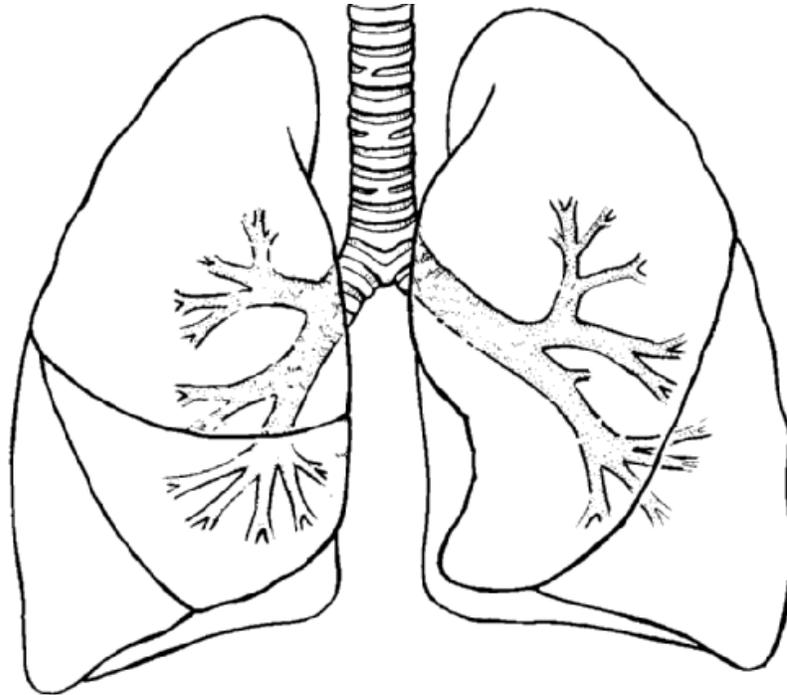


Adult Invasive Mechanical Ventilation



Self-Learning Packet

2004



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Purpose

The purpose of this self-learning packet is to educate critical care registered nurses on the principles and practice of invasive mechanical ventilation for adult patients.

Objectives

After completing this packet, the learner should be able to:

1. Define hypoxemic and ventilatory respiratory failure
2. Discuss types of invasive airways, their management and complications
3. List the indications for and complications of mechanical ventilation
4. List and discuss the parameters monitored during mechanical ventilation
5. Describe three major modes of ventilation
6. Discuss basic principles of mechanical ventilation related to disease management
7. Differentiate between airway and intrathoracic complications
8. Interpret basic flow, volume and pressure graphics obtained from the mechanical ventilator
9. Identify and discuss essential adjunctive therapies for the patient receiving mechanical ventilation
10. Discuss issues related to weaning from mechanical ventilation
11. Discuss nursing management for the patient on mechanical ventilation
12. Identify trouble-shooting techniques

Instructions

In order to receive 4 contact hours, you must:

- complete the posttest at the end of this packet
- submit the posttest to Education & Development with your payment
- achieve an 84% or greater on the posttest

Be sure to complete all the information at the top of the answer sheet. You will be notified if you do not pass, and you will be asked to retake the posttest.

Introduction to Mechanical Ventilation

Mechanical ventilation is a supportive therapy used to assist patients who are unable to maintain adequate oxygenation or carbon dioxide elimination. These patients usually exhibit signs of acute respiratory failure and are not candidates for less invasive methods of respiratory support. There are invasive and non-invasive techniques of mechanical ventilation. This packet focuses on the invasive techniques commonly used for adult patients.

Acute Respiratory Failure

Acute respiratory failure is defined as an acute onset of severely impaired gas exchange. The onset may occur over several hours or several days. Diagnosis is based upon clinical presentation and arterial blood gas values. Patients with acute respiratory failure typically present with dyspnea or increased work of breathing. Other common symptoms include increased pulmonary secretions, neurologic changes, and tachycardia. Regardless of the cause, patients with acute respiratory failure often require mechanical ventilation until the underlying pathology can be resolved. Specific manifestations will depend on the cause and the patient's underlying disease process. There are two types of acute respiratory failure: hypoxemic and hypercapnic (ventilatory). Patients may present with one or a mixture of both types.

Hypoxemic Respiratory Failure (Hypoxemia)

Hypoxemia is defined as an acute reduction in PaO_2 of 10% or more over a period ranging from several minutes to several hours.

Emergent treatment should be initiated when the PaO_2 is less than 60mm Hg indicating a rapid decline in the oxygen content of blood and subsequent tissue hypoxia. If there is not a shift of the oxyhemoglobin dissociation curve, a PaO_2 of 60mm Hg is roughly equivalent to a pulse oximetry reading of SpO_2 of 88%.

Because of the potential for inaccurate pulse oximetry measurements, SpO_2 readings have limitations and alone are insufficient to diagnose hypoxemia. Contributing factors that may cause inaccurate oximetry readings are: poor perfusion, dark skin pigmentation, motion artifact, bright lights, dyes (methylene blue), abnormal hemoglobin, nail polish/acrylic nails and hypothermia. Therefore, the SpO_2 reading should be correlated to the SaO_2 from the arterial blood gas and then used as a tool to trend the patient's progress.

SIGNS AND SYMPTOMS OF HYPOXEMIA

- Asymptomatic
- Headache
- Cognitive deficits
- Tachypnea
- Tachy or bradydysrhythmias
- Hypotension
- Central cyanosis – *late sign*

Diagnosis

Diagnosis of hypoxemic respiratory failure is made on the basis of the PaO_2 . The other components of the blood gas are not used to make this diagnosis. Treatment is focused on immediate stabilization and supportive care. An assessment of the appropriate oxygen delivery system, cardiac rhythm and hemodynamics are rapidly performed and correlated to the physical assessment. If supplemental oxygen and appropriate drug therapy is insufficient to rapidly resolve the patient's hypoxemia and respiratory distress, mechanical ventilation may be required.

Etiology

The causes and contributing factors of hypoxemia are listed in the following table.

Causes	Contributing Factors
Low FiO ₂	<ul style="list-style-type: none"> • Supplemental oxygen device removed or disconnected • Lack of oxygen in ambient air (high altitude)
Hypoventilation	<ul style="list-style-type: none"> • Low respiratory rate (<12 breaths per minute) • Medications (sedative, opioid, paralytic) • Adverse neurological event
Ventilation/Perfusion (V/Q) mismatch Deadspace Pulmonary shunt	<ul style="list-style-type: none"> • Narrowed airway due to bronchospasm, bronchial secretions, or edema • Impaired pulmonary circulation due to pulmonary embolus • Lung parenchymal injury – e.g. lung infections, near drowning, chemical or smoke inhalation (carbon monoxide poisoning), and liquid aspiration • Alveoli are filled with edema, blood or exudate • Severe atelectasis • Pneumothorax • Cardiac septal defects
Low mixed venous oxygen saturation (SVO ₂)	<ul style="list-style-type: none"> • Decrease in cardiac output (CO) superimposed on diseased lungs
Procedures/Nursing Interventions	<ul style="list-style-type: none"> • Patient positioning • Chest physiotherapy • Tracheal suction • Dialysis • Thoracentesis • Moderate to deep sedation • Bronchoscopy/Endoscopy

Treatment

Treatment for hypoxemia is based on providing adequate oxygenation to prevent life-threatening complications. Diagnostic procedures to evaluate treatment are performed as needed in addition to continuous monitoring of pulse oximetry and capnography. Diagnostic procedures may include chest x-ray, ABG measurement, bronchoscopy, VQ scanning and CT scan.

CLINICAL APPLICATION

MECHANICAL VENTILATION AND PULMONARY EDEMA

Pulmonary edema results when the alveoli fill with fluid due to fluid volume excess, poor cardiac function, or increased capillary permeability. The fluid-filled alveoli create an increase in pulmonary shunting and hypoxemia. When clinical measurements of preload are elevated as a result of fluid excess and/or poor cardiac function, pulmonary edema will resolve if preload is reduced. Patients with this type of pathology may respond quickly to drug therapy and the need for mechanical ventilation can sometimes be avoided.

Pulmonary edema resulting from an increase in capillary permeability is characteristic of Adult Respiratory Distress Syndrome (ARDS) and will persist despite low or normal measures of preload. Patients with ARDS commonly require mechanical ventilation for prolonged periods of time until the complex pathologies of this syndrome can be corrected.

Ventilatory Respiratory Failure (Hypercapnia)

Ventilatory respiratory failure (hypercapnia) is diagnosed by a $\text{PaCO}_2 > 50\text{mm Hg}$. The other components of the blood gas are not used to make this diagnosis. For patients with chronic hypercapnia, an increase in PaCO_2 above baseline is used for diagnosis.

SIGNS AND SYMPTOMS OF HYPERCAPNIA

- Headache
- Dizziness
- Change in level of consciousness
- Asterixis (abnormal muscle twitching)
- Miosis (abnormal contraction of the pupils, papilledema)
- Hypertension

Etiology

Causes of ventilatory respiratory failure are:

- Decreased Respiratory Drive

Causes of decreased respiratory drive leading to hypercapnia include: medications (opioids, alcohol, tricyclic antidepressants, barbiturates, propofol or other sedative drugs), hypothyroidism, metabolic alkalosis, structural lesion(s) in the central nervous system, infections of the central nervous system, idiopathic and alveolar hypoventilation.

- Inability to generate effective respiratory muscle contraction

Causes of an inability to generate effective respiratory muscle contraction include: medications (neuromuscular blockade drugs), tetanus, myasthenia gravis, Guillain-Barre syndrome, amyotrophic lateral sclerosis, poliomyelitis, cervical cord injury (C-4 or higher), muscular dystrophy, electrolyte abnormalities (hyper/hypokalemia, hypophosphatemia, hypermagnesemia) and diaphragmatic fatigue.

- Obstructive and restrictive pulmonary disorders

Examples include: chronic obstructive lung disease (COPD), acute respiratory distress syndrome (ARDS), near drowning, severe pneumonia, liquid aspiration, pulmonary embolism, pulmonary edema, cancer, acute airway obstruction (acute epiglottitis, secretions), status asthmaticus, smoke inhalation/chemical inhalation, laryngospasm, emphysema, severe kyphoscoliosis and morbid obesity.

Treatment

Hypercapnia is treated by increasing the amount of air moving in and out of the alveoli; thus, increasing ventilation. Immediate stabilization is achieved by ensuring an open airway and augmenting spontaneous breathing with manual ventilations. After the patient has been stabilized, treatment specific to the underlying cause of hypercapnia will be provided. If hypoventilation cannot be rapidly corrected, mechanical ventilation may be required.

CLINICAL APPLICATION

MECHANICAL VENTILATION AND RESTRICTIVE AIRWAY DISEASE

Severe exacerbations of restrictive airway disease, like that seen in status asthmaticus, results in hypercapnia. Treatment management includes supplemental oxygen, inhaled bronchodilators, intravenous glucocorticosteroids, and antibiotics (if infection is suspected). Although supplemental oxygen is part of the treatment plan, it will not be effective without the other components. The problem in this disease state is that not enough air is moving in and out of the lungs. Oxygen alone cannot correct the problem. Bronchodilators and corticosteroids function to open the airways, allowing improved ventilation. If these interventions are not successful, mechanical ventilation may be required.

Indications for Mechanical Ventilation

Invasive mechanical ventilation is hazardous, uncomfortable, and expensive and should only be utilized when indicated. Major indications for mechanical ventilation are:

- The partial pressure of oxygen in arterial blood (PaO_2) cannot be maintained above 50mm Hg despite high levels of delivered oxygen.

Clinical example: Acute Respiratory Distress Syndrome (ARDS).

- The partial pressure of carbon dioxide in arterial blood rises above 50 torr.

Clinical example: Acute Respiratory Failure (ARF) related to opioid overdose.

- Ventilation becomes inefficient and/or exhausted.
Clinical example: bronchospasm, flail chest and impending respiratory failure.
- Airway protection
Clinical example: tracheal injury, edema, severe head injury and facial fractures.

Some examples of exceptions to these indications are:

- A patient with chronic obstructive pulmonary disease (COPD) may be clinically stable with abnormal arterial blood gas (ABG) values and will not require mechanical ventilation unless CO₂ levels rise above their normal values.
- Patients with neuromuscular disease may be placed on mechanical ventilation in order to prevent respiratory distress or arrest due to their decreased strength to ventilate.

In the past, the decision to begin mechanical ventilation was based on non-specific subjective bedside observations. The primary indications were apnea, acute ventilatory failure, impending acute ventilatory failure, and severe oxygenation deficit. However today, indications are objective and based on measurements of the patient’s tidal volume, vital capacity, forced expiratory volume in the first second, functional residual capacity, respiratory rate, maximum inspiratory pressure, minute ventilation, maximum voluntary ventilation, dead space fraction, arterial PaCO₂ (mm Hg), and arterial PaO₂ (mm Hg). Indicators for mechanical ventilation are presented in the following table.

Indicators for Mechanical Ventilation

Parameter	Normal	Indications for Mechanical Ventilation
Tidal Volume (ml/kg)*	5 – 8	< 5
Vital capacity (ml/kg)*	65 – 75	< 10 - 15
Forced Expiratory Volume in 1 sec (FEV1) (ml/kg)	50 – 60	< 10
Functional residual capacity (FRC) (% of predicted value)	>80	<50
Respiratory rate (breaths/min)*	12 – 20	> 35 or < 6
Maximum inspiratory pressure (cm H ₂ O)*	80 – 120	< 20 - 30
Minute Ventilation (L/min)*	5 – 8	> 10
Maximum Voluntary Ventilation (VE) (L/min)	120 – 180	< 20; < 2 X VE
Dead space fraction (%)	0.25 – 0.40	>0.60
Arterial PaCO ₂ (mm Hg)	35 – 45	> 50
Arterial PaO ₂ (mm Hg)	75 – 100 (on room air)	< 50 (on room air)
* Denotes measurements that can be completed at the bedside		

Airway Management

Maintaining a secure airway is always the number one priority in the care of any patient. Without a patent airway, nothing can prevent death. Emergency airway management is taught in life support courses. This packet will review techniques and equipment used to provide airway access for invasive mechanical ventilation.

An artificial airway is required for management of a patient receiving invasive mechanical ventilation. Tubes used for invasive management include the standard endotracheal tube, the laryngeal mask airway (LMA) and the tracheostomy tube. All of these tubes have the potential to cause injury to the pharynx during insertion, maintenance and removal. They also increase the risk of respiratory tract infection. Tubes used for invasive mechanical ventilation prevent the patient's ability to speak. Alternate modes of communication must be available to the patient. Patients with artificial airways require frequent sedation and pain medication to cope with the discomfort and anxiety produced from the presence of a foreign object in their airway.

Artificial Airways

Endotracheal Intubation

The most common tube used to provide an airway intubation for mechanical ventilation is the endotracheal tube. It is inserted via the nasotracheal or orotracheal cavity. Orotracheal intubation is the most common route. The endotracheal tube provides a secure airway when the balloon is inflated and seals. Insertion is moderately complex, but less invasive than a tracheostomy. Compared to the LMA, the endotracheal tube provides greater protection from aspiration and air leaks.

Despite its many advantages, endotracheal intubation poses significant risk to the patient. Trauma to the nares, lips, teeth, tongue, pharynx, and trachea may occur. The tube may enter the esophagus or extend into only one of the two main stem bronchi; either situation can lead to hypoxemia and hypoventilation. As the tube passes through the pharynx, the gag reflex is triggered and may cause vomiting and potential aspiration of gastric contents. Prolonged intubation efforts may result in severe hypoxemia. Because of these risks, only practitioners who have demonstrated competency are allowed to perform intubation. The bedside nurse may be called upon to assist with intubation and to monitor the patient before, during and after the procedure. In order to carry out these responsibilities, the clinician must be knowledgeable of the intubation process and its potential complications.

Orotracheal intubation is contraindicated if oral surgery is planned, extensive oral trauma is present or if the mouth cannot be opened far enough to achieve insertion of the tube. Most relative contraindications to orotracheal intubation can be overcome through the use of medications and mechanical devices designed to limit tube movement and prevent inadvertent tube removal.

Nasotracheal intubation is useful if the mouth cannot be accessed due to trauma or other factors. Placing the tube through the nares is more comfortable, allows better communication, makes oral care easier, and reduces shifting of the tube. Nasotracheal tubes are not used for periods longer than 24 hours due to the risk of sinus infection.

Contraindications of Orotracheal and Nasotracheal Intubation

Orotracheal	Nasotracheal
<ul style="list-style-type: none"> • Potential for oral surgery • Extensive oral trauma is present • When the mouth cannot be opened far enough to achieve insertion of the tube 	<ul style="list-style-type: none"> • Basilar skull fractures • Nasal fracture • Nasal polyp • Epistaxis • Coagulopathy • Planned thrombolysis

Laryngeal Mask Airway (LMA)

The LMA is used by anesthesia as a short-term means to maintain the airway during surgery or for the patient who has a challenging airway until an endotracheal tube or tracheostomy may be placed. It is a tube with a cuffed mask-like projection at the distal end. The LMA is inserted in the pharynx and advanced blindly, thus, a less difficult procedure than endotracheal intubation. The ease of insertion may be a significant advantage for patients with cervical spine instability or whose mouths are difficult to open fully. The LMA has been shown to be equal to the endotracheal tube in quality of ventilation, but inferior in protection of the airway. The most common complications associated with LMA usage include regurgitation, vomiting, aspiration, laryngospasm and bronchospasm.

Tracheostomy

The tracheostomy is a surgically created airway used for patients who need a long-term artificial airway. A tracheostomy is not used for emergency airway management. It is considered for a patient who has been orally intubated for a period of 7 – 10 days and still requires mechanical ventilation or is otherwise unable to maintain a patent airway. A tracheostomy reduces airway resistance, eases communication, facilitates effective oral hygiene and is significantly more comfortable for the patient.

