Medical Education Systems, Inc.

Mechanical Ventilation

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Learning Objectives

Upon successful completion of this continuing education module, you will be able to:

- Identify and explain all the abbreviations associated with mechanical ventilation
- Define and identify the various modes of “Mechanical Ventilation”
- Identify the objectives of mechanical exposure to air
- Identify the clinical indications and recommendations for mechanical ventilation
- List and discuss the potential complications associated with mechanical ventilation
- Explain the “how” and “when” of discontinuing mechanical ventilation

Mechanical Ventilation in a Nutshell

Before going into the history and in-depth analysis of mechanical ventilation, we present you here with a brief synopsis/overview of the topic:

Some Basics

What Is Respiration?

The primary function of the lungs and chest wall (the "respiratory system") is to exchange oxygen and carbon dioxide between the atmosphere and the body. Every minute, an adult breathes in about 5 liters of air, containing 21% oxygen. From this inspired air, roughly 250 milliliters of oxygen diffuses from the lungs' tiny air sacs (alveoli), through thin-walled lung capillaries, and binds to hemoglobin, the oxygen-carrying protein of the red blood cells. The blood, heart, and circulation carry this oxygen to the organs and cells of the body where it is consumed in energy-producing biochemical reactions. At the same time, these cells produce carbon dioxide in proportion to the oxygen used (about 200 milliliters of carbon dioxide is produced for every 250 milliliters of oxygen consumed each minute). In a process that mirrors oxygen exchange, carbon dioxide is carried in the circulation back to the lungs, diffuses from blood into the alveoli, and is exhaled into the atmosphere. This cyclic transport of oxygen and carbon dioxide between body and atmosphere is termed "respiration."

Respiratory Failure

The adequacy of respiration can be judged by measuring the levels of oxygen and carbon dioxide in the body. In one approach, blood taken from an artery is injected into a "blood gas analyzer" which reports the partial pressures of oxygen (normally 100mmHg) and carbon dioxide (normally 40mmHg). Alternatively, a non-invasive "pulse oximeter," often applied to the fingertip, can estimate how much oxygen is bound to the blood's hemoglobin (normally 97-100% of the hemoglobin is saturated with oxygen). Because the process of respiration is complex, it is prone to breaking down when disease affects the lung, airways, chest wall, or its neurological control mechanisms. The various ways in which the exchange of oxygen and carbon dioxide falters are termed "respiratory failure." Respiratory failure may be signaled by a fall in the body's level of oxygen, a rise in carbon dioxide, or both. Symptoms may include shortness of breath, anxiety, rapid or noisy breathing, cyanosis (blue-tinged skin), sweating, and a racing heart. Significant changes in blood oxygen or carbon dioxide can provoke damage to cells or organs, even causing death. Respiratory failure, therefore, represents a medical emergency. Health care professionals treat respiratory failure by restoring normal function, assisting crippled function, or using devices such as mechanical ventilators to sustain gas exchange until the respiratory system heals.

Hypoxemic respiratory failure: Blood values of oxygen may fall critically (hypoxemia means an abnormally low pressure of oxygen in the blood) when alveoli collapse or become filled with fluid, inflammatory cells, or blood. For example, in patients with severe pneumonia, many alveoli fill with pus, preventing oxygen from reaching the lung capillaries. An even more common problem is "acute lung injury," in which many different insults (such as severe trauma, infection, or aspiration of stomach acid into the lungs) cause the lung blood vessels to leak, flooding alveoli, and preventing the transfer of oxygen. The most severe cases of this form of hypoxemic respiratory failure constitute the "acute respiratory distress syndrome" or ARDS. Even when lung blood vessels are not abnormally leaky, fluid from the bloodstream can still make its way into the alveoli if the pressure in the blood vessels rises abnormally. This is typically seen in heart failure due to coronary artery disease, valve abnormalities, or cardiomyopathy. These different forms of hypoxemic respiratory failure can advance rapidly, turning a normally delicate lung into a swollen organ incapable of supporting life, all in a matter of minutes to hours.

Hypoxemic Respiratory Failure: Mechanisms and Causes

Mechanism	Cause
Pus-filled alveoli	Pneumonia
Blood-filled alveoli	Alveolar hemorrhage
Fluid-filled alveoli	Lung edema due to heart failure
Acute lung injury (fluid and collapse) and ARDS	Severe bloodstream infection Inhalation of stomach acid Trauma Multiple blood transfusions Inflammation of the pancreas Inhaled toxins Ventilator-induced lung injury

Hypercapnic respiratory failure: A second form of respiratory failure manifests as abnormally elevated carbon dioxide (hypercapnia means abnormally elevated carbon dioxide pressure in the blood). The respiratory system's capacity to exchange carbon dioxide (even more than its ability to exchange oxygen) depends on proper, rhythmic movement of air in and out of the lungs. Normally, the brain paces the rhythm of breathing (called "central drive"), the spinal cord and nerves carry these signals to the respiratory muscles ("neuromuscular function"), and the respiratory muscles expand the chest wall, drawing air through the air passages and stretching the lung. A breakdown in any of these areas can precipitate hypercapnic respiratory failure. Narcotic overdose, for example, blunts or abolishes central drive, leading to respiratory failure. While such an overdose can be fatal, it can also be readily treated, by infusing a drug to antagonize the impact of the narcotic. More catastrophic insults to the brain (head injury, brain swelling, massive stroke) may necessitate mechanical ventilation, discussed below. Neuromuscular function can also be threatened by conditions that block nerve function (drugs, toxins, amyotrophic lateral sclerosis) or muscular contraction (muscular dystrophy, blood electrolyte disturbances, curare-like medications and toxins).

Finally, the mechanical properties of the lung, airways, or chest wall may become deranged such that even with increased drive and strong respiratory muscles, sufficient air simply cannot be moved in and out of the lungs. An example of this is a severe asthma attack, called status asthmaticus, which so narrows and plugs the airways that death may follow. A long list of diseases, including emphysema, cystic fibrosis, severe kyphoscoliosis, and idiopathic pulmonary fibrosis, also challenge the respiratory muscles by making them work harder just to move air. All of these can lead to hypercapnic respiratory failure.

Hypercapnic Respiratory Failure: Mechanisms and Causes

Mechanism	Cause
Impaired drive	Narcotics
	Brain injury
	Sleep apnea syndrome
	Obesity-hypoventilation
	Ondine's curse
Neuromuscular disease	Spinal cord injury
	Amyotrophic lateral sclerosis
	Myasthenia gravis
	Curare and curare-like drugs
	Nerve toxins
	Muscular dystrophy
Mechanical derangement of the lungs or chest wall	Idiopathic pulmonary fibrosis
	Kyphoscoliosis
	Status asthmaticus
	Exacerbation of emphysema or chronic bronchitis

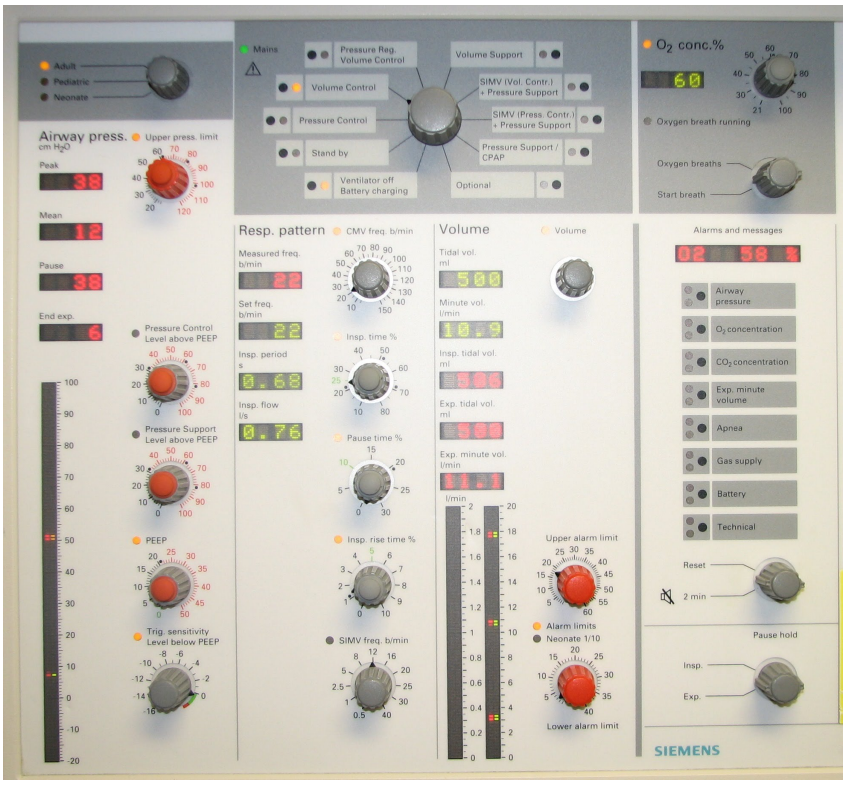
Respiratory Failure: Acute or Chronic?

In most instances, respiratory failure is an acute, dramatic, and catastrophic illness prompting acute hospitalization, typically in an intensive care unit. An example is the severe acute respiratory syndrome (SARS), an infection of the lung by a coronavirus that causes ARDS and hypoxemia, often leading to death. In other patients, however, respiratory failure is more insidious, progressing or persisting over months or years. Generally, these patients have hypercapnic respiratory failure, characterized by chronically elevated carbon dioxide levels (hypoxemic respiratory failure is almost solely an acute illness). Any of the three bases for hypercapnia can underlie chronic respiratory failure. Abnormalities of central drive include a rare condition affecting babies, called Ondine's Curse, in which the normally automatic pacemaking function of the brain is lacking, due to a genetic error in the development of certain brain cells. Damage to this area of the brain through stroke or brain trauma can produce a similar condition in adults. In either instance, breathing requires voluntary effort and fails during sleep.

