Mechanical Ventilation in Emergency Medicine

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Introduction

Patients with severe respiratory distress are frequently encountered in the practice of emergency medicine. Respiratory complaints account for about 12% of ED visits (1). Between 1992 and 1999, the rates of ED visits for asthma rose 26%, for pneumonia 12%, and for chest pain 50% (2). In a study of near fatal asthma attacks, 26% of asthmatic patients who required intubation reported the ED as their primary source of health care (3). Clearly, a thorough knowledge of mechanical ventilation, lung protective ventilatory strategies, and alternatives to invasive mechanical ventilation is essential to the practice of emergency medicine.

Modes of Mechanical Ventilation

Mechanical ventilatory support (MVS) can be provided using a variety of ventilator modes. The key to understanding the differences in the degree of support that each mode will provide lies in three variables: the trigger, limit, and cycle. The *trigger* is the event that begins inspiration, either patient-initiated respiratory effort or machine-initiated positive pressure. The *limit* refers to the parameter that limits airflow during inspiration, either a flow rate or a set pressure. The *cycle* ends inspiration, either when a set volume is delivered (volume cycled ventilation), a pressure is delivered for a set period of time (pressure cycled ventilation), or the patient ceases inspiratory effort (pressure support ventilation).

Control Mode

Control mode ventilation (CMV) is used almost exclusively in anesthesia, but knowledge of this mode's limitations aids in the comprehension of other modes' features. In control mode, all breaths are triggered, limited, and cycled by the ventilator. The physician selects a tidal volume (Vt), respiratory rate (RR), inspiratory flow rate (IFR), fraction of inspired oxygen (FiO2), and positive end expiratory pressure (PEEP). The machine then delivers positive pressure based on a time interval, at whatever pressure is required to deliver the set Vt (the cycle) at the set IFR (the limit) (Figure 1). The patient is unable to initiate or terminate a breath. If an inspiratory effort were initiated before the machine is triggered to deliver a breath, then regardless of the patient's inspiratory force, airflow would not occur. Imagine sucking on an empty coke bottle, and the concept is suddenly evident. Furthermore, if exhalation is incomplete, yet the time for the machine to deliver a breath occurs, then the ventilator provides whatever pressure necessary to cause inhalation. Imagine forcibly exhaling, or coughing, when the ventilator triggers to deliver a breath. The lack of synchrony would cause distress and risk structural lung or airway injury. For these reasons, CMV is never used except in apneic, paralyzed, anesthetized patients.

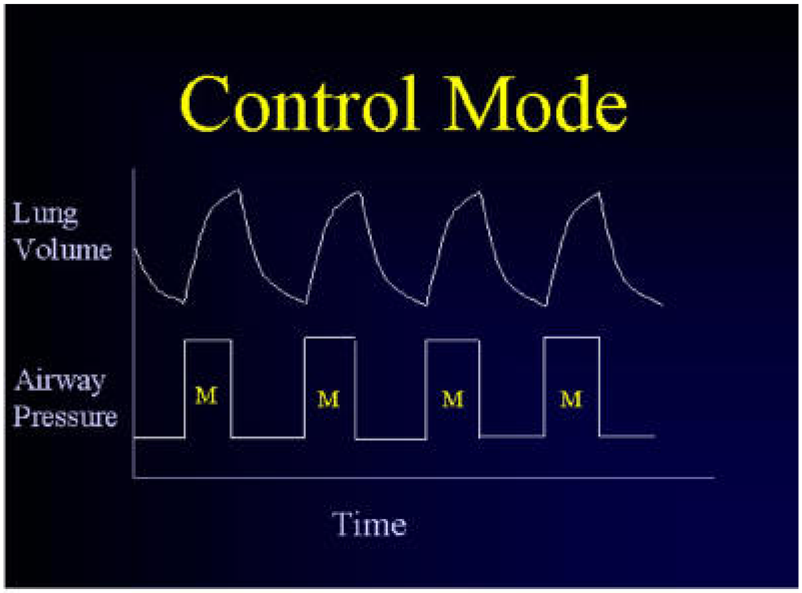


Figure 1
M = machine initiated breath

Assist Control Mode

Assist control (AC) mode usually provides the highest level of ventilatory assistance. The physician sets Vt, RR, IFR, FiO2, and PEEP. In contrast to all other modes, *the trigger that initiates inspiration can be either the ventilator, or the patient's respiratory effort*. When either event occurs, the machine cycles on and delivers the set Vt (Figure 2). The machine follows a time algorithm that synchronizes it with patient initiated breaths. If the patient is breathing at or above the set RR, then all breaths are patient initiated. If the patient breathes below the set RR, then machine-initiated breaths are interspersed among the patient's breaths. The work of breathing (WOB) is primarily the effort the patient produces that causes the airway pressure to drop to the threshold that triggers the ventilator. (Manipulating the ventilator sensitivity sets this threshold.) Further work of breathing occurs to variable degree during inspiration, depending upon how much the respiratory muscles are used. This may be particularly significant in 2 instances: when the patient draws a tidal volume greater than the set Vt, and when the patient inspires at a rate that exceeds the set IFR.