The procedure involves a surgical opening made through the cartilage rings of the trachea. A cuffed tracheostomy tube is inserted into the opening. The cuff is similar to that on the end of an endotracheal tube. Common adult tracheostomy tube sizes range from 6 – 8 mm initially. The tracheostomy tube will be gradually sized down to 4 mm before removal. Most tracheostomy tubes have an outer and an inner cannula. The outer cannula remains in place, secured by sutures and/or tracheostomy ties or a manufactured tube holder. The inner cannula can be removed and cleaned or replaced. An obturator is supplied with the tracheostomy tube and should be kept at the patient's bedside at all times.

Complications of tracheostomy include infection, tracheal trauma, inadvertent removal and bleeding. The tracheostomy itself lies near large blood vessels and over time it can erode into these vessels causing severe hemorrhage. Although this is a rare complication it is life threatening. The patient must be monitored for signs of increased bleeding and the physician notified promptly if large amounts of bleeding occur. If the outer cannula is inadvertently removed after a tract has formed it can be replaced by inserting the obturator into the outer cannula and then inserting the entire apparatus back into the tract. After the outer cannula is in place, remove the obturator and re-secure the outer cannula.

Orotracheal Intubation Procedure

Even when elective, intubations must be carried out swiftly to avoid complications. The physician must obtain informed consent for elective intubations. The process of intubation is frightening for the patient and their loved ones. Explain the process simply and calmly to help them cope.

The qualified practitioner performing the intubation must be made aware of pertinent clinical findings such as:

- The patient's primary diagnosis and reason for intubation.
- Suspected or confirmed cervical spine injury. The technique used to open the airway will depend on whether or not the cervical spine is stable. If adequate cervical stabilization is not maintained, secondary spinal injury can occur.
- When the patient last ate or drank, and the presence of gastric distention. Patients with gastric distention or those who recently ate are at increased risk for aspiration.
- Level of consciousness, anxiety and degree of respiratory difficulty. This will determine the need for sedation or paralytic agents and the patient's ability to lie flat.
- Presence of loose or false teeth or dental work. All removable dental appliances must be taken out prior to oro-tracheal intubation, they may stay in place for nasotracheal intubation.

Rapid Sequence Intubation

If the intubation is elective and the patient is alert, the physician may choose to perform rapid sequence intubation. Rapid sequence intubation is a procedure designed to allow rapid, safe intubation of a conscious patient who may have food in the stomach. To perform rapid sequence intubation the physician will require specific drugs. Typically it includes a benzodiazepine for sedation, a narcotic for analgesia and a short-acting neuromuscular blocking drug to ease passage of the tube while minimizing gagging or vomiting. To assist with rapid sequence intubation, knowledge of the medications used, their mechanism of action, and the appropriate monitoring and assessment parameters for their use is necessary.

Supplies & Equipment

When preparing for intubation, all necessary equipment must be in the patient's room and emergency resuscitation equipment nearby. Essential equipment includes:

- Personal protective equipment
- Endotracheal tube – the size will be specified by the person performing the intubation (adult female 7.5 – 8.0 mm tube; adult male 8.0 – 9.0 mm tube)
- Laryngoscope, blades (curved & straight), extra batteries and bulb
- Flexible stylet
- Bag-valve-mask device attached to 100% oxygen
- Oxygen source, tubing and connectors
- Swivel adaptor
- 10 cc syringe for cuff inflation
- Water soluble lubricant
- Rigid pharyngeal suction-tip (Yankauer) catheter, suction tubing and suction source
- Suction catheters
- Bite block or oropharyngeal airway
- Endotracheal tube securing apparatus or appropriate tape

- Stethoscope
- Medications as ordered
- Mechanical ventilator

Pre-Procedure Preparations

IV access will be required for sedation and/or rapid sequence medications and may also be needed if the patient has an adverse reaction to intubation. Adverse reactions include nausea, anxiety and respiratory distress. Serious life threatening complications include cardiac arrest, respiratory arrest, hypotension, and anaphylaxis. To position the patient, pull the head of the bed away from wall and move the patient to the top of the bed. Position the non-trauma patient supine with the head extended and the neck flexed. This position allows visualization of the larynx and vocal cords, easing the task of intubation. If cervical instability is present or suspected, in-line cervical stabilization must be provided throughout the process of intubation. Verify oxygen and suction equipment are functioning and that the laryngoscope lights up when assembled. Prepare any ordered medications. Personnel typically present for elective intubation include the physician, respiratory therapist, and one or more registered nurses. Once all needed equipment and personnel are in the room, the procedure may begin.

Insertion of the Endotracheal Tube

Pre-oxygenate the patient using the bag-valve mask and 100% oxygen for 3 to 5 minutes. Pre-oxygenation lessens, but does not eliminate the chance that the patient will become hypoxemic during the intubation procedure. The practitioner performing the intubation will open the airway, visualize the vocal cords using the laryngoscope (for orotracheal intubation) and insert the endotracheal tube. Orotracheal suctioning may be needed as the airway is opened.

During intubation, the application of cricoid pressure may be needed. Cricoid pressure is used to reduce the chances of regurgitation and aspiration. To apply cricoid pressure, locate the cricoid cartilage just caudal to the Adam's apple. Using the thumb and forefinger, apply gentle pressure in a downward and slightly cephalic direction. This maneuver compresses the esophagus between the trachea and the spine, preventing backward passage of gastric contents. If cricoid pressure is used, it must be maintained until the endotracheal tube is in place with the cuff inflated.

Procedural Monitoring

During intubation the patient must be closely monitored. Standard monitoring parameters include:

- ECG rate and rhythm: Hypoxemia may cause tachycardia and ectopic rhythms. A vagal response may also occur as the tube is passed through the pharynx, producing bradycardias.
- Respiratory rate: Patients requiring intubation often have abnormally high or low respiratory rates. Watch for signs of deterioration. If paralytic medications are used, the patient will become apneic.
- Blood pressure: Hypertension is often present in patients with respiratory distress. Sudden hypotension can occur in response to sedative and/or analgesic medications. Hypotension is also common in the period immediately after intubation as respiratory distress is resolved and the patient relaxes.

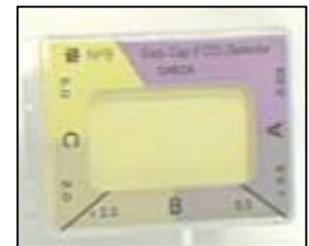
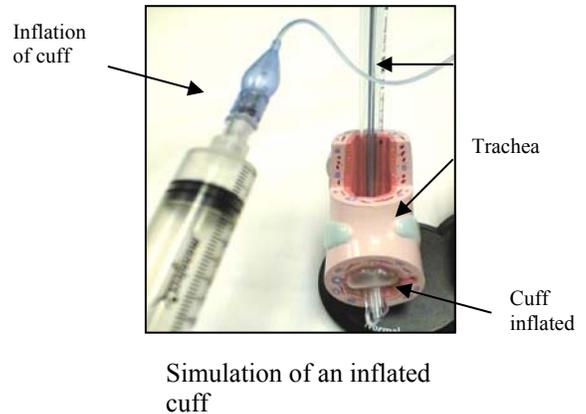
- SpO₂: The pulse oximeter is an important tool for early detection of hypoxemia. During intubation attempts the SpO₂ should not be allowed to drop below 90%. If this occurs, efforts should be suspended and the patient hyperoxygenated until the SpO₂ comes back up.

Confirmation of Tube Placement

Once the patient is intubated and the cuff inflated, confirm placement by listening first over the epigastrium, then the left upper and lower side of the chest, and the right upper and lower side of the chest. Observe for bilateral rise and fall of the chest wall. Proper placement is confirmed when air is not heard over the epigastrium and bilateral breath sounds are heard over the lung fields. Air heard over the epigastrium is a sign that the tube entered the esophagus rather than the trachea. The tube must be removed and preparation made for another attempt.

Breath sounds heard over only one side may indicate that the tube advanced down one of the main stem bronchi. The right main stem bronchus is shorter and straighter than the left, thus this happens more commonly on the right side. The tube must be repositioned. Prepare to deflate the cuff and assist the physician or respiratory therapist in pulling the tube back 1 – 2 cm. The cuff is then reinflated and placement verified again.

Secondary confirmation of placement is accomplished with the use of a CO₂ detector (different brand names). The CO₂ detector produces a color change in response to exhaled CO₂. A false negative may occur if the patient is in cardiopulmonary arrest and not producing CO₂. Otherwise, these devices are very accurate. If the CO₂ detector does not change color, the tube is probably in the wrong place. Prepare to remove the tube and reattempt intubation after the patient is hyperoxygenated again. A chest x-ray will be ordered as a tertiary confirmation of tube placement. The chest x-ray is helpful, but clinical indicators of proper placement are far more important and immediate. **If clinical indicators indicate the tube is not in proper position, do not wait for a chest x-ray to confirm; the situation must be corrected immediately.** Any delay in repositioning the tube can lead to prolonged hypoxemia and potential brain injury.



Securing the Airway

All tubes must be secured to decrease tube movement and prevent accidental extubation (removal of the tube) or self-extubation. If the patient has a head injury or possible intracranial hypertension, place the tape above the ears bilaterally (pictured) to prevent occlusion of the jugular vein. A daily chest x-ray is ordered to verify tube placement and visualize the lung fields. End-tidal CO₂ (capnography) and pulse oximetry are monitored continuously so any migration of the tube can be detected early.



Taping of ET Tube

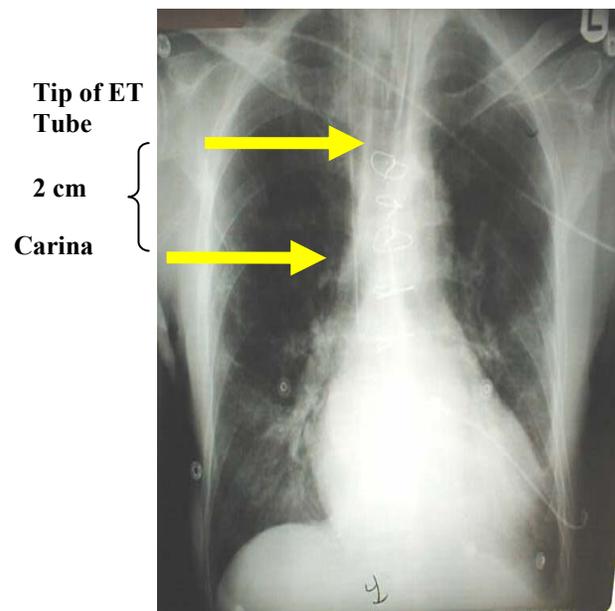
Documentation

Frequent vital signs and pulse oximeter readings are documented before, during and after intubation and as needed based on the patient's condition. Additional documentation includes medications administered, the person who performed the intubation, the type and size of tube used, the depth marking at the lips, and the method used to secure the tube. Document the methods used to confirm placement and whether tube position was adjusted or not. After tube placement is confirmed and the patient is stable, complete a reassessment of the patient and document.

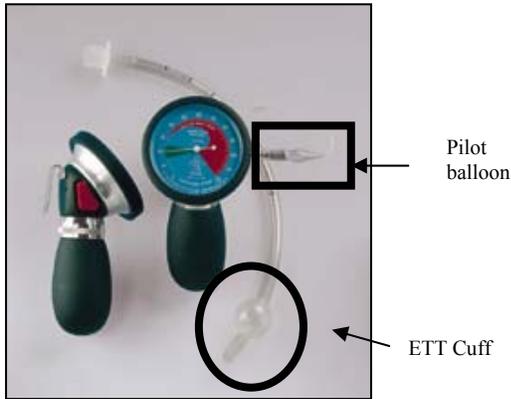
Endotracheal Tube Maintenance

Assessment of the endotracheal (ET) tube placement consists of measuring the depth of the tube and cuff pressure and the patient's secretion clearance. The depth is measured at the centimeter (cm) mark on the ET tube at the lip level and documented daily. Verify ET tube placement by evaluating daily chest x-rays and noting any changes in the respiratory assessment (breath sounds) or capnography measurements. When properly placed, the tip of the endotracheal tube is located about two centimeters above the carina as seen on the chest x-ray (pictured). When assessing secretion clearance, signs and symptoms of an adequate airway are:

- Cough and gag reflex
- Controlled secretions
- Absence of stridor, laryngospasm, or bronchospasm
- Absence of airway distress



Endotracheal Tube Placement



Measuring cuff pressure

The standard ET tube cuff (circled) is a high-volume, low-pressure cuff. This type of cuff allows a large surface area to come into contact with the tracheal wall, distributing the pressure over a larger area of the trachea to prevent tracheal injury and necrosis. Normal tracheal capillary perfusion pressure is 25 – 35 mm Hg. Cuff pressures should be lower than this to prevent tissue injury. The pilot balloon (small box) provides a rapid visual check of cuff inflation or deflation but offers no information on actual cuff pressures.

Cuff pressures are routinely measured using a cuff manometer like the Posey Cufflator™ (pictured here). The cuff prevents aspiration of oral secretions or gastric contents into the lungs. The goal is to maintain a seal without causing damage to the tracheal mucosa. There are two valid schools of thought as to how this goal is best achieved. One advocates maintaining cuff pressures of 20 – 25 mm Hg (27.2 – 34 cm H₂O) to minimize the risk of tracheal injury or aspiration. The other advocates setting the pressure to a minimum occlusion pressure or minimum leak pressure. The occlusion pressures may also be expressed as volumes, minimum occlusion volume (MOV) or minimum leak volume (MLV). If pressures are greater than 35 mm Hg (47.6 cm H₂O) or the cuff leak prevents the patient from maintaining set ventilator volumes, the physician must be notified and a clinical decision made to place a larger ET tube in a controlled setting. There are valid arguments for both schools of thought. An RN or respiratory therapist can monitor cuff pressures. Most cuff manometers measure pressure in centimeters of water (cm H₂O). To convert mm Hg to cm H₂O, multiply by 1.36. The Posey Cufflator™ will read 20 cm H₂O at a pressure of 14.7 mm Hg.



Posey Cufflator™
(cuff manometer)

Causes of High Cuff Pressures

- Endotracheal tube too small
- Cuff overfilled
- Incorrect positioning of endotracheal tube
- Tracheal dilation
- Low-volume, high-pressure cuff

Symptoms of Inadequate Seal

- Audible or auscultated inspiratory leak over larynx
- Patient able to audibly vocalize
- Pilot balloon deflated
- Loss of inspiratory and expiratory volume on mechanically ventilated patient

Occasionally the pilot balloon tubing is inadvertently cut. A “kit” can be used to temporarily correct this problem. The kit consists of a blunt needle that can be guided into the pilot tubing, and has a hub that can be manually closed after the cuff is inflated to the proper pressure. This is only a temporary fix, and close monitoring of pressures is important until the tube can be replaced.

Mechanical Ventilation

Goals of Mechanical Ventilation

The goal of mechanical ventilation is to improve ventilation, oxygenation, lung mechanics and patient comfort while preventing complications. Mechanical ventilators provide supportive therapy. The ventilator is not a cure for any disorder, rather it allows support of breathing while disease processes are treated. A ventilator is a machine that delivers and controls the flow of gas to a patient. Ventilators are classified in two ways: the method in which they deliver gas and the manner in which they end gas delivery. A ventilator can deliver gas under positive pressure with a bellows action or by negative pressure exerted on the chest wall.

A negative pressure ventilator, such as the iron lung (pictured) or chest cuirass, applies negative pressure to the thorax by a vacuum drawing air into the lungs. These ventilators do not require an invasive airway. Some of the earliest ventilators were of this type.

Positive pressure ventilators apply positive pressure to the airways and lungs through the use of an invasive airway. When adults are being mechanically ventilated via a tube, the respiratory system becomes a closed system. A closed system eliminates leakage of delivered gas, and protects the patient from aspiration of oropharyngeal secretions or gastric contents. The superiority in delivery of breaths and oxygen combined with safety features and improved access to the patient make positive pressure ventilators the primary type of ventilator found in today's critical care unit.



Iron Lung – Negative pressure ventilator



Bear 1000– Positive Pressure Ventilator

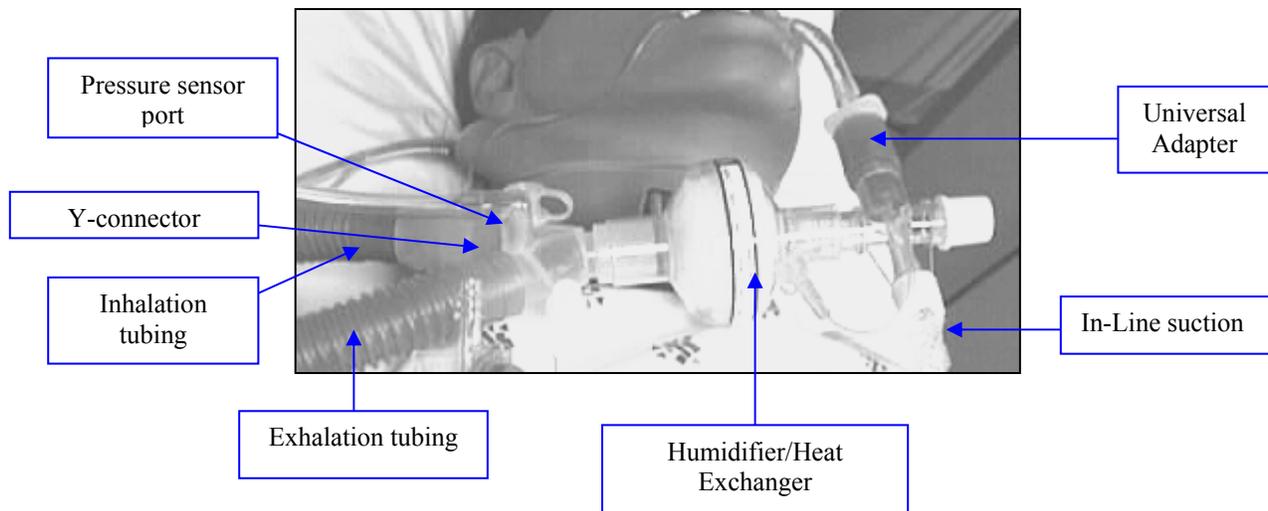
Physical Characteristics of Positive Pressure Ventilators

Positive pressure ventilators used in the critical care setting have several characteristics in common:

- A mechanical or pneumatic system to push gas under positive pressure.
- Electrical and gas connections.
- Exhalation valve that allows the patient to exhale through the closed system.
- User interface that allows the clinician to modify various parameters to individualize therapy.
- Software programming that monitors machine performance, detects alarm conditions, and produces graphic or digital data displays.
- Airway pressure sensor and display (gauge and/or digital readout). The airway pressure gauge monitors airway pressure during inhalation, exhalation and at rest. If the airway pressure exceeds or falls below preset limits an alarm will sound.