These patients usually require mechanical ventilation or they die. Much more commonly, drive may be impaired in the morbidly obese, due to the sleep apnea syndrome or the obesity-hypoventilation syndrome (the basis of which is poorly understood). Some neuromuscular diseases are accompanied by chronic hypercapnic respiratory failure. Amyotrophic lateral sclerosis (Lou Gehrig's disease) involves the nerve pathways leading to the breathing muscles and it is this feature of the disease that is ultimately fatal in most victims. Finally, numerous diseases raise the work of breathing by stiffening the lung or chest wall (idiopathic pulmonary fibrosis, severe kyphoscoliosis), by occluding the breathing passages (chronic bronchitis), or interfering with the capacity of the lung to empty during expiration (emphysema). Oftentimes, patients with chronic respiratory failure can be supported for months or years with medications (such as drugs to dilate the main airways), or partial ventilatory support (such as with nighttime mask ventilation, discussed below).

How Is Respiratory Failure Treated?

Bringing the oxygen and carbon dioxide levels back into acceptable ranges involves monitoring the condition (arterial blood gas analysis, pulse oximetry); treating the underlying pathology; or supplementing respiratory function, generally with oxygen or mechanical ventilation. For hypoxemic respiratory failure, oxygen is useful, but is often only partially effective. The same process of collapse or flooding of alveoli that prevents air from reaching the lung capillaries also blocks the exchange of pure oxygen. Nevertheless, oxygen is a mainstay of treatment, used to enrich the inspired air at the nose or mouth through small tubes or a mask. If oxygen therapy fails to restore blood oxygen levels and the underlying cause of hypoxemia cannot be quickly treated, some form of mechanical assistance is generally needed.



Mechanical Ventilation: The bellows function of the respiratory system can be assisted or replaced by a machine, called a mechanical ventilator. These devices are connected to the patient by a tube inserted into the main windpipe ("endotracheal tube"), a tracheostomy, or a tight-fitting mask ("non-invasive ventilation"). The ventilator raises its pressure, pushing air into the lungs, then lowers the pressure again, allowing exhalation. Modern ventilators are controlled by highly sophisticated microprocessors, allowing remarkable control of how the lungs are inflated (see Figures).

When the problem is hypoxemia, the ventilator can be set to deliver up to 100% oxygen. The fact that the ventilator and endotracheal tube provide a direct and closed path to the patient means that 100% oxygen can be delivered to the lung. Such high concentrations of oxygen cannot be achieved easily using typical oxygen masks. Even oxygen can be harmful and in high concentrations tends to injure the lung, causing the capillaries to leak, and risking flooding of the alveoli with edema. A complementary means to raise oxygen levels uses pressure during expiration to prevent the lungs from exhaling completely (called “positive end-expiratory pressure” or PEEP). PEEP has the effect of holding open collapsed alveoli and redistributing fluid out of alveoli and into other areas of the lung where it interferes less with oxygen exchange. PEEP also may help to protect the diseased lung from further injury. At very high levels, PEEP can enlarge the lung enough to risk its rupture (this is very uncommon) or to compromise the circulation (possibly lowering blood pressure). Remarkably, the very steps used to support the failing lung (oxygen and the ventilator) paradoxically have the capacity to further damage it, a process called ventilator-induced lung injury, discussed below.

For patients with hypercapnia, the ventilator can generate sufficient pressure to support or completely replace the bellows function of the lungs and respiratory muscles. The rate of breathing as well as its depth can be machine-controlled and adjusted as necessary. This is not always comfortable for the patient. On the one hand, by assuming the work of breathing, the ventilator can relieve the sense of shortness of breath. On the other hand, the specific settings on the ventilator (volume, rate, flow rate) may or may not match the expectations of the patient.

Further, many critically ill patients become agitated or delirious, related to their underlying disease. For this reason, ventilated patients in the intensive care unit are often sedated.

Some patients can be ventilated with a tight-fitting mask, rather than an endotracheal tube. Success is greater for patients with exacerbations of chronic bronchitis or emphysema or those with lung edema from congestive heart failure and is safer in those with stable blood pressures. Advantages of mask ventilation include less need for sedation and a reduced incidence of complications such as pneumonia. Further, the mask can be removed periodically to facilitate communication, eating, and oral hygiene.

Ventilators Can Damage the Lung

Evidence has accumulated over the last decades that ventilators not only support the lung, but also can damage it (ventilator-induced lung injury or VILI). Three potentially damaging aspects of ventilation have been identified: excessive volume, high concentrations of oxygen, and insufficient PEEP. Healthy adults tend to breathe with volumes of roughly 400 milliliters. Historically, volumes much larger than this were used in ventilated patients, such as 700-1100 milliliters. In a landmark study funded by the National Institutes of Health, critically ill patients with acute lung injury or ARDS were treated either with normal volumes (6 milliliters per kilogram of ideal body weight) or twice that amount. Survival was significantly greater in those ventilated with smaller volumes and this has now become the standard of care for such patients. The role of oxygen toxicity and insufficient PEEP is clearly evident in animal studies, but less well established in human disease, so there is less agreement about how best to use these treatments for hypoxemia.

The Role of Tracheostomy

Most patients with acute respiratory failure can be ventilated through an endotracheal tube for the duration of respiratory failure (which ranges from hours to several weeks, rarely longer). When the cause of respiratory failure resolves, the tube can be withdrawn fairly simply. With prolonged use, however, endotracheal tubes risk damage to the voice box and other complications. Tracheostomy involves a minor surgical procedure in which a passage is made from the neck into the main airway, through which a tube can be inserted. This tube, like the endotracheal tube, is connected to the ventilator, but is more comfortable, easier to remove and replace, and is less likely to cause complications over the long term. A tracheostomy can be used for permanent ventilation in those who cannot be liberated from the machine but can be removed from those recover. Once a tracheostomy tube is removed, the surgically created passage gradually closes off and heals with a small scar

Liberation from the Ventilator

Most patients mechanically ventilated in an intensive care unit will ultimately develop the ability to breathe again free of mechanical assistance. The average duration of ventilation is 6 days, but ranges from hours to many months. The prognosis is highly dependent on the quality of life and presence of other serious medical conditions before the episode of respiratory failure. Physicians have historically been slow to recognize when patients no longer need mechanical ventilation. In general, the condition that precipitated respiratory failure must be treated or remit before the ventilator can be removed. A corollary is that, once the precipitating condition has improved, the ventilator can be removed, as long as additional complications have not accumulated. This approach differs from the older notion of ventilator "weaning" in which ventilatory assistance was viewed as nurturing or benign, and was cautiously and gradually withdrawn. The current view is that unnecessarily prolonged mechanical ventilation risks excess sedation and life-threatening infection, and we emphasize active and rapid steps to withdraw it. New methods of assessment, some relying heavily on the respiratory therapist, have speeded the move toward safe liberation from the ventilator.

The two most important advances are the "daily wake-up" and "spontaneous breathing trials." In a study of mechanically ventilated adults, the routine daily discontinuation of sedative and analgesic medications ("wake-up") reduced the duration of ventilation, on average, by more than one day (10). Sedatives were withheld until the patient was able to follow simple commands or became agitated, following which sedation could be reinstituted. Remarkably, this approach reduced symptoms of post-traumatic stress disorder when patients were examined after 6 months. Coupled with the daily wake-up, most ventilated patients should be allowed to breathe for a brief period of time free of the machine. Some will become distressed and breathe rapidly and shallowly, showing that they are not yet ready to breathe independently. Others will breathe more slowly and deeply, predicting successful liberation. This simple measure, carried out by respiratory therapists or physicians, can reduce the time on a ventilator by more than one day.

In medicine, **Mechanical Ventilation** is a method to assist or replace spontaneous breathing. Mechanical ventilation can be life-saving and is a mainstay of CPR, intensive care medicine, and anesthesia.

Clinical Use

Mechanical ventilation is used when natural (spontaneous) breathing is absent (apnea) or insufficient. This may be the case in cases of intoxication, circulatory arrest, neurological disease or head trauma, paralysis of the breathing muscles due to spinal cord injury, or the effect of anesthetic or muscle relaxant drugs. Various pulmonary diseases or chest trauma, cardiac disease such as congestive heart failure, sepsis and shock may also necessitate ventilation.

Depending on the situation, mechanical ventilation may be continued for a few minutes or many years. While returning to spontaneous breathing is rarely a problem in routine anesthesia, weaning an intensive care patient from prolonged mechanical ventilation can take weeks or even months. Some patients never adequately regain the ability to breathe and require permanent mechanical ventilation. This is often the case with severe brain injury, spinal cord injury, or neurological disease.

Techniques

Positive and Negative Pressure Ventilation

While the exchange of oxygen and carbon dioxide between the bloodstream and the pulmonary airspace works by diffusion and requires no external work, air must be moved into and out of the lungs to make it available to the gas exchange process. In spontaneous breathing, a negative pressure is created in the pleural cavity by the muscles of respiration, and the resulting gradient between the atmospheric pressure and the pressure inside the thorax generates a flow of air. This is imitated by the *negative-pressure ventilation* that is employed in iron lungs. An iron lung works by creating an underpressure in a chamber which encloses the body and is sealed at the neck. With the patient's airways open, the resulting gradient to the atmospheric pressure serves to inflate the lungs. Inhalation serves to replenish alveolar gas. Prolonging the duration of the higher volume cycle enhances oxygen uptake, while increasing intrathoracic pressure and reducing time available for CO_2 removal.

All other techniques of ventilation are *positive pressure ventilation* techniques, meaning that air is forced into the lungs by an external overpressure.

Mouth-to-Mouth and Bag-Mask Systems

The simplest form of positive pressure ventilation is *mouth-to-mouth* or *mouth-to-nose* expired air ventilation by a bystander. cardiopulmonary resuscitation In expired air ventilation it is not possible to deliver oxygen-enriched air. Patients may only receive about 16 percent oxygen, in contrast to 21 percent in ambient air or up to 100 percent in other methods. However it is more than adequate. There is also a risk of disease transmission through blood contact. Mechanical devices such as a bag-mask-valve system are therefore preferred where available, but only if the bystander has had sufficient training.

A *bag-mask-valve system* consists of a face mask that is pressed over the patient's nose and mouth to achieve a tight seal, an elastic self-inflating bag that can be manually compressed to deliver air to the patient, and a valve to direct air flow. A source of oxygen can be connected to a reservoir attached to the bag to achieve a higher concentration of oxygen than that of ambient air.These simple techniques can maintain ventilation and consequently the life of an apnoeic patient for prolonged periods.