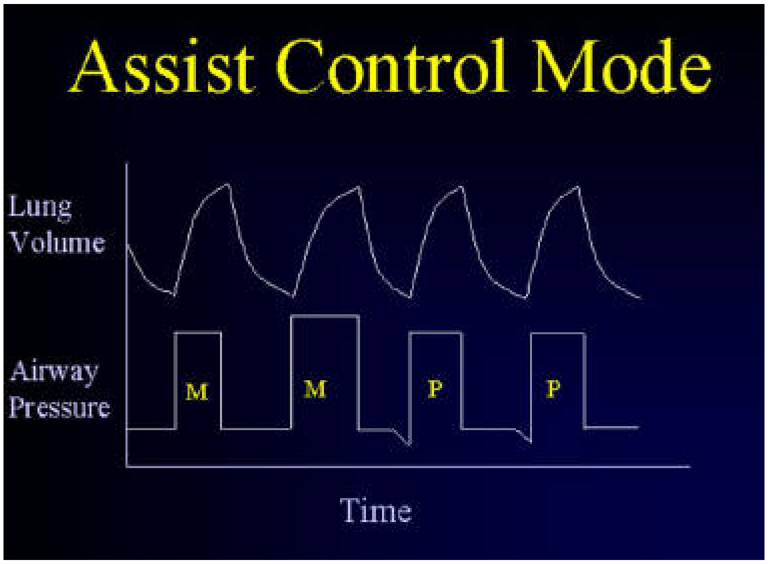


Figure M = machine initiated breath

P = patient initiated breath

Synchronized Intermittent Mandatory Ventilation

Synchronized intermittent mandatory ventilation (SIMV) is probably the most commonly misunderstood mode of MVS. The physician sets Vt, RR, IFR, FiO2, and PEEP, just as in AC. In contrast, the trigger that initiates inspiration depends upon the patient's RR relative to the set RR. *When the patient breathes at or below the set RR*, the trigger can be either the machine or the patient's respiratory effort. In that case, the work of breathing is equivalent to AC.

If the patient breathes above the set RR, the ventilator does not trigger to assist the spontaneous breaths in excess of the set RR (Figure 3). In fact, the work associated with such breaths is increased. In order to draw a breath above the set RR, the patient must generate enough negative force to pull air through the ventilator; to overcome the resistance to airflow due to the ventilator circuit tubing, the endotracheal tube, and the patient's airways; and to expand the chest cavity against the elastic recoil of the lungs and chest wall.

This limitation of SIMV can be diminished by addition of pressure support ventilation (PSV). PSV is positive pressure added 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 senses cessation of patient effort, indicated by a fall in IFR (Figure 4). Vt, IFR, and Ti are not controlled, but are determined by patient effort. The work of breathing that a patient performs during PSV involves triggering the ventilator to deliver the pressure *and maintaining inspiratory effort throughout inhalation*.

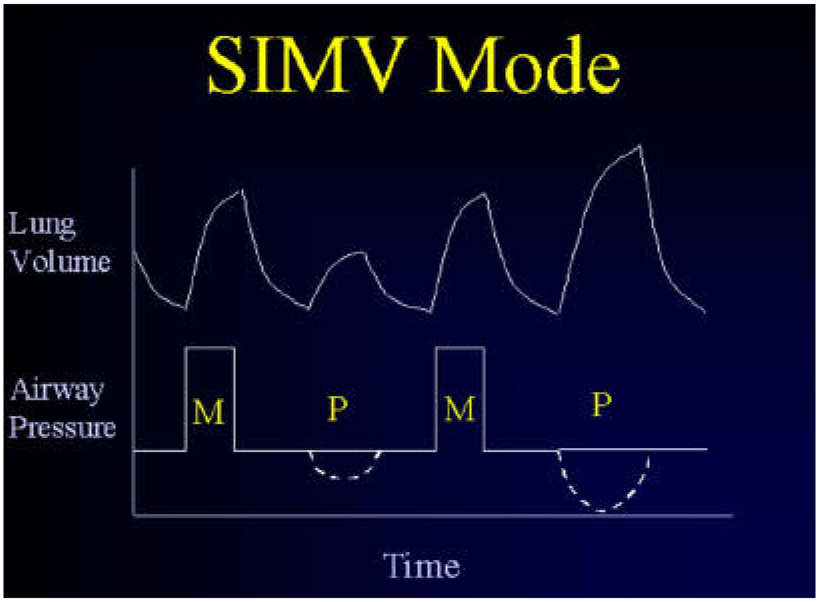


Figure 3
M = machine initiated breath
P = patient initiated breath
Dashed line = patient inspiratory effort