Ventilator Circuits

The ventilator circuit is a system of tubing that connects the patient's airway to the ventilator itself. Most ventilator circuits are disposable, but reusable circuits are still in use. Regardless of the type, all circuits include inhalation tubing, exhalation tubing, ports for pressure detectors, a Y-connector, an elbow, a universal adaptor, and some sort of humidification and heating system. The circuit may also include an adaptor for an in-line suction system or adaptors for delivery of aerosol medications.



Warming and Humidification

It is essential to provide warming and humidification of delivered gas during mechanical ventilation. The artificial airway bypasses the nose and oropharynx, where normal humidification and warming of inspired air takes place. If delivered gas is not humidified and heated, negative sequelae can occur. The physiologic effects of breathing cool dry gases are:

- Destruction of cilia which results in retention of secretions
- Drying of mucus glands which results in mucus plugging of airways
- Reduction in cellular cytoplasm which results in reduced pulmonary compliance and increased work of breathing
- Ulceration of pulmonary mucosa which results in increased airway resistance and possible hypoxemia
- Loss of surfactant which results in atelectasis, intrapulmonary shunting and possible hypoxemia
- Hypothermia caused by heat loss

There are two common ways to provide heat and humidification during mechanical ventilation. The hygroscopic heat-moisture exchanger (HHME) is the most commonly used. The HHME is placed between the Y-connector and the airway. It retains the heat and moisture present in the air exhaled by the patient and returns it with the next inspiration. HHME devices are relatively inexpensive and easy to maintain, and have been shown to be very effective in most circumstances. **There are three contraindications to the use of an HHME: thick tenacious secretions, bloody secretions, and hypothermia.** If thick or bloody secretions are deposited on the membrane of the HHME, the

resistance to gas flow through the circuit is dramatically increased. Such an increase in airway resistance could result in increased work of breathing or insufficient inhaled volumes. The HHME is unable to provide adequate relative humidity for patients with core body temperatures $< 32^{\circ}\text{C}$ or 90°F due to the physical properties of the materials it contains.



HHME Filter

HHME filters are changed every 24 hours (or as needed) because accumulated moisture increases inspiratory resistance over time.

The second way that heat and moisture are provided is through a heated water humidification system such as a Conchatherm™. Heated water humidifiers heat and evaporate water. The resultant water vapor is directed into the inspiratory tubing of the ventilator circuit. The temperature of the inspired gas must be monitored to prevent thermal injury to the patient. All devices display the temperature setting and are equipped with alarms. Heated water humidifiers can be used for any patient, but are more difficult to maintain than the HHME. The clinician must monitor the water levels and temperature on a continuous basis. Heated water humidifiers also result in a considerable amount of water condensation within the ventilator circuit. If the water accumulates in the inspiratory or expiratory tubing, it can cause increased work of breathing or insufficient tidal volumes. Accumulation of water in the circuit can also result in inadvertent delivery of the water down the patient's airway during patient movement. Water traps are placed within the circuit and must be emptied frequently to prevent these complications.

Do not use a heated water humidifier in the same circuit as an HHME. Particulate water within the HHME prevents adequate delivery of humidity from either device. If water occludes the HHME, the patient cannot be ventilated adequately and may not be able to exhale.

Principles of Mechanical Ventilation

Unfortunately there is little standardization in terminology used to describe mechanical ventilation. Terms to describe different modes of ventilation vary within the literature and between manufacturers. Pressure control ventilation on one type of ventilator is not the same as pressure control ventilation on another, and the literature may differ from either one of the manufacturer's definitions. This may cause considerable confusion for the clinician at the bedside, thus, this packet attempts to clarify as much as possible. The information provided in this packet may differ from other resources and manufacturer descriptions, due to the lack of uniformity in terminology.

Ventilator Modes and Variables

A ventilator mode is a description of how breaths are supplied to the patient. The mode describes how breaths are controlled (pressure or volume), and how the four phases of the respiratory cycle are managed. The four phases are described as:

- The change from expiration to inspiration or what triggers (initiates) a breath
- Inspiration or breath delivery; largely determined by the control variable
- The change from inspiration to expiration or what cycles (ends) a breath
- Expiration or a passive process dependent on time

Each of these phases has a set of variables associated with it. Some of the variables are set by the clinician, some are calculated by the ventilator's internal programming, and others vary with the patient's respiratory rate, pulmonary compliance and airway resistance. Detailed information on common ventilator modes is discussed later in this packet. Modes of ventilation beyond the scope of this packet include automated pressure regulated ventilation (APRV), high-frequency oscillating ventilation (HFOV), and independent lung ventilation. For more information on these and other modes of ventilation refer to the reference list.

Control Variables

There are two fundamental methods to control the delivery of a breath. The clinician can choose to keep either volume or pressure constant from breath to breath. The control variables most commonly used to describe modes of ventilation are volume-controlled (VC) ventilation and pressure-controlled (PC) ventilation. Within these two categories are multiple ways to tailor specific modes.

Many practitioners are more familiar with the volume-controlled ventilator. The ventilator delivers a set tidal volume with each programmed breath. Volume-control ventilation is advantageous when it is important to maintain specific tidal volumes. Research has revealed, however, that high tidal volumes can cause significant complications such as parenchymal injury and pneumothorax. In volume-controlled modes, the pressures used to deliver the set tidal volume will vary as a function of the patient's pulmonary resistance and compliance.

Pressure-controlled modes of ventilation have become more frequent in use in recent years. They aid in avoiding the high pressures that can be generated by volume-controlled ventilation of non-compliant lungs. In pressure-controlled modes the tidal volume is not guaranteed. Each breath is delivered at a set pressure for a duration of time calculated based on the respiratory rate and the ratio of inspiration to expiration. Most ventilators perform these calculations internally based on parameters set by the clinician. The tidal volume will vary with changes in the patients lung mechanics.

Ventilators also exert control over flow and time. Time is based on ventilatory rate. Other time functions the ventilator may control are inspiratory (I) and expiratory (E) time and the subsequent I: E ratio. Flow control is similar to volume control. The differences between flow and volume control are minimal for most basic modes of ventilation. For the purposes of this section they are considered synonymous.

Phase Variables

Beyond control variables, phase variables provide a more detailed picture of ventilator function. The phase variables control the transitions between inspiration and expiration and include trigger and cycle variables.

Trigger Variables

Trigger variables determine how a breath is started. A breath can be initiated (triggered) either by the patient, the ventilator or the clinician. Patient-triggered breaths generally occur in response to changes in pressure or flow. The exact method used depends on the manufacturer. Patient-triggered breaths can elicit variable responses from the ventilator depending on how the machine is set. Patient-triggered breaths are termed spontaneous, assisted or supported breaths.

- Spontaneous breath is completely regulated by the patient with no contribution by the ventilator.
- Assisted breath is initiated by the patient, but all other aspects of the breath are controlled by the ventilator.
- Supported breath is initiated and ended by the patient, but the breath is delivered under positive pressure by the ventilator.

Ventilator-triggered breaths are initiated in response to a timer inside the ventilator. The exact time interval is determined by the set rate.

Clinician triggered breaths are delivered manually by the clinician at the bedside by pressing a button on the ventilator. Some ventilators deliver a standard breath according to the programmed settings when the button is pressed. Others will continue to deliver gas for the duration of time the button is depressed, stopping when the button is released or the maximum pressure limit is reached.

Cycle Variables

Cycle variables determine how a breath is ended. A positive pressure breath will always end because a variable has reached a set value. The change from inspiration to expiration can be determined by:

- Volume cycle (desired volume met),
- Flow cycle (desired flow met),
- Pressure cycle (desired pressure met), or
- Time cycle (elapsed time was met)

A volume-cycled ventilator is one which stops pushing gas into a patient's lungs when it has reached a preset volume (e.g. Bear II). The advantage of a volume-cycled ventilator is that it will deliver the set tidal volume of gas in spite of conditions of higher than normal airway pressure. Clinical applications include ARDS, bronchospasm, increased secretions or mucous plugs in the airway. This advantage can also be considered a disadvantage because overly high pressures can cause injury and discomfort.

A pressure-cycled ventilator is one that stops pushing gas into a patient's lungs when a preset airway pressure is reached. An example of a pressure-cycled ventilator is a Bird ventilator, which is commonly used to give intermittent positive pressure breathing (IPPB) treatments. The major disadvantage of pressure-cycled ventilators is that they will not deliver the desired tidal volume if pulmonary compliance is decreased. This frequently occurs with bronchospasms, seizures, and chest or abdominal splinting due to pain. Pressure-cycled ventilators are not routinely used in adults.



Bird Ventilator

Some ventilators can function as pressure or volume-cycled ventilators, depending upon the programmed mode of ventilation. The Bear 1000 and Siemens Servo are examples of this type.



Bear 1000 Ventilator



Siemens Servo Ventilator

Ventilator Settings

From a clinician standpoint there are essentially seven variables of volume ventilators that are manipulated to optimize the patient's ETCO_2 , SaO_2 or arterial blood gases. These variables are tidal volume (TV), rate, fraction of inspired oxygen (FiO_2), airway pressure, PEEP, pressure support, and mode.

Terminology related to ventilator settings

Mandatory	A positive pressure breath that is controlled, triggered and cycled by the ventilator in accordance with programmed settings.
Assisted	A breath that is triggered by the patient, but controlled and cycled by the ventilator. Other than being triggered by the patient, an assisted breath is identical to a mandatory breath.
Supported	A positive pressure breath that is triggered and cycled by the patient but controlled by the ventilator. Supported breaths are delivered with positive pressure, but may vary in length, tidal volume or pressure depending on the patient's respiratory muscle compliance.
Spontaneous	A breath that is initiated controlled and ended by the patient without any input from the ventilator. Spontaneous breaths are negative pressure breaths.

Mode of Ventilation

There are three basic modes of ventilation: continuous mandatory ventilation (CMV), assist-control (A/C) and intermittent mandatory ventilation (IMV). Any of these modes can be volume-controlled (VC) or pressure-controlled (PC).

- Continuous mandatory ventilation (CMV), as the name implies, is completely controlled by the ventilator; no spontaneous breaths are allowed. It is pressure or volume-controlled, machine triggered and machine cycled.
- Assist-control ventilation (A/C) allows the patient to trigger a breath, but the machine otherwise controls all breaths. It is pressure or volume-controlled, machine and patient triggered, and machine cycled.
- Intermittent mandatory ventilation (IMV) provides a minimum number of programmed breaths; the patient may initiate spontaneous breaths at any time. In this mode a machine breath will be pressure or volume controlled, machine triggered and machine cycled. A spontaneous breath will be pressure controlled, patient triggered and patient cycled.

The modes are most appropriately identified by first indicating the control variable (volume-controlled or pressure-controlled) followed by the mode. For example, the correct abbreviation for volume-controlled continuous mandatory ventilation is VC-CMV. The choice of mode depends upon patient characteristics and the type of equipment available. Each of these modes is discussed in detail in a later section.

Tidal Volume

Tidal volume (TV) is the volume of gas delivered to the patient with each breath. Tidal volume may also be expressed as V_T . TV is only set for volume-controlled modes of ventilation and is usually 8-12cc/kg of body weight. There are circumstances where lower tidal volumes are used, and current literature favors this practice. The tidal volume is closely related to ventilation. As such, it is most often manipulated in response to abnormal levels of CO_2 . In pressure-controlled modes of ventilation the tidal volume will be displayed on the ventilator, but it is not set by the clinician. Modern ventilators graph volume changes over time. These waveforms can be used to individualize the ventilator settings and optimize therapy. A review of waveforms is provided later in this packet.

Excessive tidal volumes have been linked to ventilator-induced acute lung injury called volutrauma. Volutrauma is now believed to be one of the greatest threats to the lung posed by mechanical ventilation. Acute lung disease typically produces some areas of the lung that are severely diseased and others that are nearly normal. Gas flow will always follow the path of least resistance. When large tidal volumes are delivered under positive pressure the delivered gas flows more rapidly and forcefully into the more normal areas. This rapid forceful introduction of gas causes over-distension of the alveoli in these areas, leading to parenchymal injury, loss of surfactant and possible alveolar rupture. It is now thought that excessive volume rather than excessive pressure is the most common cause of ventilator-induced pneumothorax.

Rate

The set ventilatory rate is the minimum number of breaths delivered to the patient per minute. The actual rate may be higher than the set rate if the patient is initiating spontaneous breaths. Rate is also a determinant of ventilation, and is adjusted in response to the patient's CO_2 levels. Minute ventilation is the rate multiplied by the tidal volume.

Oxygen Percentage

The fraction of inspired oxygen (FiO_2) is the amount of oxygen delivered to the patient. FiO_2 can be expressed as a decimal fraction or a percentage. Oxygen concentrations of greater than 0.50 (50%) increase the risk of oxygen toxicity if delivered for more than 24 hours. Supplemental oxygen is administered in response to low PaO_2 , SpO_2 , or indicators of tissue hypoxia.

Listed below are some of the causes of tissue hypoxia that are unresponsive to treatment with supplemental oxygen.

- Anemia is associated with high oxygen saturation, but limited oxygen carrying capacity. Additional oxygen will not improve oxygen delivery to the tissues under these circumstances. The only effective treatment for tissue hypoxia related to anemia is to increase the hemoglobin level.
- Right to left shunt occurs when a proportion of the circulated blood passes through the pulmonary circulation without coming into contact with any functional gas exchange units. This phenomenon can occur with congenital heart defects and severe atelectasis, pneumonia or pulmonary edema. Increasing the amount of delivered oxygen will not help in this case, because the blood would still not come into contact with the extra oxygen.
- Hypoxia related to circulatory insufficiency does not improve with supplemental oxygen due to inadequate delivery from a dysfunctional cardiovascular system. To correct this type of hypoxia, adequate circulation must be restored.
- Tissue dysoxia occurs when a toxin prevents the cells from utilizing delivered oxygen. Dysoxia is associated with cyanide toxicity and severe sepsis. The only treatment is to reverse the effects of the toxin.

CLINICAL APPLICATION ADULT RESPIRATORY DISTRESS SYNDROME

The hallmark of ARDS is persistent hypoxemia despite administration of 100% oxygen. ARDS causes some alveoli to fill with fluid and others to collapse, increasing pulmonary shunting. Until the alveoli can be expanded with air, no amount of supplemental oxygen will correct the problem.

Peak Airway Pressure

Airway pressure reflects global alveolar pressure and is monitored continuously, usually by a sensor at the Y-connector. The highest pressure recorded in the ventilatory cycle is called the peak airway pressure or peak inspiratory pressure (PIP). The maximum allowable PIP is set on the ventilator. Excessive pressure can lead to barotrauma or pneumothorax. If the PIP exceeds the set value, an alarm will sound and gas delivery will halt until the next breath is triggered. In pressure-controlled modes the PIP will be constant for each breath. In volume-controlled modes, the PIP will vary from breath to breath.

The normal peak inspiratory pressure on a mechanically ventilated patient with normal lungs is approximately 20cm H_2O . The maximum allowable peak pressure varies from patient to patient. The pulmonologist or intensivist collaborates with other members of the multidisciplinary team to determine the safest pressure for each patient. PIP values should be trended in volume-controlled

modes to detect changes in pulmonary compliance. Several ventilators graph airway pressures. These graphic displays can aid in optimizing mechanical ventilation for each patient. Patient monitoring by analysis of the pressure graphic is superior to trending of isolated PIP values.

Positive End Expiratory Pressure (PEEP)

The normal airway pressure at the end of expiration and before inspiration is zero. Application of pressure by the ventilator at this stage of the ventilatory cycle is called PEEP. PEEP aids in propping open alveoli that would otherwise collapse during the expiratory phase. It is a very effective treatment modality for V/Q mismatching caused by atelectatic processes, and is a key component of ventilator therapy for patients with ARDS. PEEP enhances oxygenation by increasing the number of available gas exchange units and is adjusted in response to measures of oxygenation. PEEP is measured at the bedside by noting the airway pressure reading at the end of expiration. If the reading is greater than zero, PEEP is present. A PEEP setting of 5cm H₂O is considered equivalent to the effect of the closed glottis, and is called physiologic PEEP. Therapeutic PEEP levels range from 10 – 35 cm H₂O or more.

Pressure Support

Pressure support can be used in combination with other ventilatory modes that permit spontaneous breathing. Pressure support works by responding to a patient's inspiratory effort with a positive pressure breath delivered at a set pressure. The patient can draw more volume by contributing muscular effort to the breath, or the ventilator can deliver the entire breath if muscular effort is not sustained. A pressure support breath is pressure controlled, patient triggered, pressure limited, and patient cycled. The volume of a pressure support breath will vary in proportion to the patient's inspiratory effort.

Pressure support can be used to compensate for the increased airway resistance of an endotracheal tube, or to facilitate weaning from mechanical ventilation. Pressure support enhances spontaneous tidal volumes and therefore is adjusted in response to CO₂ levels. Pressure support typically ranges from 5 – 30 cm H₂O.

Ventilator Waveforms

Why Use Waveforms?