Mechanical Ventilators

In anesthesia and intensive care, mechanical ventilators are routinely used.

Ventilators allow various modes of mechanical ventilation ranging from assisted spontaneous breathing to fully controlled ventilation. In some cases, a patient can breathe almost naturally, receiving only an occasional "push" of air to augment individual breaths.

This is termed ***assisted (or augmented) ventilation***. Assisted ventilation modes are used in anesthesia and in the process of weaning the patient from controlled ventilation.

In sicker patients, the degree of ventilator-driven respiration can be increased, and if necessary, the ventilator can take over the work of breathing entirely (***controlled ventilation***). Modern ventilators allow a continuous adaptation of the degree of mechanical assistance according to the patient's individual demands.

The most common mode is called Synchronised Intermitant Mandatory Ventilation or S.I.M.V. Another newer mode is called B.I.P.A.P. or Bi-Level Phasic Airway Pressure. This is simply a machine cycling between two pressures (upper Inhalation and lower exhalation - see PEEP) at a minimum rate set by the Intensive Care staff. The advantage of this mode is the ability to syncrhornise quicker with the patients own breaths and the mode also copes with patients who are 'light' in analgesia and who 'fight' the ventilator, such as waking patients or 'weaning'.

The lungs of ventilated patients have a tendency to collapse partially, leading to impaired gas exchange. Therefore, many ventilation modes allow the use of PEEP (positive end-expiratory pressure). With PEEP, there is a residual pressure at the end of a breathing cycle that keeps the lungs inflated.

CPAP (Continous Raised Airway Pressure) is often confused with PEEP; but it is simply because both terms came from different respiratory support devices. Modern ventilators can offer both and in fact they are the same thing. A better term would be Continuous Raised Airawy Pressure, because that is all that happens, but the short form is not mentioned in polite society!

The gist of CPAP is that the techniques is delivered to spontaneous breathing patients (and cannot support life in an unconscious patient on it's own) and PEEP is the end expiratory phase of a ventilator.

CPAP & PEEP both act by raising the pressure in the patients lungs above that of normal atmospheric pressure. It's a bit like jet pilots having to wear O2 masks at high altitudes; but with CPAP / PEEP the enhanced O2 deleivery can be achieved at normal ground atmospheric air pressures.

Not only does CPAP / PEEP facilitate more O2 absorbtion across the lung membranes into the blood stream during respiration - but it serves to keep the Alveolar slightly pressurised and prevent them from collapsing.

Hopefully sustained CPAP / PEEP will also force fluid back across the membrane and splint the alveolar open, thus recruiting more lung space for gas exchange to take place. Remember that gas exchange is a two process, O2 inwards and CO2 outwards - plus other inert gases present in air, eg: Nitrogen (N2) - the elimination of N2 is of special importance to deep sea divers also.

Securing the Patient's Airways

Mechanical ventilation will be unsuccessful and dangerous unless the patient's airways are patent, meaning air can flow unimpeded back and forth into the lungs. It is also necessary to avoid air leakage so that air flow and pressure are maintained at the values set.

Another great risk is that of aspiration pneumonia. Aspiration is when stomach contents come back up the oesophagus and enter the trachea to enter the lungs. When stomach contents get into the lungs, the patient can actually drown due to the volume of gastic material, or, with less material, suffer damage to the lung tissue due to the acid content of the stomach. Measures to prevent aspiration depend on the situation and the individual patient - endotracheal intubation is often necessary to protect against this.

There are various procedures and mechanical devices that provide protection against airway collapse, air leakage, and aspiration:

- **Face mask** - In resuscitation and for minor procedures under anesthesia, a facemask is often sufficient to achieve a seal against air leakage. Airway patency of the unconscious patient is maintained either by manipulation of the jaw or by the use of *nasopharyngeal* or *oropharyngeal airway*. These are designed to provide a passage of air to the pharynx through the nose or mouth, respectively. A facemask does, however, not provide protection against aspiration. Facemasks are also used for "non-invasive ventilation" in conscious patients. Non-invasive ventilation is aimed at minimizing patient discomfort and ventilation-related disease. It is often used in cardiac or pulmonary disease.

- **Laryngeal Mask Airway** - The laryngeal mask airway (LMA), causes less pain and coughing than a tracheal tube. However, unlike tracheal tubes it does not seal against aspiration, making careful individualized evaluation and patient selection mandatory.

- **Tracheal Intubation** is often performed for mechanical ventilation of hours' to weeks' duration. A tube is inserted through the nose (nasotracheal intubation) or mouth (orotracheal intubation) and advanced into the trachea. In most cases tubes with inflatable cuffs are used for protection against leakage and aspiration. Intubation with a cuffed tube is thought to provide the best protection against aspiration. Tracheal tubes inevitably cause pain and coughing. Therefore, unless a patient is unconscious or anesthetized for other reasons, sedative drugs are usually given to provide tolerance of the tube.

- **Tracheostomy** - When patients require mechanical ventilation for more than days or a few weeks, tracheostomy provides the most suitable access to the patient's airways. A tracheostomy is a surgically created passage to the trachea. Tracheostomy tubes are well tolerated and often do not necessitate any use of sedative drugs.

  (*Note: the terminology for this procedure can be confusing. Often "**tracheotomy**" is used to denote the surgical procedure and "**tracheostom**y" the result of the procedure*)

Ventilation-Associated Lung Injury and Protective Ventilation

In most cases of mechanical ventilation, the patient's prognosis is determined by the underlying disease and its response to treatment. However, ventilation itself can cause significant problems that may prolong intensive care and sometimes lead to permanent injury and death. It is therefore desirable to limit mechanical ventilation to the shortest appropriate time.

Infectious complications, particularly pneumonia, occur in many patients who remain intubated for more than a few days. Tracheal intubation interferes with the natural defenses against lung infection, particularly with the process of "mucociliary clearance". This is a continuous transport of airway secretions from the lungs to the upper airways that serves to remove bacteria and foreign bodies. It is thought that the intubation-related disruption of this transport mechanism is a major factor in the development of pneumonia.

There is evidence that oxygen in higher concentrations may contribute to injury of lung tissue in ventilated patients. It is therefore recommended to set ventilators to deliver the lowest appropriate concentration of oxygen. However, in patients with severely impaired pulmonary gas exchange, high oxygen concentrations may be necessary for survival.

Most techniques of ventilation rely on an overpressure being applied to the lungs. In diseased lungs this may lead to further tissue injury caused by excessive mechanical stress (overdistension, shear forces, high peak pressure) and aggravated by inflammatory processes. Such mechanically induced lung injury can lead to severe impairment of the pulmonary gas exchange, thereby necessitating even more aggressive ventilation.

"**Protective Ventilation"** is a collective term for strategies to minimize ventilation-associated lung injury, many of which rely on sophisticated ventilator settings to reduce overdistension of the lungs.

Brief Historical Background

The iron lung was used through much of the middle 20th century, mostly for long-term ventilation. It was refined and used largely as a result of the polio epidemic that struck the world in the 1950s.

The machine is effectively a big elongated tank, which encases the patient up to the neck. The neck is sealed with a rubber gasket so that the patient's face (and airway) are exposed to the room air.

By means of a pump, the air is withdrawn mechanically to provide inspiration and released to room pressure to allow expiration.

Thus the patient inhales room air by a means of negative pressure applied to the patient's thoracic area. There are large portholes for nurse or home assistant access. patients could remain in these iron lungs for years at a time quite successfully. Some are still in use, notably with the Polio Wing Hospitals in England such as St Thomas' (by Westminster in London) and the John Radcliffe in Oxford.

The patients can talk and eat normally and see the world through a well placed series of mirrors.

A smaller device known as the curass was invented to place onto the chest wall like a giant plumber's suction plunger. It was prone to falling off and caused severe chaffing and skin damage and was not used as a long term device.

In recent years this device has re-surfaced as a modern polycarbonate shell with multiple seals and a high pressure oscillation pump. It has mostly been effective with children and is still in use in domiciliary ventilation in West England and Wales.

More Detailed History of Developments in Mechanical Ventilation

Respiratory problems have been around for as long as the respiratory system has existed. The need to help people with respiratory problems has therefore existed just as long. But the technology to assist these persons has been absent until about 250 years ago.

In 1743, an English clergyman and physiologist named Stephen Hales invented a device to aid people on ships and in mines. This was the first mechanical ventilator It operated through manual inflation of a bellows, which squeezed air into and out of the subject's lungs. As one would expect, this primitive ventilator did not work exceptionally well. It did not measure inspiratory or expiratory volume, and therefore efficient artificial respiration was not achieved.

The mechanical ventilator, although not an exceptionally helpful one, did exist, but in 1743 and for many years to come, no agency or group existed for distressed people to call upon. In 1790, that all changed. Thomas Beddoes founded the Pneumatic Institute for the treatment of diseases

by inhalation of gases. This institute gave people a ways of receiving the correct treatment for their problem. For the next 100 years, though, no real advancement occurred in artificial respiration.

In 1907, Sir Arbuthnot Lane invented the nasal catheter, which could deliver oxygen without obstructing the mouth. After World War I, John Haldane invented an oxygen mask for war victims suffering from pulmonary edema. Sir Leonard Hill invented an oxygen tent in 1920. All of these devices worked inadequately, as they did not greatly help those in need.

During the 1920s, it was discovered that the lungs worked on the principle of negative thoracic pressure to inflate the chest cavity. Attempts to duplicate this negative pressure brought about the likes of the iron lung, which was a chamber that enclosed the entire body except the head and sealed the chest cavity from the outside atmosphere. By creating a vacuum inside, the negative pressure would cause the lungs to expand and air to rush in from the outside higher-pressured atmosphere. This method was highly criticized because it caused a venous blood pool in the abdomen and reduced the patient's cardiac output.

More recent attempts at negative pressure ventilators have housed only the chest walls. Obtaining a proper seal around the chest walls is difficult at best, and the idea was dropped to focus on positive pressure ventilators.

