction agents may cause or worsen hypotension. Overly aggressive bag-valve-mask ventilation may lead to decreased venous return and hypotension. Airway trauma and mechanical complications may be caused by the act of intubation. Initiating mechanical ventilation and transitioning from negative pressure ventilation to positive pressure ventilation may lead to hypotension. Positive pressure ventilation may worsen an existing pneumothorax or give rise to pneumothorax. Auto-PEEP (also known as PEEP), dynamic hyperinflation, breath stacking can lead to hypotension and circulatory collapse. Ventilator-associated lung injury can be caused by baro-trauma, volutrauma, or trauma related to atelectasis. Long-term complications can include inability to be liberated from the ventilator, ventilator-associated pneumonia, tracheal stenosis, and vocal cord injury.

Some of the commonly underrecognized problems that arise in the support of critically ill patients fall into the category of patient-ventilator dyssynchrony. These situations can markedly increase WOB and lead to increased CO₂ and lactic acid production with both respiratory and metabolic acidosis.

**INTRINSIC POSITIVE END-EXPIRATORY PRESSURE**

Maneuvers directed at elimination of PEEP, have in common the effect of decreasing inspiratory time and therefore providing more expiratory time. Decreasing RR and VT and increasing IFR effectively accomplish this goal. Frequently, this cannot be achieved without sedation, sometimes requiring the addition of pharmacologic paralysis.

**Difficulty Triggering the Ventilator**

To trigger a ventilator, a patient must cause either a drop in pressure or an increase in airflow at the proximal part of the airway, depending on the type of ventilator in use. The magnitude of change required to trigger the ventilator is adjusted by setting the sensitivity, usually in the range of −1 to −2 cm H₂O below the level of PEEP. Difficulty triggering the ventilator is often not easy to detect. When it becomes obvious by physical examination that the patient is using the accessory muscles of respiration to trigger the ventilator, the problem may be severe. The condition can be detected earlier by inspecting the pressure-volume time curve on the ventilator display. A large negative deflection at the beginning of inhalation suggests that ventilator sensitivity needs to be increased.

More commonly, high PEEP, is the cause. The patient must first lower intrathoracic pressure enough to overcome PEEP, before airway pressure can drop to the threshold sensitivity. The solution to this problem is to raise PEEP, to a level one half to three fourths of PEEP, and allow the patient to perform less work to trigger inhalation. This process mandates frequent reassessment of PEEP, and manipulation of the ventilator during this dynamic period.

**Autocycling**

Autocycling refers to a phenomenon in which the ventilator set in AC mode begins to rapidly trigger without the patient initiating respiration. The cause is usually oscillations in airway pressure that the ventilator interprets as patient effort. Tremors, shivering, voluntary motion, convulsions, and oscillating water in the ventilator circuit are all examples of potential causes. Autocycling should prompt immediate disconnection from the ventilator circuit and ventilation with a bag-valve device until the problem has resolved.

**Rapid Breathing**

When attempting to ventilate a patient with an obstructive process, the goal is to eliminate PEEP. Permissive hypercapnia is best achieved at low RR, but at the same time hypercapnia is a powerful stimulus to breath. This can typically be quelled by using a combination of sedatives such as benzodiazepines in combination with opiates. Neuromuscular blockade should be considered a last resort undertaken only after careful consideration of the risks associated with prolonged paralysis and the potential development of neuropathy in patients with critical illness. If undertaken, it should be done only to weaken the patient sufficiently to inhibit dyssynchrony with the ventilator. Other common causes of rapid breathing include sepsis, pulmonary emboli, pregnancy, hepatic encephalopathy, intracranial hypertension, stroke or hemorrhage, and posthypercapnic status. Some of these conditions are appropriate physiologic responses, whereas others, though pathologic, are difficult to control and occasionally tolerated.

**Outstripping the Ventilator and Double Cycling**

In patients undergoing low-VT ventilation for ARDS or for an obstructive process, hypercapnia and an increased respiratory drive will develop. Outstripping the ventilator refers to the patient’s effort to draw a higher VT than is set while in a volume-targeted mode. This can be detected by observing the exhaled VT or by finding a negative deflection at the end of inhalation on the pressure-volume time plot. Double cycling occurs when the patient desires a larger VT than is set and continues to inspire despite the delivery of a breath. The ventilator will then provide a second breath almost immediately after the first. This is especially problematic because the actual VT delivered is twice the set volume. As with controlling rapid breathing, the solution is sedation and analgesia, particularly with opiates. In addition, switching to a pressure-targeted mode or increasing the set VT may alleviate this issue.
**Straining over the Ventilator**

Straining over the ventilator indicates that the patient is attempting to inhale at a flow rate in excess of the set IFR on a volume-targeted mode. When it is obvious by examination that the patient is actively inhaling, the problem may be severe. On the pressure-volume time plot the rise in pressure during inhalation will be concave rather than convex. Potential solutions are to raise the IFR, switch to pressure-targeted mode or PSV, or use sedation and analgesia.

**Coughing**

Coughing is a common problem that can arise from increased secretions, a foreign body in the airway (ETT), or the underlying pulmonary disease process. Coughing can lead to autocycling, poor patient comfort, ETT dislodgment, and rarely airway injury. Placement of the ETT above the carina should be confirmed. Suctioning plus provision of warmed, humidified air is often helpful. If these simple measures fail to provide relief, aerosolized lidocaine or suppression with opiates may increase patient comfort.

**EQUIPMENT FAILURE**

Whenever a patient decompensates while receiving mechanical ventilation, consideration should be given to equipment failure as the cause. Interruption of the oxygen supply, accidentally rotated knobs, disconnected ventilator circuitry, and obstructed tubes are all potential culprits. Immediate action should include disconnection from the ventilator and bag ventilation with 100% O₂. The mnemonic made popular by the American Heart Association’s Pediatric Advanced Life Support Course is useful to recall the causes of unexpected decompensation: DOPE (dislodgment of the ETT, obstruction of the tube, pneumothorax, and equipment failure). Confirmation of ETT placement, suctioning via an endotracheal catheter, auscultation, chest radiography, and equipment troubleshooting are necessary actions.

**NONINVASIVE MECHANICAL VENTILATION**

The main complication of NPPV is an inability to tolerate the mask or the pressure. Long-term complications can include an inability to eat or drink, nasal and oral dryness, and pressure necrosis on the bridge of the nose, the cheeks, or the chin or above the ears.

**PROGNOSIS**

Because of the wide range of causes of respiratory failure, the prognosis is highly variable and dependent on the cause and severity. The prognosis of patients with respiratory failure caused simply by oversedation from intoxicants can be quite good. Conversely, patients with ARDS as their sole organ dysfunction have a mortality of 20% to 25%. Respiratory failure with multiorgan system failure carries much higher mortality that is based on the severity of the illness. Overall, a requirement for invasive mechanical ventilation carries an approximate 34.5% in-hospital mortality.²

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**TIPS AND TRICKS**

**Pitfalls and Pearls for Mechanical Ventilation**

**Pitfalls**

- Not considering NPPV
- Not using NPPV early enough
- Not having the personnel or time to adequately monitor and make adjustments
- Not adjusting pressures quickly enough

**Pearls**

- Early use of NPPV should be considered in a wide variety of patients.
- Proper patient selection is paramount.
- Selection of the interface and adjustment of parameters are crucial for success.
- A team approach with close observation of the patient is vital.

*NPPV, Noninvasive positive pressure ventilation.*

**SUGGESTED READINGS**


**REFERENCES**

References can be found on Expert Consult @ www.expertconsult.com.
REFERENCES