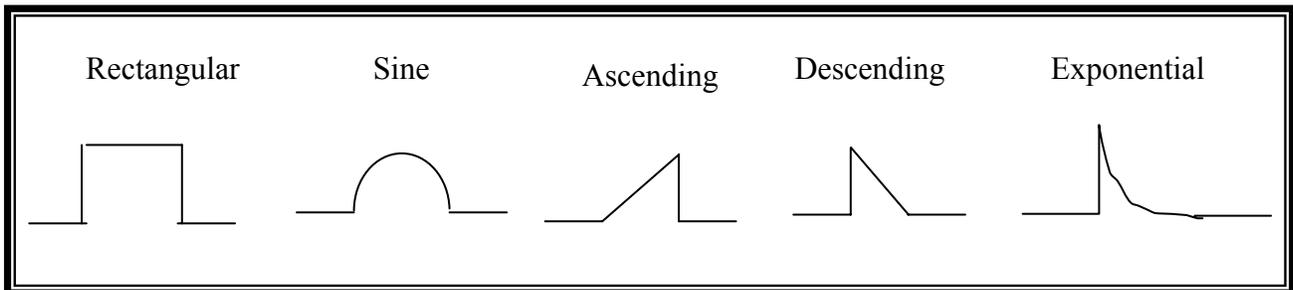
Waveforms generated by the ventilator reveal real-time characteristics of each breath that cannot be determined from static numeric data alone. Historically, the primary parameters monitored on a mechanically ventilated patient were the PIP and exhaled tidal volume. Limiting monitoring to these two numbers meant the clinician could not determine if the patient had sufficient flow, hyperinflation, air trapping, secretions, or appropriate PEEP levels. Today, the use of ventilator waveforms enables the bedside clinician to assess the patient's response to mechanical ventilation and fine-tune the settings to maximize patient comfort and therapeutic benefit. Expert waveform interpretation takes considerable time and practice to develop. This packet attempts to introduce the fundamental concepts of ventilator waveform interpretation. For more detailed information of waveform interpretation, references have been provided at the end of this packet.

Waveform Fundamentals

Waveforms are generated from parameters monitored at the patient-ventilator interface; generally at the Y-connector. Parameters can be established to monitor the flow, pressure, and volume of gases between the ventilator and the patient. These measurements are plotted graphically over time to provide waveforms or against each other to produce loops. Although a large amount of data is available from the ventilator displays, continual patient assessment must be combined with this data to provide a complete picture of the patient's status.

Below are the types (shapes) of waveforms produced by the ventilator without patient influences. These are idealized waveforms. At the bedside the waveforms don't appear exactly as pictured, but have characteristics in common with these shapes.

Classification of Ventilator Waveforms

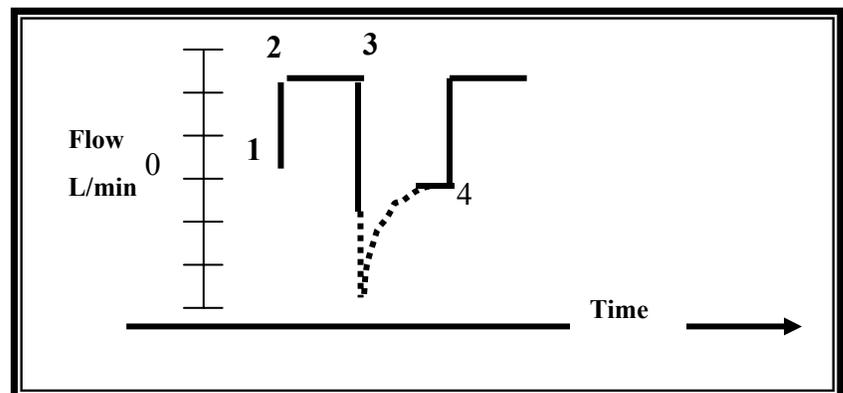


Flow Waveforms

The flow waveform shows changes in the rate of flow of gas over time. The vertical axis of the flow graphic represents L/min flow; the horizontal axis represents time. A deflection above the baseline of the flow waveform indicates gas is flowing into the patient (inspiration). A deflection below the baseline of the flow waveform indicates that gas is flowing out of the patient (expiration). The flow waveform can be used to determine whether the ventilator is well-synchronized with the patient, the presence of air trapping, and whether the patient's flow needs are being met.

Components of the Flow Waveform

In the illustration, the solid line represents inspiration from a mechanical breath and the dashed line represents expiration. (1) Represents baseline or zero flow. (2) Represents rapid increase in flow at the start of inspiration. The interval from (2) to (3) is the inspiratory time. (3) Represents the cessation of delivery of flow to the patient. The interval from (3) to (4) is the expiratory time.



Flow Settings

The ventilator and/or the respiratory muscles create the pressure that results in a flow of gas. In mechanical ventilation, flow is usually set indirectly. Most ventilators calculate the flow rate based on the selected control variable (volume or pressure) and the rate. The highest point of the flow waveform represents peak flow.

Peak Flow

Peak flow is an important determinant of patient comfort. A peak inspiratory flow that is too low can lead to increased work of breathing as the patient struggles to draw more air from the ventilator. Low flow also increases the time required to achieve set volumes (or pressures), prolonging the inspiratory time. Consequently, less time is available for expiration and may lead to air trapping. Insufficient peak flow is a common unrecognized cause of patient ventilator dys-synchrony otherwise known as “bucking the ventilator.” Consider insufficient peak flow as a source of agitation before sedating a mechanically ventilated patient.

Peak inspiratory flow that is set too high can cause unnecessarily high airway pressures and may increase the risk of barotrauma. A patient’s ideal peak flow is not static and may need to be adjusted several times during the day to provide optimal patient comfort.

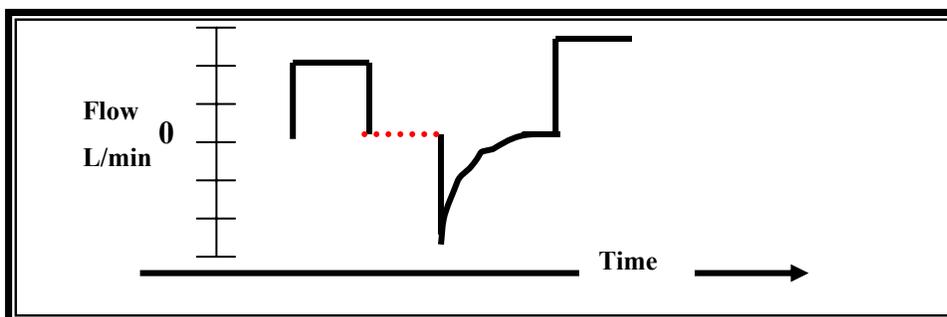
Take a Walk in My Shoes



To see how the patient feels when inspiratory flow is insufficient, try this. Exercise vigorously enough that you feel a bit short of breath. Now place a drinking straw between your lips and form a tight seal. Pinch your nose, and breathe only through the straw. How does it feel?

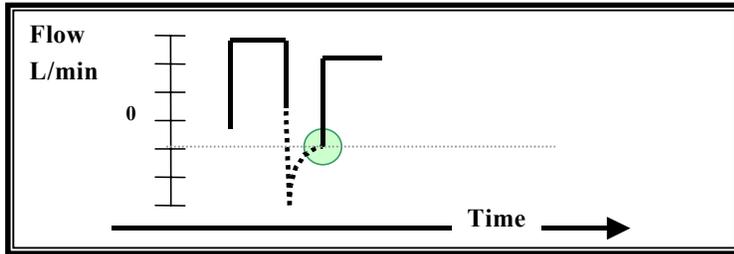
Inspiratory Pause

Inspiratory time is determined by the set volume or pressure, peak flow and flow pattern. It can also be a preset value. An inspiratory pause (broken line) lengthens the inspiratory time and may exceed the duration of flow. Inspiratory pauses can be used to increase the time available for gas mixing in the alveoli. If inspiratory pauses are used, the clinician must be sure that sufficient time remains in the ventilatory cycle for full expiration. If expiratory time is cut short, air trapping can result. Inspiratory pauses are used to manipulate the I:E ratio.



Expiratory Time and Air Trapping

The resistance of the small airways and the resistance of the artificial airway are the primary determinants of expiratory flow. If airway resistance is high, flow will be reduced. If flow is



reduced, more expiratory time will be required for the alveoli to fully empty, increasing the risk of air trapping. Air trapping is reflected on the flow waveform as an inspiratory rise occurring before the expiratory limb has reached zero flow. The illustration shows this phenomenon in the highlighted circle. Remember that air

trapping will increase inspiratory pressure and decrease tidal volumes. Air trapping also causes significant discomfort for the patient.

Take a Walk in My Shoes



To learn how the patient feels when air trapping occurs, try this. Take several deep rapid breaths in quick succession, let only one-fourth of the last breath out, and hold your breath. How does it feel?

Pressure Waveforms

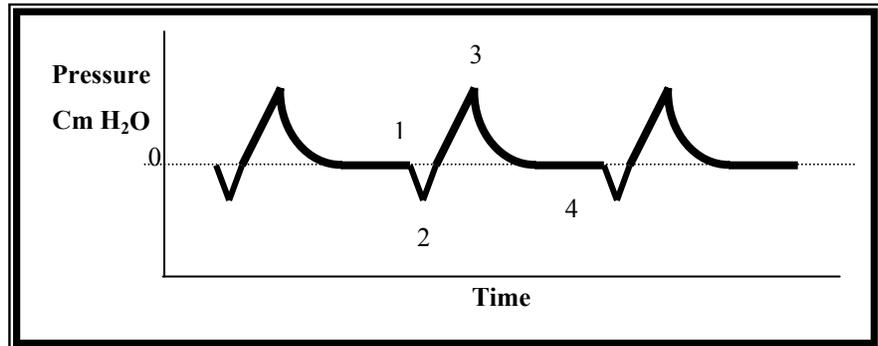
The pressure waveform depicts changes in airway pressure over time. These measurements are usually taken from the Y-connector. The vertical axis represents pressure (usually expressed in cm of H₂O) and the horizontal axis represents time. Positive deflections of the waveform reflect positive airway pressure, and negative deflections indicate negative airway pressure. The only negative pressures present in mechanical ventilation are seen when a patient initiates a spontaneous breath.

The baseline of the pressure waveform is normally zero. If PEEP is applied, the baseline pressure will equal the PEEP setting. Auto-PEEP will not appear as an increase in the baseline pressure; methods for detection of auto-PEEP are discussed later in this packet.

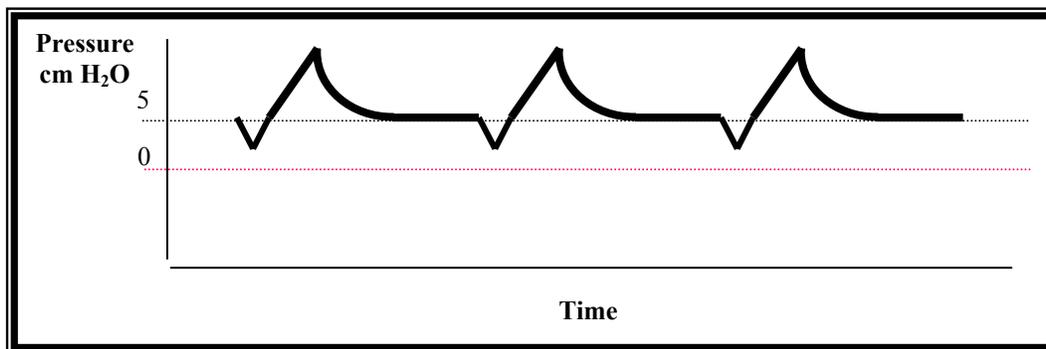
The rectangular pressure waveform is most typical of pressure-controlled modes of ventilation and the exponential pressure waveform is typical of volume-controlled modes. Spontaneous breathing tends to produce a sine waveform.

Components of the Pressure Waveform

The pressure waveform has four basic components. The illustration shows a patient triggered volume-controlled breath. (1) Represents the negative pressure of the patient's spontaneous inspiratory effort. (2) Is the onset of the positive pressure breath. (3) Is the PIP. (4) is the end of exhalation.



If the PEEP is set to zero, the breath should begin and end at the zero baseline. When PEEP is applied, the waveform baseline will be greater than zero. The following illustration shows the same waveform with 5 cm H₂O of PEEP added. Notice that the patient's inspiratory effort now does not produce a negative pressure, though it is of the same magnitude. On some types of ventilators, this would result in a failure to recognize the patient's respiratory effort.



Pressure Measurements

Pressure measurements made during the ventilatory cycle include peak pressure, inspiratory plateau pressure, mean inspiratory pressure, positive end-expiratory pressure and mean airway pressure.

- Peak pressure is the highest pressure during inspiration. The peak pressure is affected by the sum of resistance from the airway and the lung. Peak pressures are important because high peak pressure increases the risk of barotrauma, and may cause volume breaths to be cut short due to pressure-limiting.
- Inspiratory plateau pressure is the end-inspiratory pressure under no-flow conditions, such as an inspiratory pause. The inspiratory plateau pressure reflects the pressure needed to distend the alveoli. Plateau pressure eliminates the influence of airway resistance. As lung compliance decreases, inspiratory plateau pressures increases and vice versa.
- Mean inspiratory pressure is the average pressure during inspiration. As mean inspiratory pressure increases, so does the risk of volutrauma. Mean inspiratory pressure is influenced by PIP, plateau pressure and PEEP.
- Positive end-expiratory pressure (PEEP) is an increase in the baseline airway pressure. If PEEP is added, it will also increase the PIP, plateau and average inspiratory pressures. If the PEEP

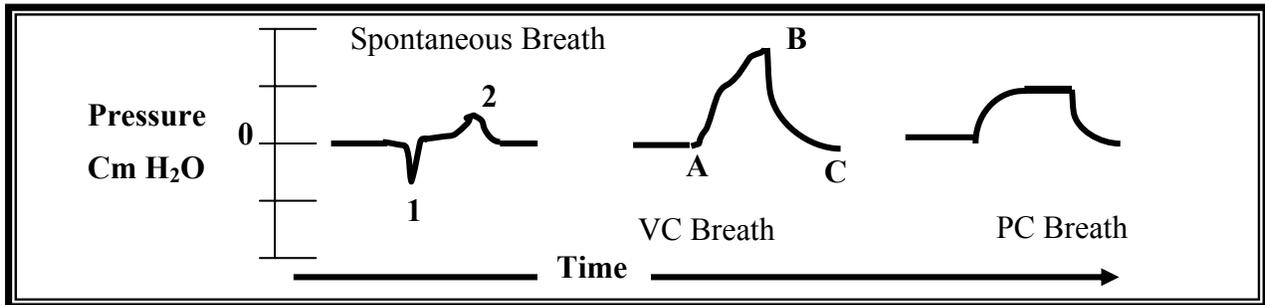
setting is high, pressure limiting of volume breaths, or volume limiting of pressure breaths may occur.

- Mean airway pressure is the average pressure throughout the ventilatory cycle. This incorporates all of the above pressures in addition to expiratory pressures and any negative pressures generated by the patient’s spontaneous attempts at breathing.

Locations for Pressure Monitoring

Pressures can be monitored in many locations; the most common is at the Y-connector. Other locations for pressure monitoring are the expiratory limb of the circuit within the ventilator, the distal end of the endotracheal tube and the esophagus. Pressures measured in the ventilator or circuitry are considered airway or airway opening pressures. Pressures measured in the distal endotracheal tube are considered tracheal pressures. Tracheal pressure monitoring is a more direct reflection of lung pressures because the influence of the endotracheal tube is removed from the measurement. Pressure monitoring from the distal endotracheal tube is not commonly used. Pressures measured in the esophagus are reflective of pleural pressures and can be used to differentiate airway pressures from lung pressures. Esophageal pressures are not commonly monitored in the clinical setting. Pressure waveforms discussed in this packet are all airway pressures.

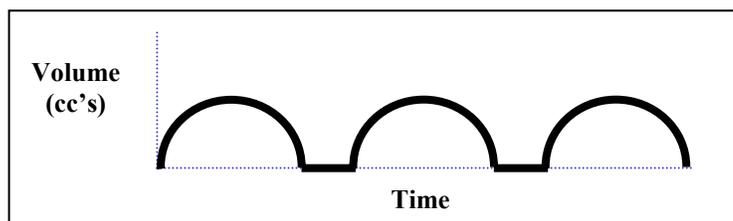
In the spontaneous breath waveform below: (1) represents a drop in pressure at the beginning of inspiration and (2) represents the rise in pressure during the expiratory phase. The depth of (1) is related to the strength of the patient’s inspiratory effort. In the volume-controlled (VC) breath, the interval from A – B represents the inspiratory time. (B) Represents the peak inflation pressure or peak inspiratory pressure. The interval from A – C represents the duration of positive pressure of the mechanical breath. In the pressure-controlled (PC) breath, the peak pressure flattens off at the top. The pressure at which this flattening occurs will vary based on the amount of pressure set by the clinician.



In volume-controlled ventilation, the pressure graphic changes depending on flow rate, flow pattern, airway resistance and lung compliance. In pressure-controlled ventilation, the pressure graphic will remain more constant and is shaped by the parameters set by the clinician.

Volume Waveforms

Volume delivered depends on the amount of flow and inspiratory time. In pressure-controlled modes, volume is also influenced by the respiratory system mechanics of compliance and



resistance. The ventilator measures both delivered and exhaled tidal volume (V_t). The volume graph displays volume in cc's on the vertical axis over time on the horizontal axis. It usually assumes a sine wave shape. In volume-controlled modes of ventilation the volume graphic will remain constant while the pressure graphic varies. In pressure-controlled modes the volume graphic will vary from breath to breath with changes in pulmonary compliance and resistance.

Modes of Mechanical Ventilation

The best mode of mechanical ventilation is the one that provides maximum therapeutic benefit with the fewest side effects. Mode selection and individual ventilator settings are geared towards the patient's diagnosis and history as well as integrated data from laboratory, radiology and physical examination. Listed is a brief review of the most commonly used modes in mechanical ventilation. For more detailed information, refer to one of the references located at the end of the packet.