Positive pressure ventilators are more recent additions to the artificial respiration arsenal. They operate on the idea that fluids move down a pressure gradient, and by creating a high pressure outside the lungs, air will move into the lungs. Two kinds of positive pressure ventilators exist, and they are open-loop and closed-loop. Open-loop ventilators have to be watched, recalibrated, and constantly operated by somebody. With technology advancing as fast as it is, open-loop systems are being replaced by their do-it-themselves counterpart, the closed-loop system.

Throughout the forty-five years that closed-loop mechanical ventilators have existed, many breakthroughs have come forth. Feedback technology and better receptors and sensors have put mechanical ventilators at their present day models. A general overview of closed-loop ventilators will explain the mistakes of the past and the reasons for the models of today.

As early as 1953, mechanical ventilators with a closed-loop control system were present. Saxton and Myers developed the first model, which relied on the end-tidal partial pressure of carbon dioxide to provide the feedback information. This variable was monitored by an infrared gas analyzer.

Many people tried to improve this pioneering model for closed-loop mechanical ventilation. In 1971, a scientist named Mitamura and his colleagues presented a ventilator which monitored the expired fraction of carbon dioxide. This model kept the patient's carbon dioxide level within 9 mm Hg of its initial value.

In 1973, a researcher named Coles developed a system that monitored the end-tidal CO_2 fraction and responded accordingly. Because of the development of the computer, this model used a proportional integral controller to adjust the tidal volume with a fixed frequency of respiration.

In 1975, Mitamura presented another ventilator which used the volume of expired CO_2 to establish the desired output. This system was mainly an improvement of his first model as the CO_2 level of his patients stayed within 3 mm Hg of its initial value.

In 1978, Coon and his colleagues developed a ventilator that had an intra-arterial sensor for monitoring the pH or pCO_2 levels of the subject.

From this point on, computers played an important part in mechanical ventilator technology. Microprocessors, algorithms, and integral controllers were used to give the correct amount of oxygen. Until the late 1980s, all of the ventilators relied on a single variable to generate desired outputs. In 1987, this changed.

The first closed-loop ventilator that utilized multiple variables to control ventilation was developed in 1987 by Fleur Tehrani. The variables that were used included the arterial carbon dioxide and oxygen pressures, the respiratory compliance factor, the airway resistance, metabolic rate, and the barometric pressure. Continuous monitoring of some variables is necessary, but some information can be stored in the memory. Respiratory failure is often caused by failure to ventilate, characterized by increased arterial carbon dioxide tension or failure to oxygenate, characterized by decreased arterial oxygen tension. Failure to oxygenate is caused by reduced diffusing capacity and ventilation perfusion mismatch.

Computed values of breathing frequency and depth are used to determine the necessary volume and pressure to be sent to the lungs. Individual parameters may be programmed into this ventilator since no two people are exactly alike. Alarms sound if the person's vital statistics exceed these parameters.

With computerization of jobs becoming more evident, human error and high cost can be eliminated by using closed-loop methods to control mechanical ventilators.

Introduction

Mechanical ventilators have the function of breathing when patients, because of diseases or injuries, are not capable of breathing efficiently on their own. The most common uses are in the treatment of heart attacks, various diseases such as emphysema, bronchitis or polio, and serious injuries to the nervous system.

Unfortunately, mechanical ventilation is uncomfortable and stressful for the patient. One of the main causes of discomfort is the endotracheal (ET) tube which makes the air flow from the ventilator to the lungs. This tube prevents the patient from eating and talking because it is placed in the mouth, down the throat, and into the trachea. Another discomfort comes from the small tube that patients have into one of their arteries to monitor oxygen and carbon dioxide levels in the blood. Dysynchrony is a term which describes a patient fighting the ventilator.

The three different types of ventilators are negative pressure ventilators, positive pressure ventilators and high frequency ventilators. Pressure ventilators and volume ventilators try to solve problematic gas exchange by producing tidal ventilation. Pressure assist ventilation (PAV) is pressure control without a set rate. This can be achieved either in combination with the patient's respiratory effort, or independently. While in inspiration, there is a forcing of gases from the ventilator circuit into the patient's lungs, and during expiration, gases are permitted to exit with minimal restriction. All of this implies that there is the need for some mechanism for switching from inspiration to expiration and then back again to inspiration.

All of the different types of ventilators provide ventilation and oxygenation. Ventilation is the product of *a tidal volume* and *respiratory rate,* while oxygenation is a function of inhaled oxygen concentration, a variable which can be regulated in all types of ventilators. Since oxygenation is proportional to Mean Airway Pressure (MAP), this parameter correlates very well with oxygenation.

It is very important to assess the right goals for both ventilation and oxygenation in managing a patient on a ventilator. These goals can depend critically on the context of the patient. In ventilation, goals can be a range of pH values and/or a range of $PaCO_2$ values. Monitoring arterial blood gases is a good way to watch these ranges. If a conventional ventilator (not High Frequency) is employed, end-tidal CO_2 monitoring is another very important non-invasive method of estimating ventilation. The most important goals in oxygenation are the ranges of arterial oxygen saturation or PaO_2 values. Monitoring pulse oximetry or arterial blood gases gives the best results for oxygenation.

Until very recently, the settings of the ventilators (which control and monitor respiratory performance) were defined heuristically. A new approach is trying to use artificial intelligence to perform these monitoring functions.

Positive Pressure Ventilator

These ventilators use pressure to inflate the lungs of the patient. During inspiration, a positive intra-thoracic and alveolar pressure is caused by the pressure created by the ventilator. The treatment for failure to ventilate is to increase the patient’s alveolar ventilation, that is the rate and depth of breathing, either by reversing the cause or by using mechanical ventilation; invasively or non-invasively. As with negative pressure ventilation, expiration is largely passive. Most of the newer volume ventilators have the capacity to regularly create small volumes. This fact is very useful for the treatment of infants.

Most portable ventilators are typically "volume ventilators." The main features of portable ventilators list both an external battery and an internal power source (able to provide power up to an hour), the possibility of measuring the volumes of delivered and exhaled gases, and also an alert system to warn patient or hospital personnel in case of low pressures.

The main advantage of volume-controlled ventilators is guaranteed minute ventilation. This is particularly important in the operating room.

Advantages: By knowing the tidal volume, it is possible to have stable minute ventilation. Also, they are relatively small in size, therefore usable outside the hospital environment, allowing patients to lead as much as possible a normal life.

Disadvantages: Because of the application of direct positive pressure to the lungs in the case of decreased lung compliance, maintaining an adequate tidal volume causes the peak airway pressure to increase, and this might lead to barotrauma. A new type of ventilator belonging to the nonconstant-flow generator family called DIGIT has been used to ventilate patients with high respiratory resistance and/or an anomalous variability compliance. Its main characteristic is the possibility of changing from a pressure generator to a flow generator and vice versa.

Another disadvantage is an increased risk for infections and airway obstruction since the patient often requires a tracheostomy.

Negative Pressure Ventilators

This type of ventilator helps patients by either assisting and/or increasing the normal respiration or by totally regulating a planned breathing pattern for patients who are not able to breathe without mechanical assistance. The ventilator generates negative extra-thoracic pressure. Negative pressure expands the thorax. Therefore, it produces a negative pressure in the alveoli, which causes air to move into the lungs. Negative pressure ventilators are devices with constant flow. They can be time cycled, in which case breaths are given at fixed intervals (newer models can sense breaths and synchronize to them). They also can be pressure limited. In this case, when a prefixed peak inspiratory pressure is reached, it is maintained for the duration of the inspiratory cycle.

Between the most common types of negative pressure ventilators are the iron lung, the cuirass, and the pneumo-wrap. The iron lung is a very big metal cylinder with a flexible diaphragm activated and controlled by a piston rod at the distal end. Because of its size, therefore requiring a lot of space, and because of the fact that it is not accessible with ease, it is uncomfortable for a lot of patients, even if it is the most effective negative pressure generator.

The cuirass ventilator is a rigid shell, which extends from the symphysis pubis to the supra sternal notch, and adheres to the chest and abdominal walls using a flexible diaphragm made of rubber.This ventilator is the least constraining of the negative pressure family because it just covers the patient's chest and not the whole body like an iron lung. One of the main disadvantages is the small tidal volume which the cuirass generates in patients with a normal chest wall and lungs. In the end, it is definitely less efficient than the iron lung.

The pneumo-wrap works by having the patient wear a suit made of plastic while lying supine on a flat rigid plate with a large plastic grate on his/her thorax. Thanks to the plastic grid, the pressure generated by the suit is applied mostly to the thorax since the plastic is sucked tightly against all other body parts. The main advantages of the pneumo-wrap are its portability, lightness, and comfort, especially compared with the iron lung. Unfortunately, it generates small tidal volumes, therefore only patients who are not completely dependent on ventilatory support can benefit from it.

Advantages: Negative pressure ventilators have a simple and reliable design and do not usually require a tracheostomy. Thus, the patient has the possibility of talking while being mechanically ventilated. Also, in case of use with infants, the constant flow allows the baby to easily take spontaneous breaths.

Disadvantages: Not all patients can use negative pressure ventilators since an acceptable ventilation for long period of times cannot be provided. Also, a poor lung compliance causes tidal volume to drop, while an improvement in compliance may result in over-distention. Another problem is the lack of complete access to patients, which blocks acceptable chest physiotherapy.

High Frequency Ventilators

This type of ventilator is the latest in ventilator design. In high-frequency ventilation (HFV), the main characteristics are the use of a smaller tidal volume (similar or less than dead space, which is the airways where no gaseous exchange takes place) and more rapid rates (namely greater than 50 breaths per minute). Because of this, the results are a lower airway and intra-thoracic pressure, which decreases the incidence of the main two side-effects of positive pressure ventilation, namely barotrauma and circulatory depression. Another important aspect of HFV is the non-interference with spontaneous breathing.

The term High-Frequency Ventilators combines together three types of ventilators:

- High-frequency Jet ventilation: It uses very short high pressure jets of air delivered from a high-pressure source into the airway via a small catheter or additional lumen in a special endotracheal (ET) tube. Rates are usually between 100 and 400 breaths per minute. It also relies on passive exhalation.
- Flow Interrupter: The technology it uses is similar to jet ventilator technology. It relies on passive exhalation but does not use a high velocity jet and does not need a special ET tube or circuit.
- High Frequency Oscillatory Ventilation (HFOV): It consists of a diaphragm piston unit which actively conveys gas in and out of the lung. It needs a special noncompliant breathing circuit.