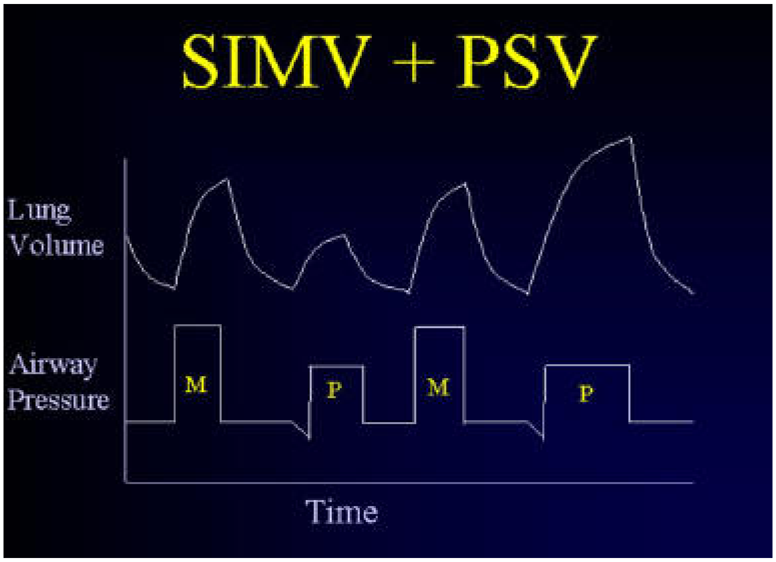


Figure 4
M = machine initiated breath
P = patient initiated breath

Continuous Positive Airway Pressure

Continuous Positive Airway Pressure (CPAP) is not a true form of assisted mechanical ventilation, because added inspiratory pressure or volume is not provided. Pressure above the ambient atmospheric pressure is supplied, but is held constant throughout the respiratory cycle (Figure 5). During inhalation, the gradient between the airway and alveolar pressure is higher than it would be if breathing ambient air. Conversely, the gradient is lower during exhalation. Inhalation requires slightly less effort, and the airways are held open during exhalation.

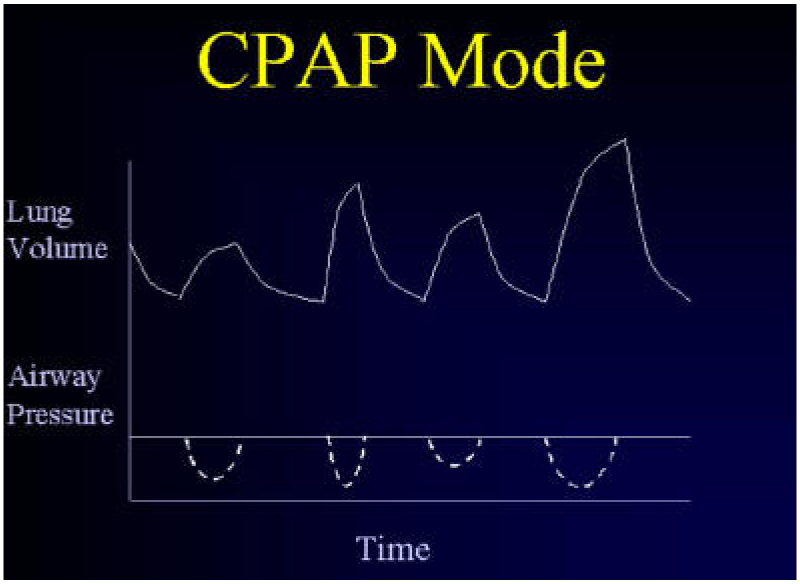


Figure 5
M = machine initiated breath
P = patient initiated breath
Dashed line = patient inspiratory effort

Selection of a Ventilator Mode

The vast majority of patients who require intubation in the emergency department have respiratory failure, hemodynamic instability, altered level of consciousness, or a combination of the three. The goal of mechanical ventilation is to take over the work of breathing. Since use of AC mode ensures that every breath is assisted, AC accomplishes this goal most effectively. Therefore it is the initial mode of choice in most emergency department scenarios. In selected cases, when the reason for intubation is protection of the airway, and the patient's drive to breath is intact, CPAP or CPAP with PSV may be appropriate. Many patients with acute respiratory failure have evolving, dynamic disease processes. Use of SIMV under such conditions risks increasing the WOB, as described above.

Monitoring Ventilator Pressures

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 over distention of the alveoli and repetitive alveolar opening/closing with shear of the alveolar wall. Controversy and lack of consensus regarding the cause of lung injury has led away from use of the terms barotrauma and volutrauma. 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, alveolar damage, and ARDS. The ability to monitor pressure at the alveolar level might allow the clinician to avoid dangerously high pressure, over distention, and VILI. However, it is impossible with current technology to directly measure air pressure within the distal airway or the alveolus. Instead, pressure is measured at the ventilator end of the circuit (the proximal airway), and the measurement used as an index of the pressures within the lung.