Continuous Mandatory Ventilation (CMV)

CMV completely controls the patient's ventilation. It "locks out" the patient's own spontaneous efforts at breathing. The rate that is set on the ventilator is exactly what the patient receives and no more. This mode can be either pressure-controlled (PC-CMV) or volume-controlled (VC-CMV). CMV is often referred to as "control mode."

The major disadvantage of CMV is that it is not synchronized with the efforts of the patient. When the patient is "out of sync" with the ventilator, he attempts to exhale as the ventilator is in the inspiratory phase. As a result, airway pressure builds to abnormally high levels and the remainder of the inspiratory volume is not delivered. This "bucking" causes a high-pressure alarm.

Signs and symptoms of ventilator dyssynchrony include:

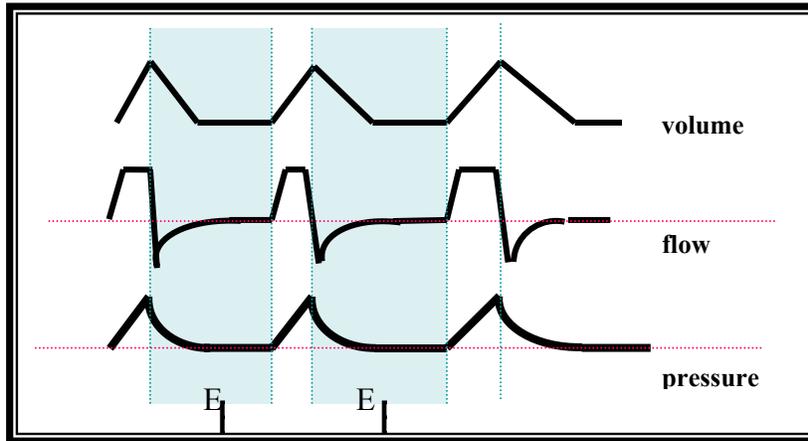
- Agitation
- Diaphoresis
- Tachycardia
- Tachypnea
- Paradoxical thoraco-abdominal breathing pattern
- Increased PIP (peak inspiratory pressure)

Note: The CMV mode is inappropriate for an alert patient with spontaneous breathing!

CLINICAL APPLICATION CMV MODE

If the patient attempts to draw a spontaneous breath while in CMV mode, the ventilator will not deliver air until the next programmed breath. This results in increased work of breathing, air hunger, and agitation as the patient struggles for a breath. Clinicians sometimes heavily sedate these patients because they forget that this anxiety and agitation can be easily resolved by switching to a mode of ventilation that permits spontaneous breaths.

Below is a concurrent display of volume, flow and pressure waveforms as seen in the VC-CMV mode. In this mode volume will remain constant unless maximum set pressures are exceeded. The flow and pressure waveforms will vary with changes in pulmonary compliance and airway resistance. The shaded areas marked with “E” represent the expiratory phase. Note that the expiratory phase is significantly longer than the inspiratory phase. If expiratory time shortens significantly there may not be enough time for the lungs to completely empty before the next breath is delivered. The inspiratory to expiratory time (I:E ratio) is usually 1:2. Patients with obstructive lung diseases (e.g. emphysema, asthma) require longer expiratory times than normal.

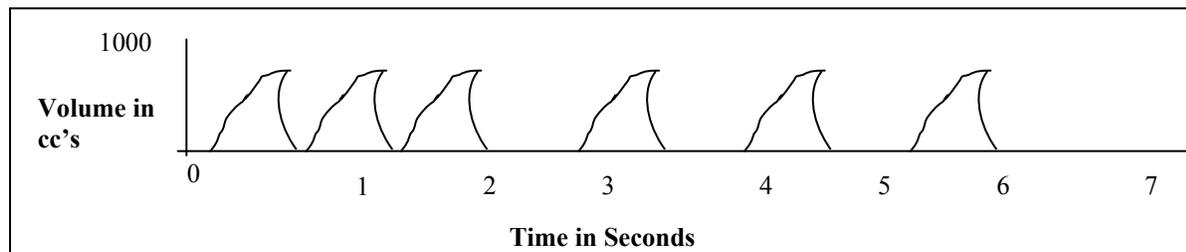


Volume Controlled CMV

Assist Control (A/C) Ventilation

The A/C mode is similar to CMV, but it allows the patient to trigger an assisted breath at any time. As with CMV, A/C ventilation can be pressure or volume controlled. The machine is set to “sense” the patient’s negative inspiratory effort, and is thus triggered to deliver the preset tidal volume or inspiratory pressure. All delivered breaths, whether mandatory or patient-triggered, will be delivered by the ventilator according to the set parameters.

The A/C rate is the minimum number of full ventilator breaths the patient will receive. The actual respiratory rate is equal to the A/C rate plus any patient-triggered breaths per minute.



A/C ventilation with patient triggering

All breaths in the assist-control mode receive the same FiO_2 and tidal volume. Hyperventilation and respiratory alkalosis may result from occurrences that increase the patient’s spontaneous rate such as anxiety or neurological factors. A high sensitivity setting that causes the machine to cycle too frequently can also cause this problem. An increased risk of air trapping with high respiratory rates may also potentially occur with the A/C ventilation.

Intermittent Mandatory Ventilation (IMV)

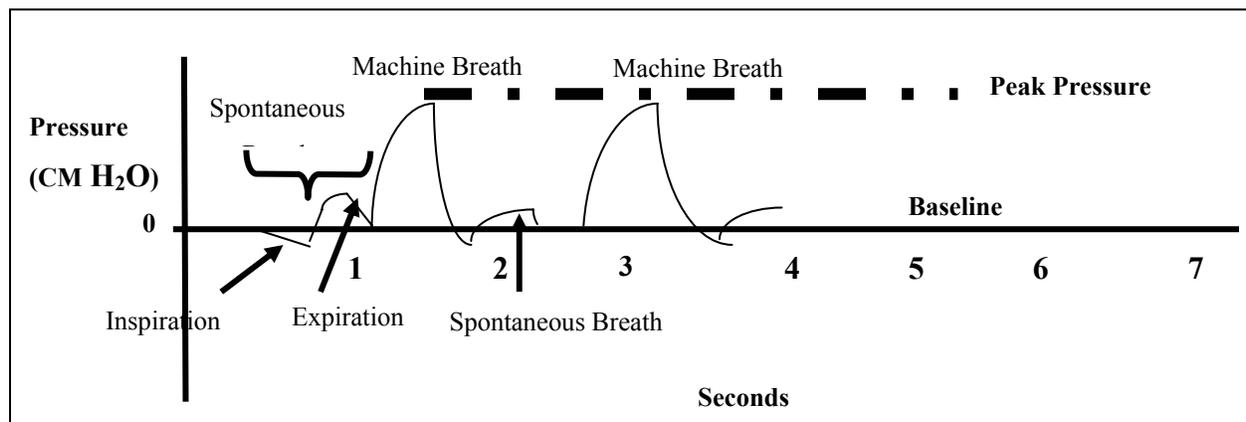
Like CMV and A/C ventilation, intermittent mandatory ventilation (IMV) can be either pressure or volume-controlled. IMV is among the most commonly used modes of ventilation. In this mode the ventilator delivers a preset rate, tidal volume (or inspiratory pressure) and FiO₂. The patient may also draw spontaneous breaths in-between mandatory breaths. Unlike A/C, breaths that the patient takes spontaneously do not trigger or cycle the ventilator. Patient-initiated breaths are completely spontaneous, neither assisted nor supported by the ventilator.

Synchronized Intermittent Mechanical Ventilation (SIMV) works similarly to traditional IMV. The advantage is that it synchronizes ventilator breaths with the patient's spontaneous respiratory effort. The ventilator senses spontaneous breathing during a window of time prior to the next mandatory breath. If spontaneous effort is detected, the breath is delivered in concert with the patient's effort. This is more comfortable for the patient and prevents "stacked" breaths; a mandatory breath delivered too soon after a spontaneous one. Most ventilators now are capable of SIMV, and many clinicians are referring to SIMV when they state the patient is receiving "IMV."

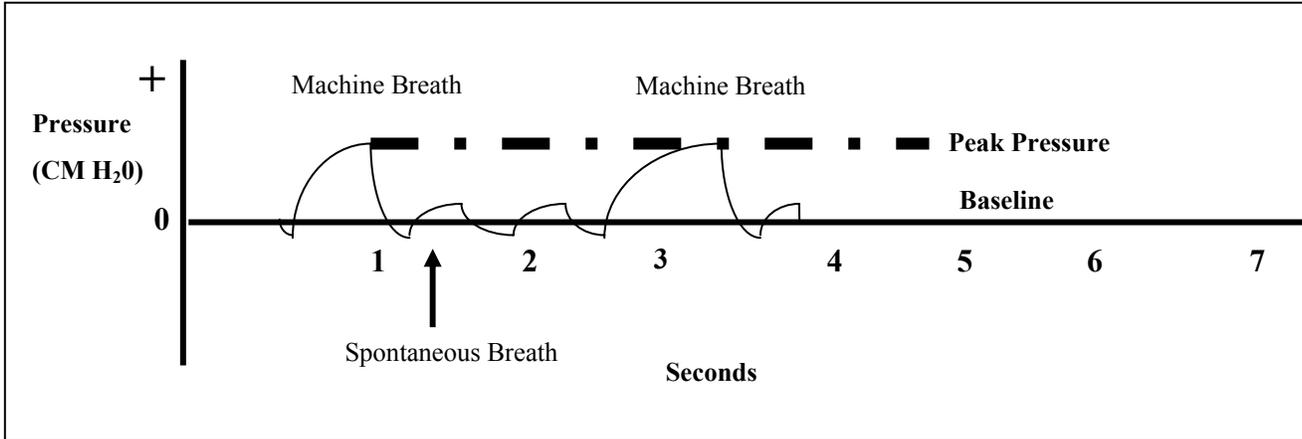


Display Setting of Ventilator

The benefits of IMV include ventilation at lower mean pressures, decreased chance of hyperventilation, decreased atrophy of accessory muscles, and improved distribution of gas throughout the lungs by the action of the diaphragm. Lower mean pressures are achieved with IMV because the patient-triggered breaths do not reach the same pressure levels as assisted breaths do in A/C ventilation. The risk of hyperventilation is lower because the spontaneous breaths rarely achieve the same tidal volumes as assisted breaths. Since all spontaneous breaths are not positive-pressure breaths, the patient's respiratory muscles continue to work more than in A/C ventilation, therefore, preventing atrophy. Conversely, the lack of assistance during spontaneous breathing may be problematic if the patient's work of breathing is too great. Improved distribution of gas occurs during any spontaneous breath that occurs under negative pressure. IMV is frequently used for weaning because it allows the patient to breathe independently between ventilator breaths.



The diagram below shows a pressure waveform for volume-controlled IMV with spontaneous breathing and breath stacking. Note the spontaneous breaths begin with a negative deflection of the pressure waveform, indicating that inspiration is occurring under negative rather than positive pressure. At the one-second mark, the ventilator has delivered a breath before the prior spontaneous breath was fully exhaled. This problem can be corrected by use of SIMV.

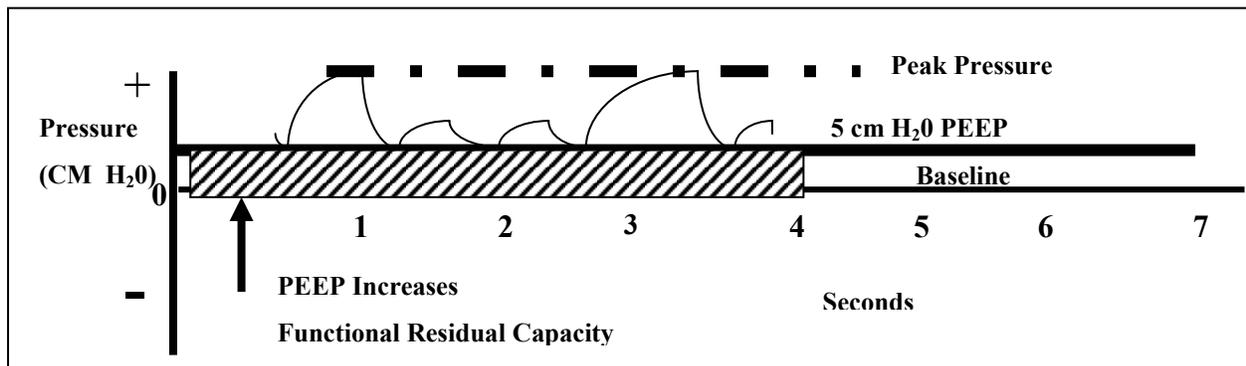


Customized Adjuncts to Ventilator Modes

Positive End Expiratory Pressure (PEEP)

One method of improving the patient’s oxygenation without increasing the FiO_2 is the use of PEEP. Basically, PEEP does not allow airway pressure to return to zero at the end of expiration. PEEP is not a mode of ventilation in itself. It is an adjunctive therapy added to other modes. PEEP is added to increase functional residual capacity (FRC) and allow for a decrease in the FiO_2 . PEEP counteracts small airway and alveolar collapse, improves alveolar ventilation and may decrease the work of breathing (at low levels). **PEEP facilitates oxygen diffusion at lower FiO_2 levels, which is safer for the patient.**

PEEP of 5cm H_2O pressure is referred to as “physiologic” PEEP because it is equivalent to the effect of the closed glottis. Therapeutic PEEP usually ranges from 10-30cm H_2O in adults. PEEP is an effective therapy for disease processes involving atelectasis; it is a cornerstone of therapy for ARDS.



Negative side effects of PEEP include decreased cardiac output, potential volutrauma and barotrauma, increased intracranial pressure and potential loss of tidal volume. Decreased cardiac output with or without hypotension occurs because PEEP increases intrathoracic pressure, which in turn decreases the venous return to the heart (preload). **Patients whose cardiac output is highly dependent on preload can suffer dramatic reductions in cardiac output and blood pressure in response to PEEP.** These reactions are especially pronounced in patients who are volume-depleted, and may be reduced in intensity with appropriate volume resuscitation. Even patients with adequate vascular volume can suffer hemodynamic compromise with high levels of PEEP. The positive effects of PEEP may be negated by the decreased cardiac output, resulting in decreased oxygen transport to tissues. Patients receiving PEEP therapy require frequent hemodynamic assessment to detect this complication and hopefully prevent hypotensive episodes. If hypotension does occur with PEEP, a clinical decision must be made whether to decrease the PEEP or support cardiac output with fluids or other modalities. The decision will be different for each patient based on his/her history, disease process, and current priorities of care.

PEEP is a continuous positive pressure applied to the airway. When PEEP is applied, the PIP, mean airway pressures and mean inspiratory pressure may also increase. If pressure-controlled ventilation is being utilized, tidal volumes will decrease with the addition of PEEP unless the inspiratory pressure setting is also increased. If volume-controlled ventilation is in use the addition of PEEP may cause early cycling of the ventilator. If the maximum PIP is reached before the breath has been delivered, pressure limiting a net loss of tidal volume results. Whenever PEEP is applied or titrated, the clinician must reassess tidal volumes for adequacy.

The overall increase in pressure increases the risk of barotrauma. Volutrauma is more likely to occur with high pressures and high tidal volumes and less likely to occur with high pressures and low tidal volumes. Therefore, adding PEEP to patients receiving high tidal volumes is risky. Monitor these patients closely for signs of pneumothorax. Avoid opening the ventilator circuit of a patient who is receiving $> 10\text{cm}$ of H_2O of PEEP, as it may take hours to reestablish the functional residual capacity (FRC) of the patient. In-line suction devices are ideal for these patients.

CLINICAL APPLICATION

ARDS

Patients with ARDS have areas of lung tissue where the alveoli open with each positive pressure breath and close again upon exhalation. This repetitive opening and closing of the alveoli causes atelectrauma. Atelectrauma damages the alveolar epithelium and reduces surfactant production, eventually causing complete atelectasis of the affected alveoli and accelerating inflammation. By maintaining these alveoli open during the expiratory phase, PEEP prevents atelectrauma.

Auto-PEEP

Some patients exhibit signs of auto-PEEP. When the expiratory time is not sufficient for the lungs to empty before delivery of the next breath (air trapping), then the alveolar pressure will be greater than the baseline at end-expiration even though PEEP has not been set on the ventilator. If pressure

continues to build in this manner, delivered tidal volumes will drop, work of breathing will increase, and the patient will experience acute discomfort until the extra volume producing the pressure is released. Patients with obstructive lung disease are prone to the development of auto-PEEP.

Auto-PEEP will not be reflected by measured end-expiratory pressure because the increase in pressure resides in the alveoli rather than the airway. To detect auto-PEEP, an expiratory hold is performed and the pressure measured. If the pressure during the expiratory hold is greater than the set PEEP, auto-PEEP is present. To correct auto-PEEP reduce airway obstruction or increase expiratory time.

CLINICAL APPLICATION COPD

Patients with COPD can have severe narrowing of the lower airways. The narrowed airways result in increased resistance to exhalation. When these patients become tachypneic, air becomes trapped in the alveoli and reduces tidal volumes. When patients are not intubated they compensate for this by performing pursed-lip breathing. Mechanically ventilated patients cannot do this so the clinician must look for auto-PEEP and either increase expiratory times or consider bronchodilators to correct the problem. Patients with COPD have this tendency toward auto-PEEP, thus PEEP is often avoided when they are mechanically ventilated.