Advantages: It reduces barotrauma (this belief has recently come under scrutiny though) and circulatory depression. It may allow gas exchange when conventional ventilation has failed. Patients can be ventilated through a small insufflation catheter, and this eliminates the competition for the airway with the surgeon.

Disadvantages: It is still not exactly clear which patients will respond. High-frequency ventilation often causes a high airway pressure which can be transmitted to the heart (especially in cases with compliant lungs) and the result might be an impaired cardiac output.

Closed-Loop Ventilators

In most mechanical ventilators, despite recent advances in biomedical engineering, the main control settings which determine total ventilation and breathing frequency of patients are manually adjusted. Closed-loop control mode mechanical ventilators automatically set these two variables. This helps reducing typical problems of traditional open-loop ventilators such as frequent and costly human interventions, and also reduces the training needed by medical personnel to use and properly adjust many vital variables on the machine. The main limitations of automatic control of ventilation is strictly connected with the reliability and accuracy of physiological sensors. To help to solve this problem, a new class of sensors, known as Symmetric Differential Capacitative (SDC) have been developed for remote respiratory monitoring.

Recent Developments

A new treatment for patients with high-level complete cervical spinal cord injury (SCI) uses electrical stimulation of abdominal muscles. A detailed analysis of the breathing mechanism showed that both tidal volume and frequency of respiration could be increased while the muscles involved in breathing are electrically stimulated, synchronously with naturally occurring breathing. The results of this new technique show that there is hope to give a better clinical outcome for SCI patients when it used in conjunction with mechanical ventilation.

Another new interesting approach involves an adaptive lung ventilation controlling device. In most of the traditional mechanical ventilators, controllers were based on mean expired CO_2, end-tidal CO_2 arterial CO_2 or pH, while the controlled variables were mainly inspiratory pressure and tidal volume. Controlling only these values can be dangerous for patients with lung emboli because physiologic dead space ventilation is not monitored. Also, patient size or lung mechanics were not taken into consideration. These facts prompted a new method for closed loop control of ventilation, defined as Adaptive Lung Ventilation (ALV). This method is based on a pressure controlled ventilation mode. Pressure Control refers to the type of breath delivered, not the mode of ventilation. It can be used for both SCI and spontaneously breathing patients, and it is characterized by using the ALV controller to match a desired gross alveolar ventilation by adjusting mechanical rate and inspiratory pressure level.

Patient Requirements

Ventilation is used as a therapeutic technique to keep appropriate arterial levels of oxygen, CO_2 and pH, and, at the same time, to minimize risk, discomfort, and time on ventilation.

The composition of the inspired gas and the selection of a breathing pattern are used to calibrate blood-gas levels and pH.

Since blood sampling of these two values occurs periodically, the therapist can consequently adjust the gas composition and the breathing pattern.
A breathing pattern is described by three factors: tidal volume, respiratory frequency and the ratio of the durations of inspiration and expiration (I:E ratio). Different combinations of these three factors are chosen by the therapist based on needs of the patient and past experiences. For example, low tidal volume and high respiratory frequency get rid of the risk for a buildup of high trans-thoracic pressures, while high tidal volume and low respiratory frequency help to increase ventilation distribution and to avoid problems due to airway resistance.

When a patient suffers from impaired gas exchange, mechanical ventilation controlling parameters that are able to remove an adequate quantity of CO_2 may not oxygenate the patient's blood sufficiently since oxygen diffuses more slowly than CO_2. In such patients, hypoxia can be avoided through hyperventilation of the lungs, and a selected CO_2 value can be provided by increasing the dead space or by including CO_2 in the mixture inspired by the patient.

Generally speaking, mechanical ventilators try to give the patient a ventilation aid which is as close as possible to normal respiration. For example, the inspired gas needs to be at a temperature between 32 C and 39 C with a 75% relative humidity to avoid the risk of airway drying. Also, the inspired gas needs to be filtered so that the patient is not exposed to factors which might cause irritation.

Another important requirement is monitoring and balancing the thoracic pressure. With positive pressure ventilators, an incorrect pressure value might reduce venous return, lower the systematic blood pressure, and even burst a lung. With a negative pressure ventilator, wrong pressure values might cause lower venous return and modification of the fluid balance. To minimize all these problems, the average pressure gradient over the breath cycle needs to be as small as possible. Also, the peak intrapulmonary pressure needs to be kept low.

Since abnormalities in the thoracic structure or in the cardiovascular functions of mechanical ventilated patients can increase the risk of these side effects and since ventilating a patient with normal thoracic structure and cardiovascular functions is not particularly problematic, a mechanical ventilator needs to be a very flexible machine. This is because a broad range of selections and alternatives in gas composition, breathing patterns, pressure and flow characteristics are required to offer the best care to the largest number of patients.

Safety Issues

Because of the vital functions performed, a mechanical ventilator must satisfy strict safety regulations. Therapists and patients need to be shielded from the possibility of electrical shocks. Mechanical ventilators must not generate electromagnetic radiation strong enough to interfere with other ICU medical devices or medical mechanical aids used by patients, such as pacemakers. Control and indicator displays/devices need to be extremely accurate to monitor carefully patients' conditions, and, also, easy to read to avoid ambiguities and/or misreading.

Because of their critical functions, mechanical ventilators need to be reliable with autonomous power sources to avoid interruption caused by external power outages. Also, it needs to be supplemented with alarms which signal immediately if interruption of service, a critical change in the vital signs of the patient, incorrect functioning, or malfunctioning occurs.

Future Developments

Over the years, measuring and treatment devices of the respiratory system have progressed technologically. Due to these advancements, most of the diseases associated with the respiratory system are treatable. Future research in this field involves attempting to find safer, cheaper, and more practical methods or devices to help patients. Many advancements made in closed-loop ventilator control and medical imaging make this possible. Other work involves investigation into fields studied before, just not in the same amount of detail, such as the use of lung acoustics. The field of cryogenics also provides the field of respiratory system study with an advantage due to the new ability to preserve associated cells. After these cells are frozen, they can be thawed and used any time while retaining their original functionality.

Ventilator Control

Until recently, ventilators could only handle simple modes of ventilation (Modes of ventilation describe the primary method of inspiratory assistance). Patients who were totally paralyzed or experienced spontaneous breathing patterns could not benefit through a mechanical ventilator. New ventilators are now able to respond to mixed modes of breathing through negative feedback control. Mechanical ventilators of the past also only controlled one artifact at a time. This would leave the uncontrolled characteristics fixed. Newer ventilators account for multiple artifacts, such as respiratory rate, tidal volume, and cycling pressure simultaneously by constantly adjusting each as required. Another advancement in ventilator control includes the redesign of particular valves in the ventilator. One such valve, the constant pressure valve at the end of the expiratory tube, controls the PEEP (positive end-expiratory pressure). The magnitude of PEEP is determined by a spring-loaded mechanism on the expiratory valve. When the PEEP value is equal to the pressure within the alveoli, more air is expired. When the pressure is not equal, hyperinflation of the alveolar sacs occurs, otherwise known as auto-PEEP. Auto-PEEP is the result of the expiratory time not comparing to the expiratory drive against the constant pressure valve. New research shows that by replacing the constant pressure valve with an on/off valve, the

ventilator can reduce minimum expiratory time and maintain the desired PEEP. This new on/off valve is controlled by parameters obtained from a respiratory model.

Lung Acoustics

While the study of lung acoustics is not new, several new advancements use this field to develop cheaper, safer, and more practical methods of helping patients. One such method for diagnosing lung disease uses TSS (transmitted sound signals). This method is simple because it involves the patient repeating several vowel sounds such as iii, while just one transducer measures the resultant sound signals from within the chest. Under normal conditions, the low frequency sounds of iii will be returned as iii to the transducer. High frequencies are usually filtered out by healthy lung tissue. In cases where diseased tissue is present, the high frequencies are enhanced and the iii sounds more like an aaah sound through the transducer.

Lung acoustics also help in mapping the regions of the lung. Research in the 1980s revealed that larger alveoli conducted sound slower than small alveoli. Using this concept, sound travels through collapsed alveoli much faster than it travels through normal functioning alveoli. Combining the different frequencies and microphone locations determines a map of the lungs. This method is much cheaper and safer than alternatives such as CAT scans, especially for routine use.

Medical Imaging

Advances in this field as it relates to the respiratory system include finding more practical methods for obtaining images and enhancing current images. Electrical Impedance Imaging (EIT) measures conductivity distribution on the surface of the chest when imaging the lungs. Although this method is not very good for anatomical images, its noninvasive and inexpensive characteristics make it ideal for obtaining functional images of the respiratory system. Comparing the results of EIT to a Finite Element Model (FEM), physicians can determine lung ventilation.

Chest radiographs provide images of the respiratory system that have a lot of gray scale area around the lungs. This gray area makes it difficult to determine the exact region of the lung. Overcoming the overlap of pixels in these images boosts the intensities of specific regions in the radiograph. This can be done by changing the pixel values in one of two ways. Anatomic region-based histogram equalization (AHEQ) equally distributes pixel intensity throughout a given region to enhance that particular region. Anatomic region-based dynamic range compression (ADRC) makes the pixel range of the lungs narrower while preserving distribution of the lungs, heart, and diaphragm. This improves the visualization of the lungs. These two methods can also be combined to improve image quality.

Cryogenic Applications

Recent studies demonstrate that human bronchi cells survive cryogenic freezing and retain the majority, if not all, of their functionality upon thawing. This discovery allows researchers to collect human tissues over time and preserve them until enough cells are available to conduct studies on them. It is often the case that the supply of human tissue is irregular. This possibility for more research on human respiratory tissue provides a new outlook for advances in this field.