Peak Inspiratory Airway Pressure

The peak inspiratory airway pressure (P_{peak}) is the highest pressure that is generated during inflation of the lung (Figure 6). In order to cause airflow, the ventilator generates enough pressure to overcome the resistance of the ventilator circuit tubing, the endotracheal tube, the patient's large and small airways, and the elastic recoil of the lung, pleura, and chest wall.

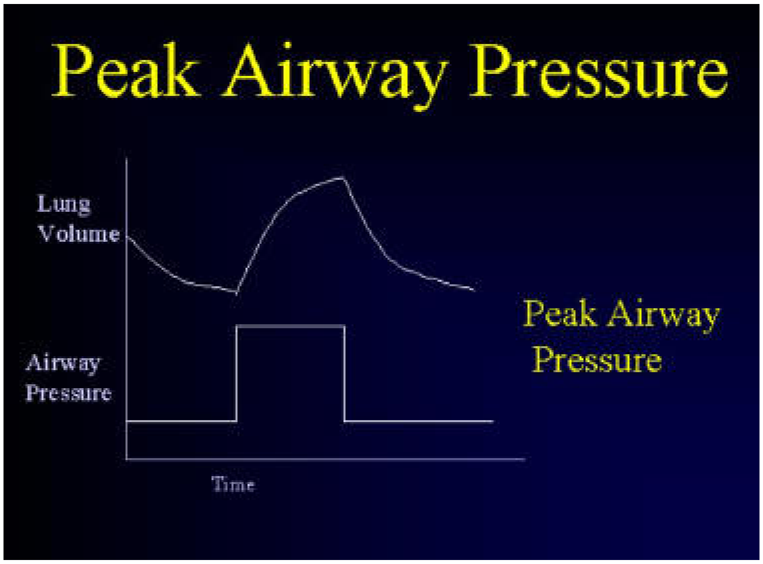


Figure 6

Since pressure decreases incrementally along the path at each point of resistance (Figure 7), the pressure delivered at the alveolar level may be significantly less than the measured P_{peak}, particularly when there is high resistance to airflow. Therefore, P_{peak} is not an ideal surrogate measurement for alveolar pressure.