Continuous Positive Airway Pressure (CPAP)

Continuous Positive Airway Pressure (CPAP) is PEEP with no set rate on the ventilator. CPAP is primarily used as a mode of non-invasive mechanical ventilation. It is occasionally used in the final stages of ventilator weaning, but has minimal application for the mechanically ventilated patient. Patients on CPAP do not receive positive pressure breaths from the ventilator. All breaths are initiated and ended by the patient; tidal volumes and pressures are variable from breath to breath. **CPAP aids in promotion of oxygenation in the same way PEEP does. It has no influence on ventilation.**

Pressure Support

Pressure support (PS) is an adjunct to mechanical ventilation. PS provides pressure assistance to each *spontaneous* breath. Pressure support used alone (without a mandatory rate) is called pressure support ventilation (PSV). A PS breath is supported, which means the supported breath is delivered under positive pressure but is triggered and cycled by the patient rather than the ventilator. PS reduces respiratory work of the patient by providing positive pressure during inspiration. The higher the PS setting, the more support is provided and the less work is required of the patient. Pressure support aids in ventilation, and is adjusted in response to CO₂ levels.

The set pressure level in conjunction with pulmonary compliance and resistance determines the delivered tidal volume. Tidal volumes will be variable from breath to breath and must be trended to ensure adequacy. Patients will require varying amounts of pressure support as pulmonary compliance and resistance change. In some instances, small amounts of pressure support are utilized to eliminate the increased airway resistance imposed by the endotracheal tube.

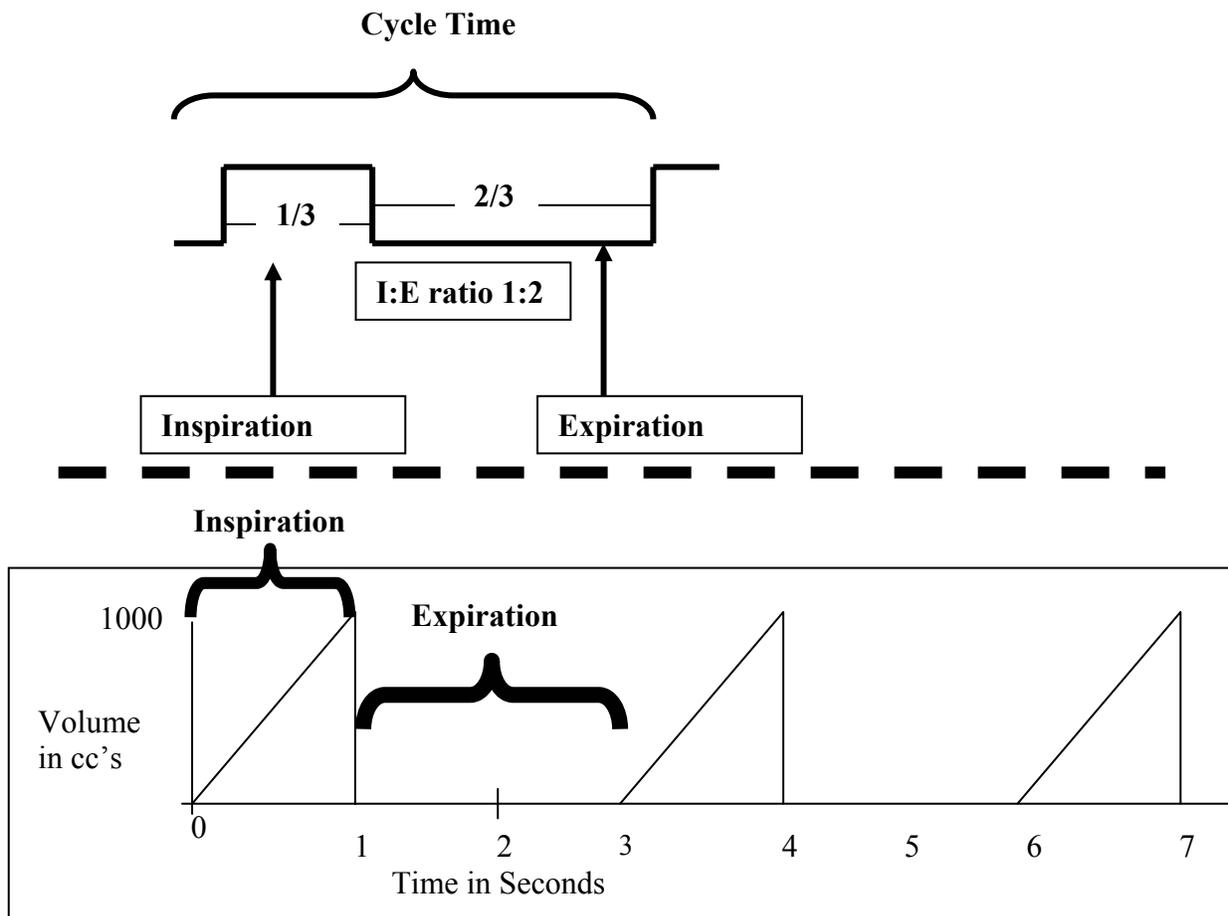
Pressure support is very helpful in weaning patients from the ventilator because it decreases the muscular work of breathing. The pressure support can be gradually reduced to provide gradual reconditioning of the respiratory muscles. The level of PSV should be set high enough to provide a

tidal volume of 8 – 12 ml/kg and to maintain a total respiratory rate ≤ 20 breaths per minute. PSV is usually used with the SIMV mode and can also be used in combination with PEEP. PSV usually ranges from 10-30 cm H₂O pressure.

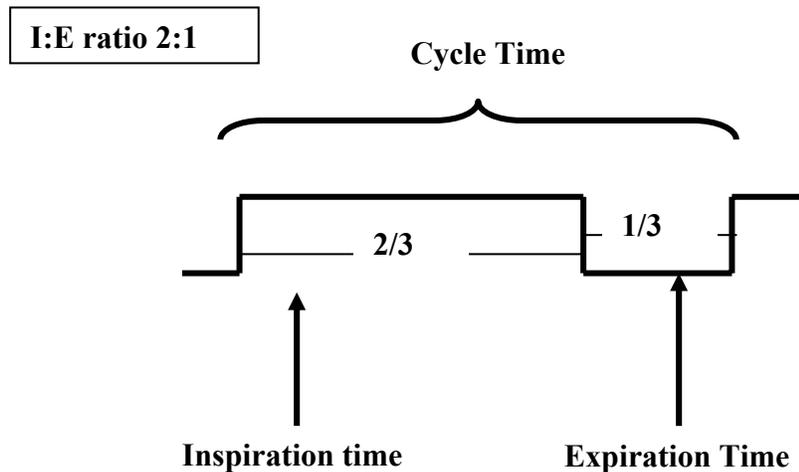
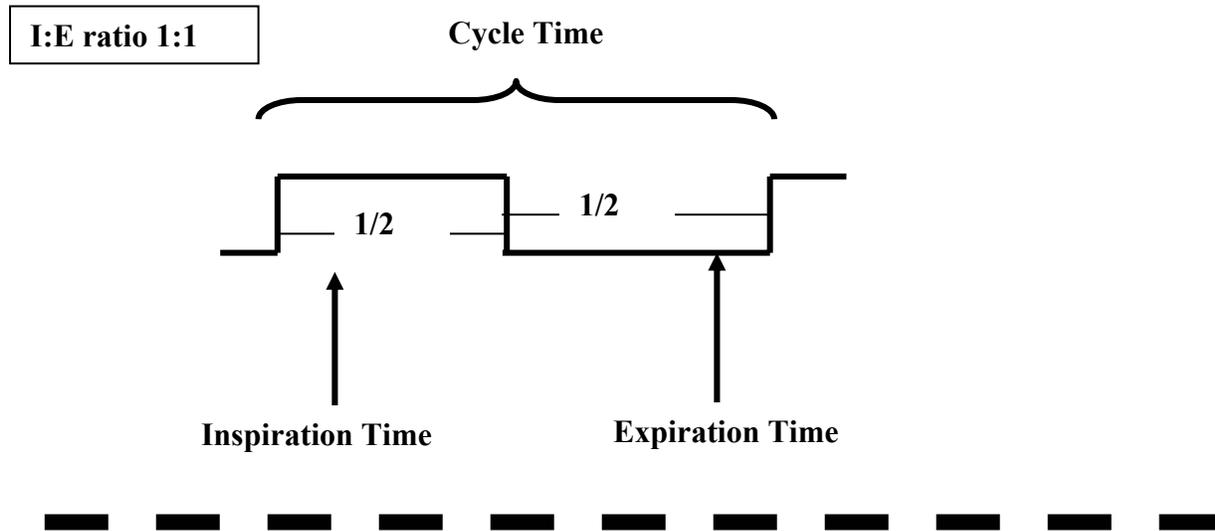
Inverse Ratios

The normal ratio of inspiration to expiration is 1:2. Inspiratory to expiratory ratios can be adjusted to improve oxygenation in refractory hypoxemia. The improvement in oxygenation is achieved by allowing the inspired oxygen more time for mixing in the alveoli. Ratios are set at 1:1, 2:1, 3:1, or 4:1 by adjusting the inspiratory time on a set rate. **Warning:** invasive hemodynamic arterial pressure monitoring is necessary as the increase in intrathoracic pressure compromises cardiac output in higher settings (> 2:1 ratio). The changes in cardiac output are similar to those described for high levels of PEEP.

Normal Respirations



Inverse I:E ratios are used in conjunction with pressure-controlled CMV. The increase in inspiratory time feels very uncomfortable to patients, and often provokes severe anxiety and agitation. Therefore, PC-CMV with inverse ratios is reserved for patients with hypoxemia refractory to other modes of ventilation. Patients who do require this treatment modality are usually gravely ill and often must be sedated and paralyzed in order to tolerate the therapy.



Pressure Control Ventilation

Pressure control ventilation (PCV) allows the clinician to limit pressure to a level that is less likely to result in barotrauma while improving alveolar ventilation and oxygenation. It can combine with an inverse I:E ratio. By slowing the flow pattern of pressure it enhances gas distribution. Volumes will vary according to lung compliance. If volumes decrease, the clinician will need to increase the pressure to maintain adequate tidal volumes. If changes in rate are needed, adjustment in inspiratory time will be required to maintain the same I:E ratio. Additionally, the pressure may also be adjusted to maintain the required tidal volumes.

Complications of Invasive Mechanical Ventilation

Airway Complications

Pre-Intubation

Airway complications begin prior to the insertion of the artificial airway into the patient. If anesthesia or heavy sedation are used apnea and respiratory depression may result, leading to hypoxia. The patient may lose protective airway reflexes which increases the risk of aspiration. The use of topical or regional anesthetics can interfere with or eliminate protective airway reflexes. Autonomic stimulation may manifest as tachycardia and hypertension (sympathetic stimulation) or bradycardia and hypotension (vagal stimulation). Either one can produce hemodynamic instability. Complications of intubation are discussed fully in the airway management section of this packet.

During Mechanical Ventilation

If the patient can make noise or sound, the ET tube has become displaced into the pharynx. The low exhaled volume alarm should sound. Stay with the patient, call for assistance and provide supplemental oxygen and/or ventilation as needed. Notify the physician and respiratory therapist to assist with possible re-intubation.

The most frequent cause of airway obstruction is secretions blocking the lumen of the ET tube. Additional causes of airway obstruction are a herniated cuff blocking the end of the ET tube, the end of the tube pressed against the wall of the trachea, kinking the tube when turning the patient, and the patient biting the ET tube. Symptoms of airway obstruction include agitation, minimal or absent chest wall movement, hypoxemia, difficulty or inability to pass a suction catheter, and/or thick and tenacious secretions. Airway obstruction produces a high-pressure alarm on the ventilator. When this occurs correct any obvious cause of obstruction immediately, such as suctioning the ET tube, and then call for assistance.

Other airway complications that may occur while the patient is intubated include nasal or oral ulcerations, infections of the sinuses and ear infections. Tracheal or laryngeal injury may result from high cuff pressure or decreased tracheal perfusion due to shock states. Tracheal or laryngeal injury may also occur if the patient accidentally self-extubates with the cuff inflated.

Post-Extubation

Complications following extubation include sore throat, stridor, hoarseness, vocal cord immobility/paralysis and aspiration. Stridor is a symptom of acute airway stenosis, and sometimes occurs due to laryngedema following extubation. Racemic epinephrine has a unique role in urgent treatment of acute laryngedema. Inhaled epinephrine is an adrenergic agonist, and causes rapid reduction in the size of blood vessels in the laryngeal area. As administration of racemic epinephrine is prepared, position the patient's head to maintain a patent airway using the jaw-thrust maneuver and administer positive pressure ventilations using a bag-valve-mask device with supplemental oxygen based on the patient's condition. Emergency resuscitation equipment must be readily available in case the patient's status deteriorates and the physician and respiratory therapist notified immediately that an emergency intubation may be necessary.

Persistent hoarseness (>12 – 24 hours) may be the result of cranial nerve damage. If this occurs, the patient may not have a safe swallow and need a swallow study. Occupational or speech therapy is frequently consulted to assist with treatment of these patients.

Intra-thoracic Complications

Pneumothorax

Intra-thoracic complications of mechanical ventilation include pneumothorax, ventilator-associated pneumonias, and ventilator-induced lung injury. Pneumothorax can result from high-pressure or high-volume ventilation causing rupture of alveoli, emphysematous blebs or suture lines. Symptoms of pneumothorax include unequal chest expansion, absent breath sounds, hypoxemia, high-pressure alarms on the ventilator, hemodynamic instability and the appearance of crepitus or subcutaneous air. If a pneumothorax is suspected, notify the physician immediately. Anticipate orders for a chest x-ray and the need for chest tube insertion.

A pneumothorax in a mechanically ventilated patient can quickly become a tension pneumothorax. Symptoms of tension pneumothorax are like those of any pneumothorax. A tension pneumothorax will also produce tracheal and mediastinal deviation away from the affected side. Tension pneumothorax is a medical emergency. Notify the physician and respiratory therapist immediately and prepare for needle thoracostomy and/or chest tube insertion.

Ventilator Associated Pneumonia (VAP)

VAP is a significant source of morbidity and mortality in the critical care area. Diagnostic criteria for VAP in adults are published by the Centers for Disease Control (CDC)/National Nosocomial Infection Surveillance (NNIS). Criteria are based upon either a clinical presentation or a radiographic result along with one other diagnostic test.

Clinical Presentation	Radiographic Result
<p>Rales or Dullness to percussion <i>and one of the following:</i></p> <ul style="list-style-type: none"> • new purulent sputum • positive blood culture • pathogen from tracheal aspirate, bronchial, brush or biopsy 	<p>CXR with new or progressive infiltrate, consolidation cavitation or pleural effusion <i>and one of the following:</i></p> <ul style="list-style-type: none"> • new purulent sputum • positive blood culture • pathogen from tracheal aspirate, bronchial, brush or biopsy • isolation of virus or viral antigen • diagnostic serology

The most invasive diagnostic test is the bronchosopic protected specimen brush (PSB). It may become the gold standard for diagnosis once the procedure, technique and interpretation have been fully validated.

The clinical pulmonary infection score (CPIS) is a tool that may aid the practitioner in determining the diagnosis of VAP. The CPIS score is based on temperature, WBC, tracheal secretions, PaO₂/FiO₂, CXR, and endotracheal aspirate culture.

Intubation bypasses the protective mechanisms of the upper airway and provides a warm, humid environment for bacterial growth in the intra-thoracic area. Signs of nosocomial pneumonia include change in the color, odor and consistency of sputum, fever and tachycardia.

CLINICAL APPLICATION VENTILATOR-ASSOCIATED PNEUMONIA

Significant amounts of research have been focused on prevention of VAP. Prevention strategies include

- The HOB elevated > 30 degrees to prevent aspiration
- Do not lavage with saline prior to suctioning
- Maintain adequate nutrition to ensure immune competence
- Minimize bacterial colonization of the oropharynx with meticulous oral care and suctioning of subepiglottic secretions
- Use duodenal or jejunal feeding tubes to prevent aspiration
- Turn patients at least once every 2 hours to mobilize secretions and reduce atelectasis

Ventilator-associated Lung Injury

Volutrauma, barotrauma and atelectrauma are all sources of ventilator-induced lung injury. Volutrauma and atelectrauma are the most dangerous of the three. These types of lung injury result in decreased surfactant production and excessive inflammation of lung tissue. At its most severe, this type of injury results in ARDS and possibly systemic inflammatory response syndrome (SIRS).

Weaning from Mechanical Ventilation

This packet will review the principles for goal-directed weaning. Specific goals are set for airway, ventilation and oxygenation. All of the goals must be met before the patient can be successfully weaned from the ventilator.

The length of time required to successfully wean a patient from mechanical ventilation is based on the individual's age, diagnosis and co-morbid conditions. The goal is to wean aggressively enough so that minimal time is spent on the ventilator, but cautiously enough so that untoward complications do not occur. It is safe to consider ventilator weaning after hemodynamic stability is achieved using minimal to moderate doses of vasoactive infusions. Successful weaning from mechanical ventilation not only requires healthy lungs but also adequate hydration, nutrition, pain control, and anxiety relief.

Indications for weaning:

- Stable BP and not receiving vasoactive IV medications
- RR >10 and < 30 breaths/min
- Temp > 36° C
- ETCO₂ 35 – 50 mm Hg (indicating adequate pH)

The initial goal is to wean the patient to minimal ventilator settings, gradually increasing the amount of work required by the patient. The evaluation of the patient's ventilation and oxygenation status are performed initially and are often done simultaneously.

Goals of Weaning:

- Minimal ventilator support
- IMV = 2
- PSV = 5
- PEEP = 5
- FiO₂ = 0.30

Evaluation of ventilation includes the monitoring of respiratory rate, spontaneous tidal volumes, ETCO₂ or PaCO₂, breath sounds and accessory muscle use. The overall goal for ventilation is to maintain a spontaneous rate between 10 and 30 bpm that is comfortable for the patient. Parameters weaned on the basis of the quality of ventilation include rate, pressure support and tidal volume.

Signs and Symptoms

This table lists signs and symptoms the patient must exhibit prior to extubation.