Mechanical Ventilation: What You Need To Know

Abbreviations used in this course:

A/C=assist-control ventilation; APRV=airway pressure release ventilation; ARDS=adult (or acute) respiratory distress syndrome; auto-PEEP=increase above set PEEP level, in end-exhalation alveolar pressure (also termed dynamic hyperinflation, intrinsic PEEP, also quantified as $\dot{V}EE$); BPF=bronchopleural fistula; CaO_2=concentration of oxygen in arterial blood; CMV=conventional mechanical ventilation; CPAP=continuous positive airway pressure; DH=dynamic hyperinflation (see auto-PEEP); DO_2=oxygen delivery; ECMO=extracorporeal membrane oxygenation; $ECCO_2R$=extracorporeal CO_2removal; ET=endotracheal; FIO_2=fractional concentration of inspired oxygen; FRC=functional residual capacity; HFJV=high-frequency jet ventilation; HFV=high-frequency ventilation; ICP=intracranial pressure; I:E=inspiratory:expiratory ratio; ILV=independent lung ventilation; IMV=intermittent mandatory ventilation; IRV=inverse ratio ventilation; IVOX=intravascular blood gas exchanger; LFPPV=low-frequency positive pressure ventilation; LV=left ventricle; MalvP=mean alveolar pressure; MAP=mean airway pressure; MMV=mandatory minute ventilation; OAD=obstructive airways disease; PAP=peak airway pressure; Paw=airway pressure; PCIRV=pressure-controlled inverse ratio ventilation; PEEP=positive end-expiratory pressure; PIP=peak inspiratory pressure; Pmus=pressure generated by muscle contraction; Ppl=pleural pressure; PPV=positive pressure ventilation; PS=pressure support; PSV=pressure support ventilation; Ptot=total distending pressure; P-V=pressure-volume; RA=right atrium; RV=right ventricle, residual volume; SaO_2=oxygen percent saturation (arterial); SIMV=synchronized intermittent mandatory ventilation; T_I=inspiratory time; TLC=total lung capacity; VC=vital capacity; $\dot{V}CO_2$=CO_2 production (elimination); V_D=dead space; $\dot{V}E$=minute ventilation; $\dot{V}EE$=end-expiratory lung volume; $\dot{V}EI$=end-inspiratory lung volume; $\dot{V}/Q$=ventilation/perfusion ratio; V_T=tidal volume; $\dot{V}O_2$=oxygen consumption (uptake)

Much of this course is taken from a report issued by a consensus committee of the American Chest Physicians.

Objectives and Specific Recommendations

Section 1: Objectives of Consensus Committee

Although the concept of artificial respiration was recognized in the 16th century by Vesalius, it was not until the 20th century that mechanical ventilation became a widely used therapeutic modality. Over the past 30 years, and especially over the past decade, there has been an explosion of new ventilatory techniques that present a bewildering array of alternatives for the treatment of patients with respiratory failure. Unfortunately, although the number of options available to the clinician has appeared to increase exponentially, well-controlled clinical trials defining the specific role for each of these modes of ventilation and comparing them with other modes of ventilation have not been forthcoming. In addition, over the past few years, our understanding of the detrimental as well as beneficial effects of mechanical ventilation has increased, along with novel strategies for limiting these negative effects.

The consensus committee was international in scope (Europe, North America, New Zealand) and consisted of individuals from a broad range of backgrounds (anesthesia, critical care, pulmonary, respiratory therapy). The purpose of the conference was to summarize key concepts related to mechanical ventilation and to present recommendations based on these concepts for clinicians applying mechanical ventilation in the adult ICU setting. Due to the lack of randomized clinical studies on most aspects of ventilatory care, the underlying theme of this document is a physiologic one, the basic tenet being that if the clinician understands the physiologic principles, he or she can apply mechanical ventilation at the bedside in a rational manner. We recognize that rational application of mechanical ventilation does not, in and of itself, guarantee the therapy will be beneficial to the patient. We await randomized clinical trial results for ultimate guidance.

The purpose of the consensus conference was not to deal with every possible aspect of mechanical ventilation. Rather, the focus was on treatment of patients with acute ventilatory failure and the principles of ventilation *after* the decision to initiate mechanical ventilation has been made. The technical needs and medical issues of ventilatory support in non-ICU settings were not dealt with specifically by this conference. These issues include ventilatory support during anesthesia and surgery; patient treatment during intrahospital, land, or air transport; and long-term mechanical ventilation in the home. Also not covered were noninvasive (nonintubated) ventilatory support, negative pressure ventilation, and methods and devices for respiratory support not primarily applied to the lungs, such as extracorporeal membrane oxygenation (ECMO), extracorporeal carbon dioxide removal ($ECCO_2R$), or intravascular blood gas exchanger (IVOX).

Despite the multidisciplinary and international composition of the conference, we were able to reach agreement on many difficult clinical issues. Yet even though we engaged in considerable discussion and debate, consensus was not possible on a number of aspects of ventilator care. This is hardly surprising and merely reflects the variety of acceptable approaches that can be used to treat patients with respiratory failure. For example, it was not possible to agree that there was an optimum *mode* of ventilation for any disease state or an optimum method of weaning patients from mechanical ventilation. However, in addition to the specific recommendations discussed in Section 2, there was general agreement on the following principles that should guide the use of mechanical ventilation:

1. The underlying pathophysiology of various disease states varies with time, and thus the mode, settings, and intensity of ventilation should be reassessed repeatedly.

2. Mechanical ventilation is associated with a number of adverse consequences, and as such, measures to minimize such complications should be implemented wherever possible.

3. To minimize side effects, the physiologic targets do not have to be in the normal range. For example, at times it may be beneficial to allow the $PaCO_2$ to increase (controlled hypoventilation, permissive hypercapnia) rather than risk the dangers of lung hyperinflation.

4. Alveolar overdistention can cause alveolar damage or air leaks (barotrauma). Hence, maneuvers to prevent the development of excess alveolar (or transpulmonary) pressure should be instituted if necessary. While recognizing that the causes of ventilator-induced lung injury are multifactorial, the consensus committee generally believed that end-inspiratory occlusion pressure (*i.e.,* plateau pressure) was the best, clinically applicable estimate of average peak alveolar pressure, and thus, was the most important target pressure when trying to avoid alveolar overdistention. Many individuals on the consensus committee believed that high plateau pressures (>35 cm H_2O) may be more harmful in most patients than high values of FIO_2.

5. Dynamic hyperinflation (DH) (gas trapping, auto-PEEP, intrinsic PEEP) often goes unnoticed and should be measured or estimated, especially in patients with airway obstruction. Management should be directed toward limiting the development of DH and its adverse consequences in these patients.

This consensus document is divided into two major sections: part 1 summarizes the objectives and specific recommendations of the consensus committee; part 2 reviews key principles regarding physiology, complications, and modes of ventilation that form the basis for the recommendations.

Section 2: Objectives of Mechanical Ventilation

Mechanical ventilation and continuous positive airway pressure (CPAP) are methods of supporting intubated patients during illness, and are not, in and of themselves, curative or therapeutic. Indeed, in certain clinical settings, there may be effective alternative therapies that do not require intubation and mechanical ventilation. The fundamental objectives for ventilatory support in acutely ill patients may be viewed physiologically and clinically, as detailed below. The following objectives should be kept in mind, not only when mechanical ventilation is initiated, but also at frequent intervals during the period of support; mechanical ventilation

should be withdrawn whenever the underlying pathophysiologic rationale for initiating mechanical ventilation is no longer present.

A. Physiologic Objectives

1. To Support or Otherwise Manipulate Pulmonary Gas Exchange:

*(i) Alveolar Ventilation (*eg, *Arterial P_{CO_2} and pH).*

In most applications of ventilatory support, the objective is to normalize alveolar ventilation. In certain specific clinical circumstances, the objective may be to achieve an alveolar ventilation greater than normal (as in deliberate hyperventilation to reduce intracranial pressure [ICP]), or adequate but less than normal (as in permissive hypercapnia or acute-on-chronic ventilatory failure).

*(ii) Arterial Oxygenation (*eg, *PaO_2, SaO_2, and CaO_2).*

A critical objective of mechanical ventilation is to achieve and maintain a level of arterial blood oxygenation that is acceptable for the clinical setting, using an inspired oxygen concentration that is also acceptable. In most applications of ventilatory support, this means an SaO_2 >90 percent (roughly equivalent to a PaO_2 ?60 mm Hg assuming a normal position of the oxyhemoglobin dissociation curve), although other end points are appropriate in certain settings.

There is no clinical evidence that a PaO_2 greater than normal is advantageous. Given other techniques for improving oxygenation, this objective would seldom be the only reason for initiating mechanical ventilation. Because arterial oxygen content is determined by hemoglobin as well as PaO_2, and because systemic oxygen delivery is directly related to cardiac output (Qt), as well as concentration of oxygen in arterial blood (CaO_2), these factors must also be considered in therapy aimed at improving tissue oxygenation.

2. To Increase Lung Volume:

(i) End-Inspiratory Lung Inflation.

To achieve sufficient lung expansion, with every breath (or intermittently), to prevent or treat atelectasis and its attendant effects on oxygenation, compliance, and lung defense mechanisms.

(ii) Functional Residual Capacity (FRC).

To achieve and maintain an increased FRC using PEEP in settings in which a reduction in FRC may be detrimental (*eg,* decreased PaO_2, increased lung injury) as in adult respiratory distress syndrome (ARDS) and postoperative pain.

3. To Reduce or Otherwise Manipulate the Work of Breathing:

(i) To Unload the Ventilatory Muscles.

To reduce the patient's work of breathing when it is increased by elevated airway resistance or reduced compliance and the patient's spontaneous efforts are ineffective or incapable of being sustained. In these situations, ventilatory support will be used until specific therapies (or other mechanisms) reverse the condition leading to the increased work load.

B. Clinical Objectives

Because, at best, mechanical ventilation serves only to support the failing respiratory system until improvement in its function can occur (either spontaneously or as a result of other interventions), a primary objective should be to avoid iatrogenic lung injury and other complications.

The other primary clinical objectives of mechanical ventilation are as follows:

1. *To Reverse Hypoxemia:* To increase PaO_2 (generally such that $SaO_2 \geq 90$ percent), whether through increasing alveolar ventilation, increasing lung volume, decreasing O_2 consumption, or other measures, to relieve potentially life- or tissue-threatening hypoxia.