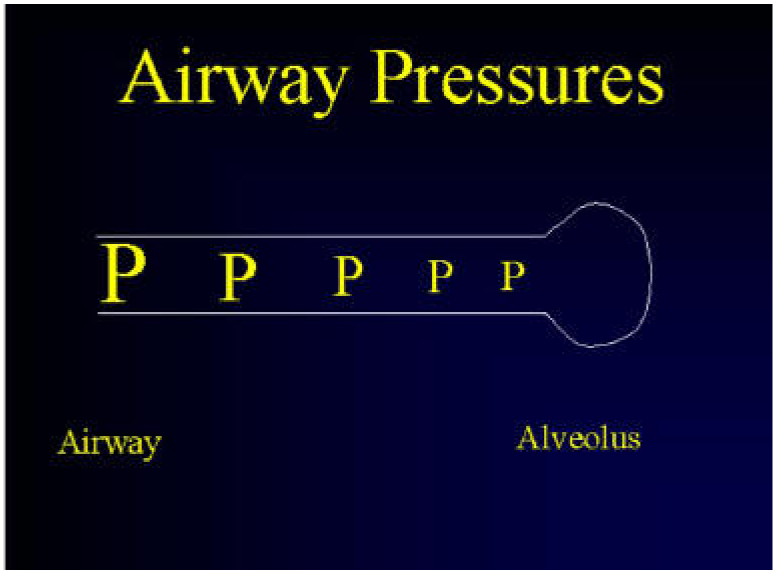


Figure 7

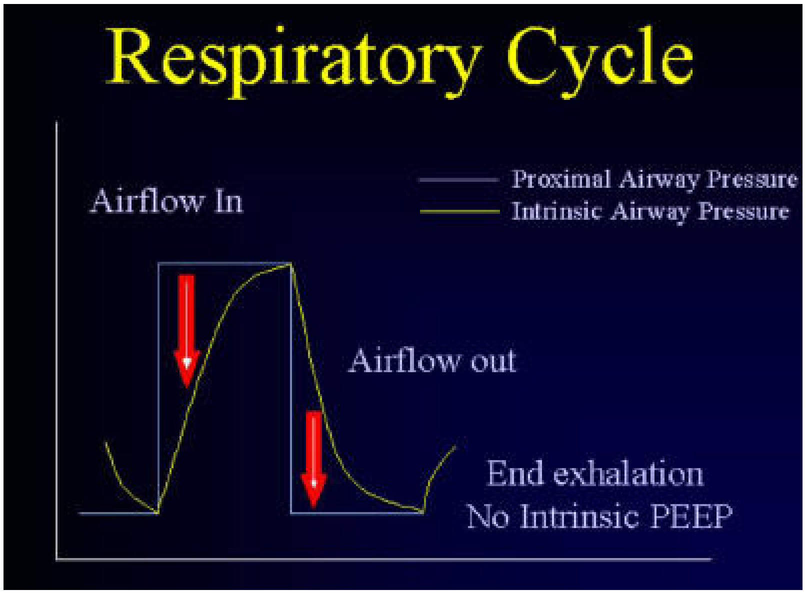
Plateau Pressure

Plateau pressure (P_{plat}) is the end inspiratory airway pressure, measured just after airflow has ceased. Because this is a static measurement (there is no airflow), resistance of the circuit and airways do not play a role. Pressure equalizes along the ventilator circuit, so that the pressure measured at the proximal airway equals the *mean* pressure in the terminal respiratory bronchioles and alveoli (the mean alveolar pressure). Therefore, P_{plat} is a logical surrogate measurement for alveolar pressure. The primary limitation of this measurement lies in the fact 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 in heavily diseased lung regions, at the same P_{plat} . P_{plat} in a healthy adult with normal lung compliance undergoing mechanical ventilation is low, usually in the range of 5-15 cm H2O. Patients with alveolar disease (pneumonia, cardiogenic pulmonary edema, acute lung injury (ALI), and acute respiratory distress syndrome (ARDS) have noncompliant lungs, therefore P_{plat} is typically much higher. Measures to maintain P_{plat} below 30 cm H_2O, the currently recommended limit, are discussed below.

Intrinsic Positive End Expiratory Pressure

Positive end expiratory pressure (PEEP) indicates that the measured airway pressure 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 ($PEEP_E$). In contrast, intrinsic PEEP ($PEEP_I$, auto-PEEP) arises when exhalation is incomplete, either due to intrathoracic airway obstruction, early airway closure during exhalation, or inadequate exhalation time (Figures

8,9). The common denominator among these mechanisms is trapping of air in the lung at the end of exhalation, ultimately leading to increased intrathoracic pressure.



Respiratory Cycle

Airflow In

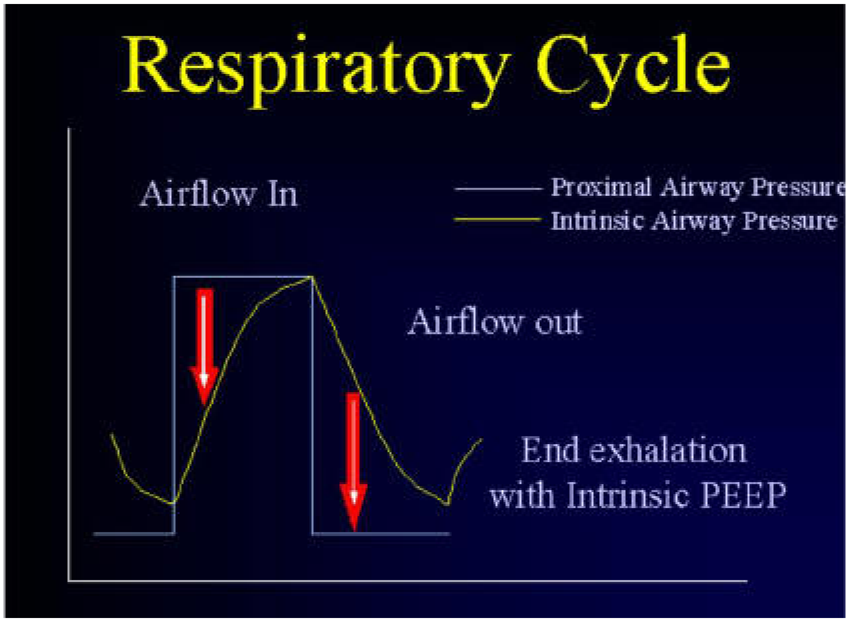
Proximal Airway Pressure

Intrinsic Airway Pressure

Airflow out

End exhalation
No Intrinsic PEEP

Figure 8



Respiratory Cycle

Airflow In

Proximal Airway Pressure

Intrinsic Airway Pressure

Airflow out

End exhalation
with Intrinsic PEEP

Figure 9

$PEEP_I$ can cause problems by several mechanisms. First, because exhalation is incomplete, air is progressively trapped in the lungs leading to early airway closure and dynamic hyperinflation with associated risk of VILI. Second, $PEEP_I$ leads to difficulty triggering the ventilator and increased WOB. Third, $PEEP_I$ can cause patient ventilator dysynchrony if the patient is actively contracting the respiratory muscles at end exhalation as the ventilator triggers. Lung inflation may begin while the patient is attempting to complete exhalation. Finally, increased intrathoracic pressure can impede venous return to the heart, leading to hemodynamic instability. Simultaneously, impaired venous return may compromise pulmonary blood flow, increase physiologic dead space, and lead to worsening hypercapnea. Control of $PEEP_I$ is discussed below.