Adequate Ventilation	Adequate Oxygenation	Adequate Airway
Comfortable breathing pattern Spontaneous breathing of 10-30 breath/min PaCO ₂ that give acceptable pH No ventilatory muscle fatigue	SaO ₂ >0.90, SpO ₂ >0.92 PaO ₂ > 55 mm Hg FiO ₂ < 0.50 Adequate systemic CO ₂	Awake and alert Cough and gag reflex present and normal Controlled secretions Absence of stridor or laryngospasm Absence of severe bronchospasm Absence of airway distress

Assessment and evaluation of the oxygenation status includes monitoring the SpO₂ or PaO₂ and observing for signs of central cyanosis or poor tissue perfusion. FiO₂ settings should be reduced to <0.50 before aggressive weaning is begun. The ultimate goal is to decrease the FiO₂ to 0.30 before extubation, but this may not always be possible. PEEP/CPAP should be weaned as the patient's overall condition is stabilized to an end-point of +5 cm H₂O (physiologic PEEP). PEEP should be weaned in 2 – 3 cm H₂O decrements and not exceed a change of greater than 10 cm H₂O in a 24-hour period of time.

Finally, evaluation of the airway must be made prior to extubation. The patient must be able to maintain his/her airway. Airway problems that may delay extubation include inability to clear secretions, decreased level of consciousness with absent protective reflexes, and laryngeal edema.

Nursing Management of Mechanically Ventilated Patients

Being placed on a mechanical ventilator can be a terrifying experience. Although patient's respiratory distress is relieved, intubation has deprived him of his ability to move, clear his own secretions and communicate easily. Caregivers must be sensitive to the psychological impact of mechanical ventilation and anticipate their special needs. If possible, explain the procedure to the patient prior to the initiation of mechanical ventilation. Document and continually reinforce the education provided.

Communication

Encourage friends and family members to continue to talk to the patient. If the patient cannot write, use questions that can be answered with a nod or gesture to avoid frustration. Being mechanically ventilated can be a dehumanizing experience. Maintaining personal contact with the patient can lessen such feelings. Alternate communication devices such as letter boards, magic slates, magnetic slates and picture boards may be helpful.

Patient Safety

Patients who are dependent on mechanical ventilation are extremely vulnerable. Clinicians must ensure that ventilator and monitor alarms are customized to the needs of each patient. Alarm settings must be re-assessed each shift and with any significant clinical change. The alarm volumes need to be loud enough to be heard outside of the patient's room and all alarms are responded to promptly. Ventilator equipment must always be plugged into outlets supplied with emergency power. A manual resuscitation bag and oxygen source are kept at the bedside at all times and are to accompany the patient during transport.

It is often necessary to use restraints or alternative devices in order to prevent dislodgment of the ET tube despite best efforts to avoid them. Attempt to use the option with the least amount of restriction for the patient. If the patient is awake and can cooperate, do not restrain his arms so tightly that he cannot move them at all. This impairs joint mobility and increases feelings of powerlessness. Check peripheral circulation frequently and release the restraints periodically to provide range of motion and skin care. Collaborate with the interdisciplinary team to identify other therapies that could lessen the need for restraints. Provide a call light within the patient's reach as well as other means of communication (magic slate, letter board). Continually re-evaluate the need for physical restraints and adjust the treatment plan as appropriate.

Suctioning

Suctioning produces intense discomfort and anxiety for the patient. Explain to the patient that the feeling of being suctioned may feel as if it "takes the breath away" momentarily. Reassure the patient that his/her oxygenation status will be closely monitored during the procedure and precautions are taken to ensure his/her safety. Document education provided.

Suctioning should not be performed as a routine intervention. Provide suctioning when the patient requires it, based on assessment findings. Complications of suctioning include hypoxemia, cardiac arrhythmias, hemodynamic instability, trauma to the airway mucosa, atelectasis and contamination of the lower airway. Other complications that can occur are coughing, atelectasis, bronchospasm, and increased intracranial pressure. In cases of frequent bronchospasm, with a physician's order, lidocaine diluted with sterile saline may be instilled down the ET tube prior to suctioning. There is wide variability in suctioning practices among nurses and respiratory therapists. Suctioning practices have changed over the years as bedside research revealed that earlier practices caused patient harm. A prime example is the use of saline lavage with suctioning. It was once thought that saline lavage thinned secretions. Research has shown that this practice increases the incidence of nosocomial pneumonia by dispersing bacteria down the ET tube and into the lung parenchyma. Further, it is not an effective intervention to thin secretions; adequate hydration is most effective for that purpose.

Suctioning is only effective at removing secretions that have reached the level of the carina. Interventions that can be performed to facilitate effective secretion removal are:

- Frequent changes in position to facilitate movement of secretions.
- Adequate hydration to thin secretions so ciliary action can transport them up to the carina.
- Adequate nutrition to prevent respiratory muscle atrophy and strengthen the patient's cough effort. It also enhances the health of the cilia in the lungs.
- Percussion and postural drainage may facilitate drainage of secretions if it can be tolerated by the patient.

Endotracheal tube suctioning is performed as an open-circuit or closed circuit. Open circuit suctioning is not recommended for patients who are on high levels of PEEP (> 10 cm H₂O), > 0.60 FiO₂, or pressure control ventilation. Pulmonary and hemodynamic instability may result. Closed circuit suctioning is recommended for these patients. Closed circuit suctioning is also recommended for patients who require frequent suctioning or who have a respiratory infection. It has also been shown to decrease incidence of nosocomial pneumonia in intubated patients. Guidelines for suctioning for each system are provided in the following tables.

Open Circuit Suctioning Guidelines

1. Select an appropriate catheter (14 Fr, 10 Fr).
2. Prior to suctioning, hyperoxygenate the patient by administering 100% oxygen for a period of 3 – 5 minutes. Some ventilators have a "100% O₂" button that will increase the FiO₂ to 100% for a period of 3 minutes and then return to the set FiO₂. Hyperventilation with the bag-valve device (Ambu-bag) is no longer advised.
3. Prepare a sterile field for the equipment and maintain sterile technique during the procedure. If the patient is uncooperative, it may be helpful to have a second clinician in the room during suctioning.
4. Don personal protective equipment and sterile gloves. Connect the suction catheter to suction tubing keeping one hand sterile.
5. Disconnect the patient from the ventilator with your non-sterile hand.
6. Insert the suction catheter no farther than 1 cm below the ET tube using your sterile hand. DO NOT apply suction during insertion of the catheter.
7. If resistance is met, withdraw the suction catheter at least 0.5 cm before applying suction. Research has shown that catheters coming in contact with the tracheal mucosa cause significant cellular damage.
8. Damage includes: epithelial denudement, hyperemia, loss of cilia, edema fibrosis, and granuloma formation.
9. Apply intermittent suction for 5-10 second intervals while simultaneously rotating and withdrawing the catheter.
10. Re-connect the patient to the ventilator circuit. Monitor oxygen saturation and heart rate during each pass and maintain SpO₂ $> 90\%$ or at baseline.
11. Document the characteristics of any sputum obtained and the patient's tolerance of the procedure.

Closed Circuit Suctioning Guidelines

1. Prior to suctioning, hyperoxygenate the patient with 100% oxygen for 3 – 5 minutes.
2. Don appropriate personal protective equipment.
3. Advance the inline suction catheter to 1 cm below the tip of the ET tube. If resistance is met, withdraw the suction catheter at least 0.5 cm before applying suction. DO NOT apply suction during insertion of the catheter.
4. Apply intermittent suction for 5-10 second intervals while simultaneously rotating and withdrawing the catheter.
5. Flush the in-line suction catheter at the end of suctioning with sterile saline. The photo shows where the saline is attached (circled). Depress the suction button while squeezing the saline tube to ensure the saline is drawn into the catheter. Failure to apply suction at this point can result in inadvertent saline lavage of the patient.
6. Monitor oxygen saturation and heart rate during each pass and maintain SpO₂ > 90% or at baseline.
7. Document the characteristics of any sputum obtained and the patient's tolerance of the procedure.



Attach saline here.

Closed Circuit Suction Device

Pharmacology

Medications administered to facilitate mechanical ventilation include:

- Bronchodilators
- Anti-inflammatory Agents
- Analgesics
- Sedatives and hypnotics
- Haloperidol
- Neuromuscular blocking drugs

The choice of medications and their doses are made based upon the patient's disease process, history, co-morbid conditions, and physical assessment. A reference list of these medications has been provided at the end of this packet. For further information consult a current drug reference or clinical pharmacist.

Treatment of pain, anxiety and delirium is particularly important to preserve patients' mental health. Post-traumatic stress disorder (PTSD) is common in survivors of critical illness, especially those who require long-term mechanical ventilation. Provision of adequate analgesia, anxiolysis and amnesia has been shown to reduce the incidence of PTSD in survivors. Research indicates the best outcomes occur with the use of protocol-driven sedation.

Bronchodilators

Bronchodilators are used to reduce bronchoconstriction by relaxing bronchial smooth muscle. They are helpful for patients with bronchitis, COPD or asthma. Bronchodilators are usually administered via in-line nebulizers or metered dose inhalers. They are available as short-acting and long-acting preparations. Significant side effects of bronchodilators are tachycardia and nervousness.

Anti-inflammatory Agents

Anti-inflammatory medications are used in pulmonary diseases that have an inflammatory component, such as asthma. Steroids are a common anti-inflammatory medication used for mechanically ventilated patient. Steroids may be administered locally as metered-dose inhalers or systemically via IV or NG routes. Significant side effects of systemic steroids are immunosuppression, hyperglycemia, and adrenal insufficiency. Locally administered steroids have minimal side effects.

Analgesics

Analgesics are used for control of pain. Morphine is the preferred agent, but Fentanyl and hydromorphone are acceptable alternatives. Pain control is an essential component of care for mechanically ventilated patients. Acute pain (especially abdominal or thoracic pain) interferes with breathing patterns. Patients in pain tend to breathe rapidly and shallowly, and may splint. These behaviors can interfere with delivery of adequate tidal volume. If the patient is receiving A/C ventilation, tachypnea may result in respiratory alkalosis. Agitation due to pain increases oxygen demand and may be poorly tolerated by patients with limited respiratory reserve. Chronic pain must also be controlled so the patient is capable of participating fully in the treatment plan.

Assessing pain in mechanically ventilated patients is challenging. Use of a visual analog scale may prove helpful. If the patient cannot communicate at all, the clinician must rely on physical and behavioral cues such as writhing, wincing, grimacing, tachycardia, and hypertension. Pain control should be addressed before other types of sedation are considered.

Sedatives

The appropriate use of sedatives is a debated aspect of patient care. Generally, the best sedative is one that relieves anxiety and/or pain, yet does not totally depress spontaneous respiratory effort, impair hemodynamics or cause confusion. The most commonly used sedatives for mechanically ventilated patients are benzodiazepines (midazolam, lorazepam) and Propofol (Diprivan).

Benzodiazepines are the drugs of choice. The benzodiazepines produce amnesia, hypnosis and anxiolysis. Propofol does not produce amnesia and should be utilized in conjunction with a drug that has this effect. Current evidence-based recommendations are: Propofol or midazolam for patients who require sedation for periods of less than 24 hours; for longer periods, lorazepam is preferred.

Titration of medication dose to the patient's response is the most prudent method. Sedatives should always be used in conjunction with analgesics. Sedatives themselves do not relieve pain and use of sedatives without analgesia actually intensifies the pain experienced by the patient. These guidelines typically incorporate the use of a sedation scale. Sedation scales allow objective assessment of the depth of sedation and, when used appropriately, may help prevent over-sedation. Over-sedation may prolong the duration of mechanical ventilation and length-of-stay.

Haloperidol

Haloperidol (Haldol) is the drug of choice for treatment of delirium in mechanically ventilated patients. Signs of delirium include disorientation, altered perception, agitation, psychomotor abnormalities, decreased attention, disorganized thinking, and disturbed sleep-wake cycles. Significant side effects of haloperidol are QT prolongation and decreased seizure threshold.

Neuromuscular Blocking Drugs (NMBD)

If the patient has very unstable respiratory status and is "fighting" or "bucking" the ventilator, neuromuscular paralysis with agents such as Pavulon (pancuronium bromide), or Nimbex (cisatracurium) may be indicated. Chemical paralysis may also be used when the patient's capacity for tissue oxygenation cannot support muscle activity.

Indications for use of NMBDs

Facilitate mechanical ventilation

- Patient agitated or combative despite adequate sedation and analgesia
- Danger of self-extubation
- Elimination of ventilator discoordination
- Decrease the work of breathing
- High PEEP or Inverse I:E ratios

Decrease oxygen consumption

- Reduce oxygen deficit in compromised patients with pulmonary disease

Maximal use of sedation (amnesia, hypnosis, anxiolysis) and analgesia (opioids) is essential prior to utilization of neuromuscular drugs. NMBDs themselves have no effect on anxiety or pain. A patient who is chemically paralyzed without analgesia or sedation can feel and hear everything, but cannot move or communicate. Patients who live through such experiences can suffer extreme psychological damage that may persist for years.

NMBDs paralyze the muscles at the level of the neuromuscular junction. The reduced muscle activity decreases tissue metabolism and oxygen demand. Continuous infusions are preferred to bolus dosing.

NMBDs

Long-Acting Agents		Short-Acting Agents	
Drug	Elimination	Drug	Elimination
Atracurium (Tracrium)	Hofmann degradation*	Mivacurium (Mivacron)	Hydrolysis by plasma esterases
Cisatracurium (Nimbex)	Hofmann degradation	Rapacuronium (Raplon)	Kidneys and Liver
Doxacurium (Nuromax)	Kidneys and Liver	Rocuronium (Zemuron)	Kidneys and Liver
Pancuronium (Pavulon)	Kidneys and Liver	Succinylcholine (Anectine)	Kidneys and Liver
Pipecuronium (Arduan)	Kidneys and Liver		
Tubocurarine (Tubarine)	Kidneys and Liver		
Vecuronium (Norcuron)	Kidneys and Liver		

* Hofmann degradation is an enzymatic process that occurs when there is a change in the pH of the medication.

Nursing Care for NMBD

Specific nursing care is required for patients receiving NMBDs. The nurse must monitor the patient’s Train of Four (TOF) responses using a peripheral nerve stimulator (PNS) **PRIOR** to the start of the infusion, with changes in dosage, and as ordered.

During infusion the patient’s airway must be protected and adequate ventilation maintained. Ventilator alarms are set to detect the slightest change in pressure. All alarms must be attended to immediately because disconnection from the ventilator can be fatal when the patient is chemically paralyzed. A sign is placed on the patients outside door notifying all staff that the patient is receiving NMBDs. Turn the patient every two hours to prevent pressure decubitus and provide pressure-reducing devices on the patient’s bed. Provide passive range of motions to maintain mobility of joints. Eye lubrication (ophthalmic drops or ointment) can be administered to prevent corneal ulcerations. Ophthalmic drops should be used if the patient has the potential to be a corneal donor. Monitor appropriate laboratory values (LFT’s, BUN, Creatine and Creatine clearance). Collaborate with the physician regarding the need to hold NMBDs on a daily basis in order to assess the patient’s baseline status and identify changes.

Train of Four (TOF) Procedure

INITIAL PROCEDURE (PRIOR TO ADMINISTRATION OF NMBD's)

1. Place stimulator over target nerve (ulnar, facial, or posterior tibial).
2. Negative electrode applied towards target site (Red to Black 2 – 3 inches apart).
3. Depress “TOF” key (4 stimuli at 0.5 second intervals indicated by 4 flashing red light signals).
4. Do NOT remove until light stops flashing.
5. Increase mA until target site responds with 4 equal twitches.
6. Note mA at this point.
7. Increase current in 10mA increments until no further increase in intensity of response. This is known as the Supramaximal Stimulation (SMS) Point.
8. SMS is the ONLY setting used hereafter to monitor the twitch response.
9. Administer the NMBD.
10. Monitor the number of twitches in response to a TOF stimulation indicating the percentage of blockage.
11. Preferred level of blockade for optimal patient management is 80 – 90 %.



Train of Four Device

# of Twitches	Extent of Blockade	Clinical Interpretation
4 out of 4 (4/4)	Less than 75%	Spontaneous recovery
3 out of 4 (3/4)	75 – 80%	Maintenance doses may be needed to extend the duration of muscle relaxation
2 out of 4 (2/4)	80 – 90%	Adequate for short term relaxation and long-term mechanical ventilation
1 out of 4 (1/4)	90%	Conditions suitable for endotracheal intubation and long-term mechanical ventilation
0 out of 4 (0/4)	100%	Considered TOO HIGH a level of blockade. Turn off NMBA until 1/4 or 2/4 responses are obtained

- **Reversal Agent for Non-Depolarizing NMBD:**

The dose for neostigmine (Prostigmin) is 0.5-2 mg IV and repeated PRN. In most cases less than 5 mg is needed. The administration of Atropine sulfate 0.6-1.2 mg IV several minutes prior to the neostigmine injection is recommended. The only depolarizing NMBD is succinylcholine, which has no reversal agent. Succinylcholine is not indicated for maintenance neuromuscular blockade, but is used for anesthesia.

Prone Positioning

Anatomically, 60% of the lungs are dependent in the supine position compared to 40% in the prone position. Fluid moves by gravity into dependent lung fields. This means that in the supine position most of the lung has a greater proportion of fluid than gas in it. The weight of this fluid has a tendency to produce atelectasis and limits the number of alveoli available for gas exchange. When the patient is placed in the prone position more alveoli are exposed. Placed in a prone position facilitates an even distribution of tidal volume by decreasing chest wall compliance and increasing the movement of secretions.

There are several contraindications for placing a patient in the prone position: spinal cord instability, hemodynamic instability, cardiac rhythm disturbances, and recent thoracic or abdominal surgery. Additionally, there are specific clinical concerns that must be addressed by the multidisciplinary team. These concerns include the ability to gain access to wounds and drains, handle emergency procedures, and communicate with the patient. These concerns must be weighed against the potential benefit that proning may provide.