2. *To Reverse Acute Respiratory Acidosis:* To correct an immediately "life-threatening" acidemia, rather than necessarily to achieve a normal arterial P_{CO_2}.

3. *To Relieve Respiratory Distress:* To relieve intolerable patient discomfort while the primary disease process reverses or improves.

There are well-defined circumstances in which attempts to improve PaO_2 or pH to their normal ranges would present greater overall risks to the patient, and lower values of these parameters may be appropriate in such circumstances. In addition to the main clinical objectives listed above, other specific goals for mechanical ventilation, in appropriate settings, include the following:

4. *To Prevent or Reverse Atelectasis:* To avoid or correct the adverse clinical effects of incomplete lung inflation, as, for example, in postoperative splinting or neuromuscular disease.

5. *To Reverse Ventilatory Muscle Fatigue:* In most instances, this means unloading the ventilatory muscles in circumstances of acutely increased and intolerable loads.

6. *To Permit Sedation and/or Neuromuscular Blockade:* To allow the patient to be rendered incapable of spontaneous ventilation, as for operative anesthesia and certain ICU procedures and in certain disease states.

7. *To Decrease Systemic or Myocardial Oxygen Consumption:* To lower systemic and/or myocardial oxygen consumption ($\dot{V}_{O_2}$) when the work of breathing or other muscular activity impairs systemic O_2 delivery or produces an overload of the compromised heart. Examples include cardiogenic shock or severe ARDS.

8. *To Reduce Intracranial Pressure (ICP):* In certain circumstances (*e.g.,* acute closed head injury), to lower elevated ICP through controlled hyperventilation.

9. *To Stabilize the Chest Wall:* In the unusual circumstance of loss of thoracic integrity sufficient to prevent adequate bellows function (*e.g.,* chest wall resection, massive flail chest), to provide adequate ventilation and lung expansion.

Section 3: Clinical Recommendations

A. Mechanical Ventilation for Specific Entities

Adult Respiratory Distress Syndrome (ARDS): Although it has been argued that patients with ARDS are now more severely ill than those encountered in the past, the failure of ARDS mortality to decrease during the past 15 to 20 years is disappointing, particularly in light of the many technical advances in ICU care. Criteria that selected a severely hypoxemic subset of patients with ARDS were established in the 1974 to 1977 ECMO clinical trial. Recent work indicates that the average survival of such patients with severe ARDS treated with conventional mechanical ventilation only ranges from 0 percent to 15 percent (mean=12.8±5.2 percent). This is not statistically significantly different from the ECMO clinical trial survival of 9 percent ($p=0.15$). Recent changes in ventilatory management may have led to increased patient survival, but to our knowledge, this has not been evaluated with controlled clinical trials.

There are no convincing data indicating that any ventilatory support mode is superior to others for patients with ARDS. Nevertheless, reported increases in ARDS patient survival have been ascribed to new techniques such as pressure-controlled inverse ratio ventilation (PCIRV) and low-frequency positive pressure ventilation-extracorporeal CO_2 removal (LFPPV-ECCO$_2$R). If they are harbingers of new and effective therapy, their putative benefit may be linked to current concern about iatrogenic lung damage induced by mechanical ventilation.

Animal studies have clearly established the damaging effects of overdistention produced by the application of high peak transthoracic pressures to normal and injured lungs. In humans, the nonuniformly injured severe ARDS lung retains only a small fraction of compliant lung still capable of gas exchange. It has been argued that the application of commonly used tidal volumes (0.7 L, 10 ml/kg) to this small fraction of lung may produce similar damage. The best correlate of this injury in animals is the plateau pressure.

Conventional mechanical ventilator (CMV) therapy might thus superimpose iatrogenic lung injury. The “lung rest” strategy used in neonatal ECMO therapy, intentional hypoventilation used in patients with ARDS, and reduction of peak pressures permitted by PCIRV or by LFPPV-ECCO$_2$R are consistent with this argument. There is, however, no proof that any of these techniques alter the outcome of patients with ARDS. A recent randomized controlled clinical trial of PCIRV plus LFPPV-ECCO$_2$R failed to produce evidence of improved survival. Of interest is the fourfold increase in survival of control patients treated with mechanical ventilation only, according to a computerized protocol.

Guidelines:

1. The clinician should choose a ventilator mode that has been shown to be capable of supporting oxygenation and ventilation in patients with ARDS and that the clinician has experience in using.

2. An acceptable SaO_2 (usually $\geq$90 percent) should be targeted.

3. Based primarily on animal data, a plateau pressure $\geq$35 cm H_2O is of concern. We, therefore, recommend that when plateau pressure equals or exceeds this pressure that tidal volume (V_T) can be decreased (to as low as 5 ml/kg, or lower, if necessary). With clinical conditions that are associated with decreased chest wall compliance, plateau pressures somewhat greater than 35 cm H_2O may be acceptable.

4. To accomplish the goal of limiting plateau pressure, $PaCO_2$ should be permitted to rise (permissive hypercapnia) unless the presence or risk of raised ICP or other contraindications exist that demand a more normal $PaCO_2$ or pH. Rapid rises in $PaCO_2$ should be avoided. In the presence of normal renal function, slow reduction of tidal volume may also allow renal-induced compensatory metabolic alkalosis and the potential for a higher pH at a given tidal volume.

5. Positive end-expiratory pressure (PEEP) is useful in supporting oxygenation. An appropriate level of PEEP may be helpful in preventing lung damage. The level of PEEP, however, should be minimized as PEEP may also be associated with deleterious effects. The level of PEEP required should be established by empirical trial and reevaluated on a regular basis.

6. The current opinion is that FI_{O_2} should be minimized. The trade-off, however, may be a higher plateau pressure and the relative risks of these two factors are not known. In some clinical situations when significant concerns over both elevated plateau pressure and high FI_{O_2} exist, consideration for accepting an SaO_2 slightly less than 90 percent is reasonable.

7. When oxygenation is inadequate, sedation, paralysis, and position change are possible therapeutic measures. Other factors in oxygen delivery (*i.e.,* Q_T and hemoglobin) should also be considered.

Bronchopleural Fistula: Bronchopleural air leak (bronchopleural fistula [BPF]) occurs during mechanical ventilation in two general circumstances: as a localized lung or airway lesion (*eg,* following trauma or surgery; complicating central line placement) and as a complication of diffuse lung disease (*e.g.,* ARDS, *Pneumocystis carinii* pneumonia). Although most leaks are physiologically insignificant, BPF can predispose to atelectasis or inadequate inflation of ipsilateral or contralateral lung, interfere with gas exchange, and predispose to pleural spread of infection. It can also prolong mechanical ventilation thus predisposing to additional morbidity. Some BPFs are amenable to direct surgical repair (*e.g.,* suture of bronchial tear; lobectomy for

necrotizing pneumonia), but in most instances, resolution of the BPF depends on resolution of the primary disease process.

Ventilatory support should provide adequate inflation for the uninvolved areas of lung and assure adequate gas exchange. No single ventilatory mode or approach has been shown to be more effective than any other in treating patients with BPF. In the presence of a large air leak and difficulty in maintaining adequate ventilation, a ventilator capable of delivering high inspiratory flow rates and large delivered tidal volumes may be required.

Use of independent lung ventilation (ILV) should be considered in the uncommon circumstance of inability to maintain contralateral lung inflation using adjustments of volumes and/or flows. Fortunately, ventilation of patients with BPF is usually adequate. The major problem is usually to facilitate closure of the leak, so that mechanical ventilator support may be withdrawn. In this circumstance, minimizing inflation pressures and tidal volume is the goal. Chest tube suction is necessary to evacuate continued gas leak, but the degree of suction exerts a variable effect on flow through the fistula.

Guidelines:

1. To facilitate closure
a. Use the lowest tidal volume that allows adequate ventilation.
b. Use a ventilatory mode and settings that minimize peak and plateau pressures necessary to maintain adequate ventilation.
c. Consider permissive hypercapnia (discussed under guidelines 3 and 4 under ARDS) to minimize inspiratory pressures and volumes.
d. Minimize PEEP

2. Consider independent lung ventilation (ILV) or high frequency jet ventilation (HFJV) in cases where a large air leak produces inability to inflate lung or failure to adequately oxygenate/ventilate.

Head Trauma: Mechanical hyperventilation to arterial carbon dioxide levels of 25 to 30 mm Hg acutely lowers ICP. Controlled data on the impact of hyperventilation in patients with head trauma are not available. Decreases in ICP do not necessarily reflect increases in cerebral perfusion pressure. Nevertheless, hyperventilation remains a mainstay of emergency therapy for acutely elevated ICP. There is no evidence to support the application of prophylactic hyperventilation in patients with head injury who do not have raised ICP.

In fact, there is a strong rationale for maintenance of normocarbia in most head-injured patients without elevated ICP because a patient who has prophylactically been hyperventilated to a $PaCO_2$ in the high 20s and who suffers an acute increase in ICP may not be expected to have an effective reduction in ICP following a moderate increase in minute ventilation ($\dot{V}E$). Since the

patient is already hyperventilated, a marked increase in $\dot{V}E$ may be required to effect a significant lowering of $PaCO_2$. The increase in mean airway pressure associated with a dramatic increase in $\dot{V}E$ may cause a paradoxic increase in ICP. Therefore, it is likely that stable head-injured patients should receive mechanical ventilatory support at a level sufficient to produce normal arterial blood gas values. Effective monitoring should be instituted to allow a rapid increase in ventilation and oxygenation should signs of increased ICP or hypoxemia occur.

Recommendations:

1. In the presence of increased ICP, maintain $PaCO_2$ 25 to 30 mm Hg or titrate to ICP if monitoring of ICP is available. Monitoring of ICP is desirable.

2. Maintain normocarbia in head-injured patients with normal ICP.

3. When hyperventilation is used to decrease ICP, return to normocarbia should be gradual (over 24 to 48 h).

Myocardial Ischemia and Congestive Heart Failure: In patients with myocardial ischemia, modes of mechanical ventilation that increase work of breathing will increase oxygen demand and may detrimentally affect the myocardial oxygen supply/demand relationship. Resultant myocardial ischemia may decrease compliance of the left ventricle (LV). The increasing pulmonary capillary pressure and decreasing lung compliance create a vicious cycle, as resistance and work of breathing increase further. Therefore, in patients with myocardial ischemia and some combination of high lung resistance and/or poor respiratory muscle function, spontaneous ventilation associated with an increase in the work of breathing is likely detrimental.