Lung Protective Ventilator Strategies

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

1. high airway pressure during lung inflation
2. high $PEEP_I$ due to obstructive airways disease
3. patient/ventilator dysynchrony
4. equipment failure

Controlling Airway Pressure

ARDS, ALI, and Pulmonary Edema

Elevated plateau pressures are encountered in patients with poor lung compliance due to parenchymal lung disease (e.g. pulmonary edema, either cardiogenic or non cardiogenic) or obstructive airways disease with air trapping. Clearly, treatment of reversible causes of the underlying problem is the cornerstone of therapy. Mechanical ventilation is purely a supportive measure, not a therapeutic intervention. The goal is to support the respiratory system while avoiding iatrogenic injury. The effect of lowering P_{plat} has been studied in patients with ARDS in a prospective, randomized fashion, but evidence in other disease states is limited to reports of case series and retrospective chart reviews.

Initial studies compared conventional MVS strategy (Vt 10-15ml/kg, with a goal of obtaining normal PaO_2 and $PaCO_2$) with a lung protective MVS strategy (Vt 6-8 ml/kg with correction of hypoxia but allowing hypercapnea in favor of avoiding high airway pressures). The results were conflicting (9-13). The most recently and largest reported trial, the ARDS Network Trial (9), prospectively compared a conventional MVS strategy (12 ml/kg Vt and a P_{plat} limit of 50 cm H_2O) with a protective MVS strategy (6 ml/kg Vt and P_{plat} limit of 30 cm H_2O). After enrollment of 861 patients with ARDS and an interim analysis, the trial was stopped early due to a 22% reduction in mortality, 20% fewer days requiring mechanical ventilation, and fewer organ system failures in the group receiving the low Vt strategy.

MVS strategy in patients with other disease processes has not been as extensively studied. The extrapolation of these findings to patients with CHF, ALI, pneumonia, pulmonary fibrosis, lung cancer, and other lung pathology is not based on experimental evidence.

In summary, based on the available literature, emergency physicians should employ a lung protective strategy using low Vt, limiting P_{plat} to 30 H_2O, and permissive hypercapnea in a patient with ARDS or pulmonary edema, in order to avoid iatrogenic lung injury.

Obstructive Airways Disease

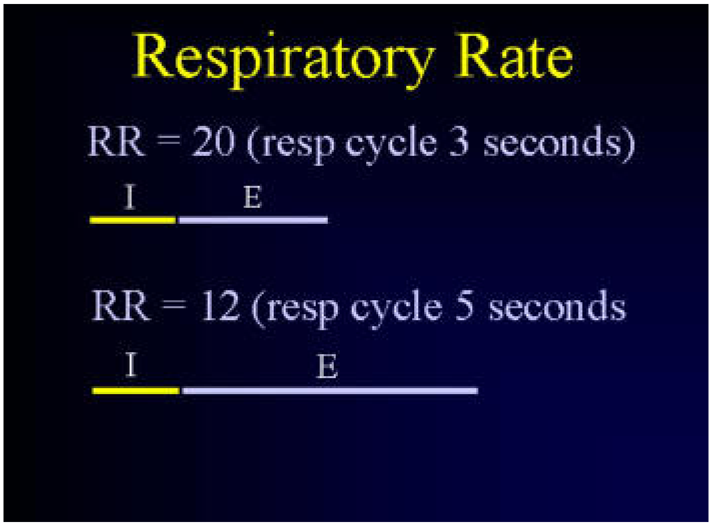
Obstructive airways disease (OAD) exacerbation requiring mechanical ventilation is often associated with air trapping and dynamic hyperinflation of the lungs. High P_{peak} arises as a result of inspiratory airflow limitation, a phenomenon more common in the support of patients with severe asthma than those with COPD. High P_{plat} arises as a result of lung over-distention and resultant diminished compliance. Those patients with both high P_{peak} and P_{plat} comprise a group of high-risk patients with both obstruction and over distention. They are at risk for complications including pneumothorax, tension pneumothorax, pneumomediastinum, dysrhythmias, and hemodynamic collapse. There have been no prospective trials comparing MVS strategies in such patients. It is common practice to employ a strategy of permissive hypercapnea/controlled hypoventilation to both eliminate $PEEP_I$ and avoid high $P_{plat.}$ This strategy employs use of a low Vt, low RR, and high IFR to shorten inspiratory time and prolong expiratory time. Although this strategy often leads to hypercapnea, it is considered safer to allow the patient to develop a respiratory acidosis than to ventilate at excessive airway pressures. A lower limit of acceptable pH has not been established, but general recommendations have been to allow pH as low as 7.0 – 7.2.