It is not easy to place a critically ill patient in the prone position. When turning the patient, obtain proning devices and adequate personnel to assist. Take special care not to dislodge any tubes or drains. Monitor the patient closely for changes in oxygenation and hemodynamic status during and immediately after the process.

Monitoring the Patient and the Equipment

Monitoring of a patient on ventilatory support integrates a total patient assessment with checks of the equipment utilized to support the pulmonary system. Mechanical ventilators will only deliver oxygenated gas to the alveoli. The oxygen must still diffuse across the alveolar-capillary membrane and be transported to the tissues. Monitoring of tissue perfusion is an essential component of the pulmonary assessment. The cardiac and pulmonary systems are intimately interconnected, and therapies aimed at the cardiovascular system are often vitally important to pulmonary treatment.

Nursing Responsibilities:
Provide patient with means of communication (alphabet board or paper)
Explain all nursing procedures to patient and discuss plan of care with patient
Daily check of Chest X-ray for ET tube placement (proper position 2 cm above carina)
Routinely monitor ventilator settings
Monitor pressure readings and breath sounds
Monitor patient for respiratory muscle fatigue, accessory muscle usage
Monitor oxygenation – SaO ₂ , ETCO ₂ (ABG – if needed) and patient's subjective information (if able)
Perform suctioning, based on adventitious breath sounds maintaining sterile technique
Perform chest physiotherapy, as appropriate (stop NG feedings 30 – 60 minutes before chest PT)
Perform oral care – cleaning mouth and teeth with toothpaste and toothbrush every 2 – 4 hours; suction oropharyngeal secretions
Monitor for Adverse effects of mechanical ventilation–infection, barotrauma, decreased cardiac output
Provide sedatives, narcotics analgesics as appropriate or alternative methods (audio, video or TV)

Position patient to facilitate ventilation/perfusion (good lung down) as appropriate
Monitor fluid and nutritional status and support as needed
Utilize a multidisciplinary approach for ventilatory management– Initially - Respiratory therapist (RT), RN, MD and patient. Later - Social worker (SW), physical therapist (PT), Speech therapist and Occupational therapist (OT).

Equipment checks are performed at regular intervals to ensure patient safety and delivery of the prescribed therapy. Both nurses and respiratory therapists can perform equipment checks. The table below lists essential components of ventilator equipment checks.

Equipment Check

Ventilator tubing is free of water
Ventilator tubing is kink free
Ventilator tubing “free” i.e. not pulling on ETT, not caught in side-rails
Connections are secure
Verify that ventilator alarms are on and set appropriately for the patient. (with NMBD’s an external audible alarm may be indicated)
Monitor pressure readings [inspiratory force, peak inspiratory pressure (PIP) tidal volume, minute ventilation, airway pressure, respiratory rate (total – patient and machine breaths) and FiO_2].
Monitor effectiveness of ventilator changes (if any) associating them with the patients response

Troubleshooting the Ventilator

Anytime that the ventilator is malfunctioning and cannot be corrected immediately, manually ventilate the patient!! One person needs to manually ventilate the patient, while another troubleshoots the ventilator. The bag-valve-mask device must be readily available.

The goal of the ventilator alarm system is to warn of events. The alarms are individualized for each patient. Events are categorized into mechanical/technical and patient generated events. They are categorized according to levels of priority.

Level 1 - Ventilator malfunction (life threatening)

- No gas delivery to patient
- Excessive gas delivery to patient
- Exhalation valve failure
- Loss of electric power

Level 2 - Ventilator malfunction (not immediately life threatening)

- Blender failure
- Loss of PEEP or excessive PEEP
- Autocycling
- Circuit leak
- Circuit partially occluded
- Inappropriate I:E ration
- Inappropriate heater/humidifier function

Level 3 – Patient event affecting ventilator-patient interface

- Change in ventilatory drive (CNS, peripheral nerves or muscle function)
- Change in compliance/resistance (air trapping, barotrauma)
- Auto PEEP

Level 4 – Patient event not affecting ventilator-patient interface

- Change in gas exchange (Capnograph, oximeter,)
- Change in respiratory system impedance
- Change in muscle function
- Change in cardiovascular function

Ventilator Alarms

Alarms bolded are Life threatening (Level 1) and need to be addressed immediately		
Ventilator Alarms	Cause	Action to be taken
Ventilator inoperative	Ventilator Failure	Manually ventilate patient and have someone else troubleshoot the ventilator
Low Pressure Alarm <ul style="list-style-type: none"> • Low PEEP/CPAP Alarm • Low Exhaled Volume 	Patient is losing some or all of his tidal volume	Is the patient disconnected? Is the tubing disconnected? Is airflow adequate? Is there an ETT cuff leak? Manually ventilate patient until cause found!
Apnea Alarm	No spontaneous breath taken in a preset number of seconds	Most often seen at low IMV's. Encourage patient to breathe or give patient a single breath. Consider whether rate should be increased.
Pressure Limit Alarm	<ul style="list-style-type: none"> • Patient's PIP reached preset limit • Tidal volume dumped when limit reached • Patient has obstruction in airway 	<ul style="list-style-type: none"> • Are there increased secretions? • Does the patient need to be suctioned? • Is patient biting on the ETT? • Has water accumulated in the ventilator circuits? • Has the patient's compliance decreased, i.e. bronchoconstriction or ARDS? • Is the patient asynchronous with the ventilator (Bucking the vent)? • Treat the problem do not increase the pressure limit because of risks of high PIPs to your patient
Decreased Minute or Tidal Volume	<ul style="list-style-type: none"> • Leak around endotracheal tube, from the system, or through the chest tube • Decreased patient-triggered respiratory rate • Decreased lung compliance • Airway secretions • Altered settings • Sensor malfunction 	<ul style="list-style-type: none"> • Check all connections for leaks • Check respiratory rate • Evaluate patient • Clear airway secretions • Check patient-ventilator system • Change sensor

Increased minute or Tidal Volume	<ul style="list-style-type: none"> • Increase patient-triggered respiratory rate • Altered settings • Hypoxia • Increased lung compliance • Sensor malfunction 	<ul style="list-style-type: none"> • Check respiratory rate • Check patient-ventilator system • Evaluate patient; ETCO₂, SaPO₂, consider obtaining an ABG • Patient improvement (+lung compliance) • Change sensor
Change in Respiratory rate	<ul style="list-style-type: none"> • Altered setting • Increased metabolic demand • Hypoxia • Hypercarbia 	<ul style="list-style-type: none"> • Check patient –ventilator system • Evaluate patient: ETCO₂, SaPO₂, consider obtaining an ABG
Sudden increase in maximal inspiratory pressure	<ul style="list-style-type: none"> • Coughing • Airway secretions or plugs • Ventilator tubing kinked or filled with water • Kinked endotracheal tube • Changes in patient position • ETT in right main stem bronchus • Patient-ventilator asynchrony • Bronchospasm • Pneumothorax 	<ul style="list-style-type: none"> • Alleviate uncontrolled coughing • Clear airway secretions • Check for kinks and water • Check for kinks • Consider repositioning • Verify position • Correct asynchrony • Identify cause and treat • Decompress chest
Gradual increase in maximal pressure	<ul style="list-style-type: none"> • Increased lung stiffness • Diffuse obstructive process 	<ul style="list-style-type: none"> • Measure static pressure • Evaluate for reversible problems: Atelectasis, increased lung water, bronchospasm
Sudden decrease in maximal inspiratory pressure	<ul style="list-style-type: none"> • Volume loss from leaks in system • Clearing of secretions; relief of bronchospasm; increasing compliance 	<ul style="list-style-type: none"> • Check patient-ventilator system for leaks • Positive information
FIO ₂ Drift	<ul style="list-style-type: none"> • O₂ analyzer error • Blender piping failure • O₂ source failure • O₂ reservoir leak 	<ul style="list-style-type: none"> • Calibrate analyzer • Correct failure • Check ventilator reservoir

I:E Ration < 1:3 or > 1:1.5	<ul style="list-style-type: none"> • Altered inspiratory flow rate • Alteration in other settings that control I:E ration • Alteration in sensitivity setting • Airway secretions (pressure ventilator) • Subtle leaks 	<ul style="list-style-type: none"> • Check IFR setting and correct • Check settings and correct • Clear airway of secretions • Measure minute ventilation
Inspired gas temperature inappropriate	<ul style="list-style-type: none"> • Altered settings • Thermostat failure 	<ul style="list-style-type: none"> • Correct temperature control setting • Replace heater (or ventilator)
Changes in delivered PEEP	If ventilator control used: <ul style="list-style-type: none"> • Changes in compliance • Changes in tidal volume 	Correct problem if possible; If not: <ul style="list-style-type: none"> • Increase PEEP setting to deliver desired level of PEEP
Changes in static pressure	<ul style="list-style-type: none"> • Changes in lung compliance 	<ul style="list-style-type: none"> • Evaluate patient and correct if possible
Changes in Inspiratory flow rate, sigh volume, assist or control mode, alarm status, deadspace volume	<ul style="list-style-type: none"> • Changes in these settings resulting from deliberate or accidental adjustment of dials or knobs 	<ul style="list-style-type: none"> • Check to determine whether current settings are the ones intended

Troubleshooting Patient Problems

Solving any patient problem related to mechanical ventilation starts with the ABC's; airway, breathing and circulation.

Hypoxemia

Mechanically ventilated patients who present with hypoxemia need improved oxygenation. First, make sure the airway is patent, then verify that the prescribed therapy is administered appropriately. Then consider increasing the FiO₂ or PEEP to improve oxygenation. Other interventions that may improve oxygenation are repositioning or prone positioning.

Altered CO₂ Levels

When the patient presents with elevated or depressed levels of CO₂, consider ventilation. The ventilator settings that affect ventilation are rate, tidal volume and pressure support.

Conditions that may produce hypercapnia include hypoventilation, fever, shivering, seizures, and excess carbohydrates. Hypocapnia is most commonly caused by hyperventilation, especially in patients on A/C modes. Hyperventilation is associated with metabolic acidosis, pain, anxiety, and some neurologic injuries.

Abnormal Ventilator Patterns

Abnormal patterns from ventilatory support modes include trigger problems, flow dys-synchrony, cycle dys-synchrony, alveolar over distension, alveolar under-recruitment, air trapping and circuit leaks. Trigger problems are related to the ventilator sensitivity settings and variability in the patient's inspiratory effort.

Airflow obstructions (secretions, kinked ET tube) can inhibit inspiratory flow due to increased pressure and cause the ventilator not to deliver a scheduled breath.

Synchronous interaction means that the ventilator is sensitive to the patient's initiation and termination of ventilatory effort and is responsive to the flow characteristics of the patient's ventilatory demand. Synchrony improves patient comfort. Dys-synchrony occurs when ventilator gas delivery and patient efforts are not coordinated or are out of cycle and causes the patient to "buck the vent". Cycle dys-synchrony occurs when the ventilator breaths are too short in duration leaving the patient air hungry and demanding more flow. Insufficient peak flow is a commonly unrecognized cause of patient dys-synchrony.

To assess for alveolar over-distension, monitor the plateau pressure during an inspiratory hold or pause. Values greater than 35 cm H₂O may indicate excessive regional distension. Alveolar over-distension increases the risk of volutrauma. Alveolar under-recruitment may cause problems with oxygenation. To determine alveolar under-recruitment, the pressure-volume graphs are monitored at the end of a full inspiration and lung compliance is calculated. Collapsed alveoli will cause a decrease in compliance of the lung. PEEP is utilized to recruit collapsed alveoli.

Air trapping (auto-PEEP) occurs when the patient has not fully exhaled and the next machine breath is given. This increases the peak and plateau pressures. Auto-PEEP can be detected by checking the pressure during an expiratory hold.

Summary

Mechanical ventilation is a complex supportive therapy for patients with respiratory failure. Optimal outcomes for these patients are achieved through the skilled delivery of care by a multi-disciplinary team. Essential components of care include management of the airway and ventilator, physical care, emotional support, pain and anxiety control, and prevention of complications.

This packet has introduced the fundamental components of mechanical ventilation. A reference list is provided for additional education. Policies for individual institutions must be consulted prior to any intervention.

Glossary

Acute Respiratory Distress Syndrome (ARDS): a severe pulmonary inflammatory response to a variety of insults, resulting in capillary leak, interstitial edema, intra-alveolar hemorrhage and exudate, decreased ventilation-perfusion matching, and progressive hypoxemic respiratory failure.

Airway pressure: pressure in the airways of the lung, often assumed to be identical to ventilator circuit problems.

Aspiration: the introduction of oral, nasal, pharyngeal, or gastric contents into the lung.

Barotrauma/volutrauma: injury to the lung due to excessive pressure and/or volume in the lung.

Chronic Obstruction Pulmonary Disease (COPD): a disease process involving chronic inflammation of the airways. Variants include chronic bronchitis (disease predominance in the large airways) and emphysema (disease predominance in the smaller airways and alveolar regions).

Colorimetric CO₂ detector: A device which detects the presence of carbon dioxide in expired gas and indicates the presence of CO₂ by changing color (usually yellow to purple).

Constant positive airway pressure (CPAP): a therapeutic modality that maintains a constant transrespiratory pressure. CPAP is not a ventilatory mode because it does not generate a tidal volume.

Cricoid pressure: pushing down on the cricoid membrane, thereby collapsing the esophagus against the cervical vertebrae. Cricoid pressure has been shown to prevent gastric insufflation during mask ventilation.

Dual control: modes of ventilation whereby two or more variables may control breath delivery depending upon certain circumstances.

End-Expiratory Pressure (EEP): the baseline transrespiratory pressure that exists at the end of the expiratory time. This pressure is often positive (PEEP).

Endotracheal tube: An artificial airway passed through the nose or mouth past the vocal cords and into the trachea.

Expiratory flow time: the time during which expiratory flow occurs.

Flow: rate of gas delivery in and out of the lung.

Flow synchrony: matching of ventilator flow delivery to patient efforts during interactive breaths.

High frequency ventilation (HFV): ventilatory support characterized by frequencies greater than physiologic breaths per minute.

Hygroscopic heat and moisture exchanger filter: a passive humidifier in which both physical and chemical means of heat and moisture exchange are used; it incorporates a breathing circuit filter.

Indirect calorimetry: a technique that measures oxygen consumption and CO₂ production to predict nutritional needs and quantify metabolic activity.

Inspiratory phase (inspiration): the part of the ventilatory cycle from the beginning of inspiratory flow to the beginning of expiratory flow. Any inspiratory pause is included in the inspiratory phase.

Laryngeal mask airway (LMA): a device used to direct gas delivery into the larynx without an endotracheal tube.

Laryngoscope: device designed to permit visualization of the larynx and airways through the mouth.

Mandatory breath: a mechanical breath that is initiated and terminated by the ventilator rather than by the patient's ventilatory drive.

Mean airway pressure: the average pressure that exists at the airway opening over the ventilatory period. It is usually measured as gauge pressure.

Mechanical ventilation: the technique of providing by means of a machine either some or all of the work of breathing for a patient.

Minimal seal technique: the technique for maintaining the endotracheal tube cuff in which a volume of gas is used in the cuff sufficient to prevent a leak at end-inspiration.

Minute ventilation (MV): the total amount of gas moving in or out of the lungs during 1 minute.

Noninvasive ventilation: techniques of assisting or controlling ventilation using devices that do not require endotracheal tube placement.

Obstructive lung disease: disease characterized by airway narrowing.

Oscillators: a technique to deliver HFV utilizing oscillating piston or membrane.

Overdistension: the process of providing excessive volume to lung regions, thereby causing a "stretch" injury.

Parenchymal lung injury: lung injury resulting from processes affecting the alveolar capillary interface, the interstitium, or the vasculature.

Partial ventilatory support: mechanical ventilatory support in which the patient and the ventilator share the ventilatory load.

PEEP: acronym for positive end-expiratory pressure.

Pleural pressure: pressure inside the pleural space (between the lungs and chest wall) often reflected as esophageal pressure.

Pneumonia: Infection in lung parenchyma.

Positive pressure ventilation: use of positive airway pressure to support ventilation.

Postextubation stridor: the sound that occurs in an extubated patient from flow through an upper airway narrowed by inflammation from an endotracheal tube.

Pressure gradients: the difference in pressure across a resistance or a compliance structure.

Pulmonary artery hypertension: high pressures within the pulmonary vasculature. This is usually caused by an increase in pulmonary vascular resistance secondary to lung disease, and/or hypoxia.

Resistance: impedance to flow in a tube or conduit; quantified as ratio of the difference in pressure between the two points along a tube length divided by the volumetric flow of the fluid per unit time.

Respiratory distress syndrome (RDS): a result of surfactant deficiency and/or a pulmonary insult in the neonatal period, this condition is marked by tachypnea, hypoxemia, decreased pulmonary compliance, and alveolar collapse.

Respiratory insufficiency: the inability of the body to provide adequate arterial oxygenation.

Sellick maneuver: technique of providing cricoid pressure named for its inventor.

Sensitivity: a measure of the amount of effort that must be generated by a patient to trigger a mechanical ventilator into the inspiratory phase; alternatively, the mechanism used to set or control this level.

Shunting: pulmonary capillary blood completely bypassing ventilated alveoli.

Spontaneous breath: breath that is both patient initiated and patient terminated.

Total ventilatory support: mechanical ventilatory support supplying total unloading of a patient's ventilatory muscles.

Trigger: to initiate the inspiratory phase of an assisted breath.

Ventilation distribution: the description of how the tidal volume is distributed to the millions of alveolar units.

Ventilation/perfusion (V/Q) relationships: quantification of the relationship of ventilation to perfusion in alveolar capillary units. This is normally 1. Very high V/Q units are effectively dead space. V/Q units of 0 are shunts.

Weaning: gradual reduction in partial ventilatory support.

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Internet Resources

Ventilator Information

www.ventworld.com

www.viasyhealthcare.com

Ventilator Products

www.posey.com