Controlled hypoventilation is required more often in the management of status asthmaticus than in COPD. Evidence in support of controlled hypoventilation comes from retrospective studies. In a report of 26 patients requiring 34 episodes of mechanical ventilation for status asthmaticus, a strategy using an initial RR of 6-10 cycles/min, Vt of 8-12 ml/kg, and FiO_2 adjusted to obtain normal PaO_2, P_{peak} was maintained below 50 cm H_2O by subsequent reduction of Vt and decreasing IFR. All patients required sedation, and 9 required pharmacologic paralysis to control respiratory rate. All patients survived. Complications included transient hypotension (57%), pneumonia (27%), SVT (15%), and pneumomediastinum or subcutaneous emphysema (11%) (10). This report led to widespread acceptance of the concept of permissive hypercapnea and controlled hypoventilation in the management of acute asthma exacerbation (11). Subsequent reports suggest that P_{peak} and P_{plat} are not adequate indicators of pulmonary hyperinflation (12), and recommend that expiratory volumes be measured in these patients (13). This technique has not gained widespread acceptance.

In sharp contrast, a retrospective study described a series of 18 asthmatic patients ventilated using a "conventional approach" with rapid correction of PaO_2 and $PaCO_2$ regardless of the airway pressures required. That study demonstrated no mortality attributable to MVS. Complications included transient hypotension (35%) and pneumomediastinum / subcutaneous emphysema (5%). These complication rates are comparable to those reported with use of controlled hypercapnea.

Controlling Intrinsic PEEP

Elevation of $PEEP_I$ indicates that the patient has obstruction to expiratory airflow, most commonly from COPD or asthma, but it may develop in patients with ARDS, ALI, cardiogenic pulmonary edema, and pneumonia. While virtually all patients with elevated P_{plat} due to OAD have high $PEEP_I$, not all patients with $PEEP_I$ have high $P_{plat.}$ In other words, patients with lung over distention represent a subset of patients with $PEEP_I$. Maneuvers directed at elimination of $PEEP_I$ have in common the effect of decreasing inspiratory time therefore providing more expiratory time. Decreasing RR, Vt and increasing IFR effectively accomplish this goal (Figures 10-12). Often, this cannot be achieved without sedation and pharmacologic paralysis.

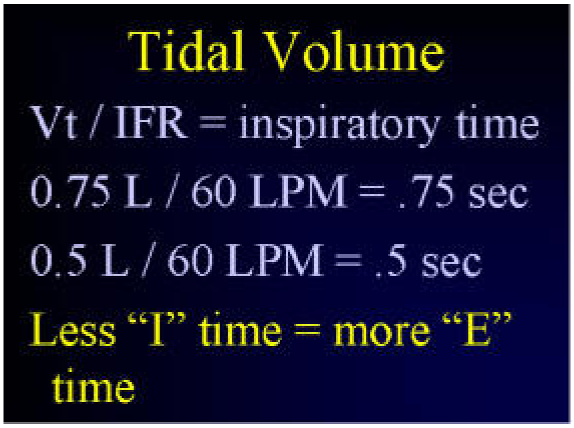


Respiratory Rate

RR = 20 (resp cycle 3 seconds)

RR = 12 (resp cycle 5 seconds

Figure 10



Tidal Volume

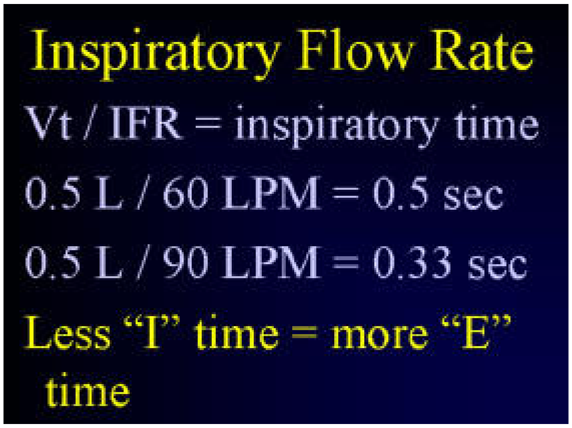
Vt / IFR = inspiratory time

0.75 L / 60 LPM = .75 sec

0.5 L / 60 LPM = .5 sec

Less "I" time = more "E" time

Figure 11



Inspiratory Flow Rate

Vt / IFR = inspiratory time

0.5 L / 60 LPM = 0.5 sec

0.5 L / 90 LPM = 0.33 sec

Less "I" time = more "E" time

Figure 12

Enhancing Patient Ventilator Synchrony

Some of the most commonly unrecognized problems that arise in the support of the critically ill patient fall into the category of patient/ventilator dysynchrony. These situations can markedly increase the WOB, leading to increased CO2 and lactic acid production, with both respiratory and metabolic acidosis.

Difficulty triggering the ventilator

In order to trigger a ventilator, a patient must cause either a drop in pressure or airflow in the proximal airway, depending upon 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 H_2O below the level of $PEEP_E$. Difficulty in triggering the ventilator is often difficult 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 v. time curve on the ventilator display, if available. A large negative deflection at the beginning of inhalation suggests that the ventilator sensitivity needs to be increased.

More commonly, high $PEEP_I$ is the cause. The patient must first lower the intrathoracic pressure enough to overcome $PEEP_I$ before the airway pressure can drop to the threshold sensitivity. The solution to this problem is to raise $PEEP_E$ to a level ½ to ¾ of $PEEP_I$, allowing the patient to perform less work to trigger inhalation. This process mandates frequent reassessment of $PEEP_I$ and manipulation of the ventilator during this dynamic period.

Autocycling

Autocycling refers to a phenomenon when the ventilator set in AC mode begins to rapidly trigger without the patient initiating respiration. The cause is usually small vacillations in airway pressure that the ventilator "interprets" as patient efforts. Tremors, shivering, voluntary motion, convulsions, and crying all are examples of potential causes. Autocycling should prompt immediate disconnection from the ventilator circuit and ventilation by bag-valve device until the problem is resolved.

Rapid breathing

When attempting to ventilate a patient with OAD, the goal is to eliminate $PEEP_I$. Permissive hypercapnea is best achieved at low respiratory rates, but at the same time hypercapnea leads to increased respiratory drive. This can be typically quelled using a combination of sedation with benzodiazepenes and analgesia using opiates. Neuromuscular blockade should be considered a last resort undertaken only after careful consideration of the risk of prolonged paralysis and potential development of neuropathy critical illness.

Overbreathing the ventilator

Patients who are receiving low Vt ventilation for ARDS or for OAD develop hypercapnea and increased respiratory drive. Overbreathing the ventilator refers to the patient's effort to draw a higher Vt than is set. This can be detected by observing the exhaled Vt, or by finding a negative deflection at the end of inhalation on the pressure v. time plot (Figure 13). As with controlling rapid breathing, the solution is sedation and analgesia.

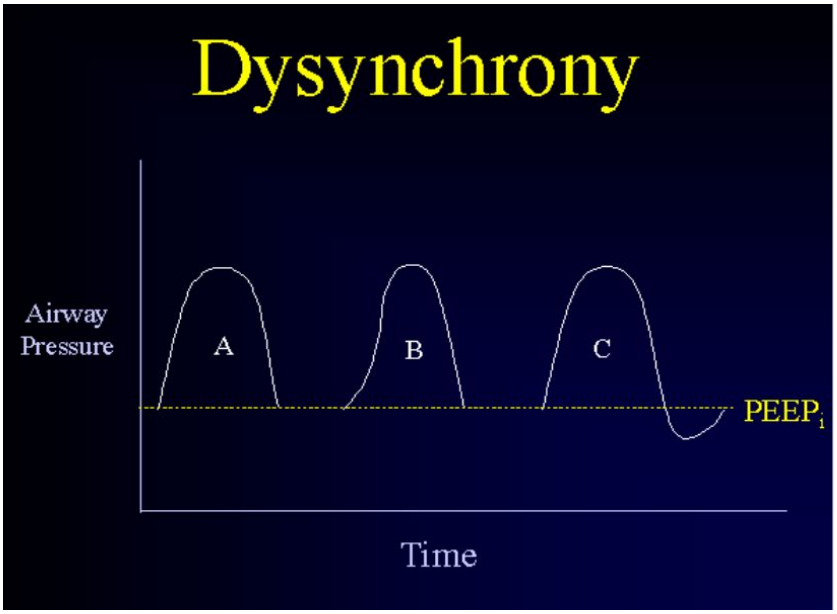


Figure 12

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. When it is obvious by examination that the patient is actively inhaling, the problem may be severe. On the pressure v. time plot, the rise in pressure during inhalation will be concave, rather that convex. (Figure 18). Potential solutions are to raise the IFR, to switch modes to pressure support ventilation, or to use sedation and analgesia.

Coughing

Coughing is a common problem, arising from increased secretions, airway foreign body (ET tube), or the underlying disease that led to the need for MVS. Coughing can lead to autocycling, poor patient comfort, ET tube dislodgement, and rarely airway injury. Placement of the ET tube above the carina should be confirmed. Suctioning and provision of warmed, humidified air is often helpful. If these simple measures fail to provide relief, then 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 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% O_2. The mnemonic made popular by the American Heart Association Pediatric Advanced Life Support Course is useful to recall the causes of unexpected decompensation: DOPE (*D*islodgement of the ET tube, *O*bstruction of the tube, *P*neumothorax, and *E*quipment failure). Confirmation of endotracheal tube placement, suctioning via endotracheal catheter, auscultation, chest radiography, and equipment troubleshooting are necessary actions.

Summary

Respiratory distress and failure are common problems in the emergency department. The emergency physician should be skilled not only in management of the airway, but also in the initiation of mechanical ventilatory support. The frequency of complications associated with invasive ventilation can be reduced through implementation of lung protective ventilator strategy. Patient comfort and safety are improved by attention to and correction of patient ventilator dysynchrony.

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