In severe congestive heart failure, positive pressure ventilation (PPV) would be expected to decrease venous return. Positive pressure ventilation is likely to increase PaO_2 by increasing lung volume and reducing right-to-left shunting (Qs/Qt). Although the effect of PPV on the normal ventricle would be to decrease cardiac output by decreasing LV filling (preload), the effect of PPV on a dilated failing LV operating on the flat (depressed) portion of the cardiac function curve will be different.[16,17] In this circumstance, a reduction in transmural aortic pressure and associated decrease in wall stress and afterload might increase stroke volume.

Guidelines:

1. In the presence of acute myocardial ischemia, choose modes of mechanical ventilation that minimize work of breathing.

2. When life-threatening hypoxemia accompanies severe congestive heart failure, consider the potentially beneficial effect of PPV on decreasing venous return and improving oxygenation.

3. Consideration should be given for assessment of the effect of PPV on hemodynamics.

Neuromuscular Disease: Patients with ventilatory failure due to neuromuscular disease (*eg,* Guillain-Barré syndrome; cervical spinal cord injury) typically have normal ventilatory drive and normal or nearly normal lung function. Because the primary physiologic defect is ventilatory muscle weakness, these patients are predisposed to develop atelectasis (from inadequate lung inflation) and pneumonia (from impaired cough and mucociliary clearance). Their main needs during ventilatory management are provision of adequate lung inflation and aggressive airway management. There is no evidence that either PPV or negative pressure ventilation is superior in this situation.

These individuals are at less risk for barotrauma than patients with intrinsic restrictive or obstructive lung disease, and they frequently prefer large tidal volumes (*eg,* 12 to 15 ml/kg). Typically, they are also more comfortable with high inspiratory flow rates. Whether full or partial ventilatory support should be provided depends on the patient's capabilities and the disease process: a patient with a high quadriplegia (e.*g.,* C1-2) lesion needs full ventilatory support whichever mode is used, whereas partial ventilatory support may be appropriate for individuals with some ventilatory capability, particularly during recovery, as long as patient comfort is maintained.

Guidelines:

1. Large tidal volumes (12 to 15 ml/kg) with or without PEEP (5 to 10 cm H_2O) may be needed to relieve dyspnea. Adjust peak flow rate as needed to satisfy patient inspiration needs ($\geq$60 L/min peak inspiratory flow will typically be required).

2. Use total or partial ventilatory support based on the patient's inherent ventilatory muscle strength.

Obstructive Airways Disease (OAD): Acute respiratory failure in patients with asthma and chronic OAD is associated with significant expiratory obstruction and hyperinflation. Resistance to inspiration is likely to be greater in the patient with asthma because of airway edema and mucus. Both patient groups benefit from mechanical ventilation settings that maximize expiratory time, thus decreasing end-expiratory lung volume (VEE), intrinsic (auto) PEEP, and the risk for hemodynamic compromise. With the same tidal volume, a higher VEE would produce a higher end-inspiratory lung volume (VEI) and a greater risk for barotrauma. The ideal level of PEEP is that which puts the majority of lung units on the favorable part of the pressure-volume curve (remember each lung unit has a different curve), maximizes gas exchange and minimizes over-distention over-distention.

The use of high inspiratory flow rates will maximize expiratory time and minimize VEE and intrinsic PEEP (auto-PEEP, dynamic hyperinflation). This is accomplished, however, at the expense of higher peak airway pressure (PAP) in the central airways.

Although PAP generated by increasing flow rate does not correlate closely with barotrauma as well as plateau pressure, the amount of central airway pressure that is actually transmitted to the alveolus (the actual risk factor for barotrauma) is difficult to judge. End-inspiratory plateau pressure with volume-cycled breaths rises as dynamic hyperinflation increases and may be reflective of increasing risk of barotrauma.

Guidelines:

1. No evidence exists that one ventilator mode is better than another for initial management of OAD. The clinician should choose a ventilator mode he or she is familiar with and has used successfully in this setting.

2. Adjust the peak inspiratory flow rate to meet patient demands.

3. Monitor for and minimize dynamic hyperinflation (auto-PEEP). See Section 4, A-6.
a. It is desirable to utilize the least $\dot{V}E$ that produces acceptable gas exchange and leads to the greatest expiratory time. Maneuvers likely to accomplish this goal are as follows:
i. Decrease in $\dot{V}E$
ii. Increase in expiratory time
iii. Acceptance of hypercapnia
b. When dynamic hyperinflation exists in the presence of patient-initiated mechanical ventilation, application of small amounts of ventilator-applied PEEP may be helpful in reducing the work of breathing (see Section 4, A-6). Application of ventilator PEEP above the level of initial auto-PEEP may lead to further hyperinflation and complications.

4. Based primarily on animal data and as discussed in the ARDS section, end-inspiratory plateau pressure is also a concern in OAD, as it reflects hyperinflation. We believe that attempts to maintain plateau pressure less than 35 cm H_2O are worthwhile even though the impact on patient outcome is unknown. In the acutely ill patient with OAD, measurement of plateau pressure usually requires sedation and paralysis.

5. Use of volume-cycled assist control ventilation in the initial treatment of the awake patient with OAD may be associated with significant risk of increasing hyperinflation and should be avoided.

Asthma: 1. We believe that a high plateau pressure is predictive of hyperinflation of lung units in the patient with asthma. A high PAP may also predict hyperinflation. We recommend that plateau and peak pressures be minimized in patients with asthma. Unfortunately, PAP is significantly influenced by endotracheal (ET) tube size and inspiratory flows.

2. Accept an elevated $PaCO_2$, as long as pH can be maintained at an acceptable level.

3. Paralysis and/or sedation may be necessary in some patients if the ventilation mode cannot be matched to the patient's needs (*i.e.,* patient "fighting" the ventilator). Following paralysis and/or sedation, a decrease in active expiratory effort may be associated with less airway collapse. The associated decrease in CO_2 production (especially of respiratory muscles) may also be advantageous in some circumstances. Paralysis is associated with acute and long-term complications.

COPD:

1. Mechanical ventilation of COPD patients with acute respiratory failure is unlikely to require high PAP or to present problems with CO_2 removal. In the presence of high PAP or difficulty in CO_2 removal in patients with COPD, coexisting abnormalities should be considered (pneumothorax, pulmonary edema, mucus plugging, high degree of bronchospasm).

2. Patients with chronic respiratory acidosis should have alveolar ventilation titrated to pH, not $PaCO_2$.

Postoperative Patients: Few patients require mechanical ventilatory support past the immediate postanesthetic period. However, residual anesthetic effects, usually due to narcotics or muscle relaxants, may require a variable period of mechanical ventilation. An anesthetic-induced decrease in FRC potentially coupled with thoracic or upper abdominal incision predisposes to atelectasis. These patients with little or no significant lung disease may be optimally treated with relatively little difficulty.

The greatest concern for the clinician is to avoid iatrogenic complications of ventilatory support, including infection, decreased cardiac output, prolonged support requiring unnecessary sedation, hyperventilation, inspissated secretions, and unnecessary exposure to potentially toxic high concentrations of inspired oxygen. Often, patients are unnecessarily ventilated in the postoperative period because of the mistaken belief that mechanical ventilation ***per se*** is beneficial in establishing a more physiologic cardiopulmonary status. Thus, patients who have undergone major operative procedures involving the head, thorax, or abdomen may remain intubated and ventilated unnecessarily.

Although prospective analysis has not determined whether such therapy has a significant rate of complication, to our knowledge, no prospective analysis has provided evidence of beneficial effects of such therapy. Future studies should be designed to determine which, if any, patients and/or surgical procedures require ventilatory support past the immediate anesthetic emergence period.

Unilateral Lung Disease:

Patients with unilateral lung disease who require mechanical ventilation are infrequently encountered. Therapeutic efforts have included placement of double-lumen tracheal tubes to effect ventilation of each lung separately, positional changes of the patient, bronchial blockers, pneumonectomy, and alteration of inspiratory gas flows in order to improve overall lung function. Usually, such maneuvers ignore the effect of hypoxic pulmonary vasoconstriction on the affected lung and the physiologic principles underlying inflation of the lung during PPV. Were it not for the restricting effect of the rib cage and diaphragm on lung inflation, the unaffected lung would receive the preponderance of the positive pressure breath and/or increase in lung volume secondary to application of CPAP.

However, in the presence of an intact thorax, lung inflation depends on increase in transpulmonary pressure, the difference between airway and intrapleural pressure. The individual with unilateral lung disease usually has a marked discrepancy in compliance between the two lungs. A lung with marked decrease in compliance will receive less volume for a given applied airway pressure. Therefore, the noncompliant lung will be ventilated less than will the normal, compliant lung. Should such patients experience significant arterial hypoxemia and/or hypercarbia, more aggressive means of ventilatory support must be considered, as with any type of advanced lung disease. These might include $ECCO_2R$, ECMO, high-frequency ventilation, etc. The ability of these interventions to alter outcome is not known. To date, split-lung ventilation with a double-lumen tracheal tube and differential application of positive airway pressure has likewise not been shown to improve morbidity and mortality.

Guidelines:

1. Initially utilize conventional ventilation techniques independent of presence of unilateral lung disease.

2. In the presence of difficulties in oxygenation, a trial of ventilation with the least involved lung in the dependent position is appropriate.

3. If PEEP is applied, initially utilize a single-lumen ET tube.

4. In cases of inability to oxygenate with traditional PEEP application, ILV with a double-lumen ET tube may be tried (synchronization of ventilation is not necessary). This mode of ventilation, however, has not been proved to alter outcome.

B. Discontinuation of Mechanical Ventilation

The consensus committee agreed there were many correct ways to discontinue patients from mechanical ventilation. There are a number of basic principles that are important in discontinuing ventilatory support and these are covered in Part 2 (Section 7). Specific recommendations are given below:

1. Whichever technique is used for discontinuation of ventilatory support, the clinician should know the signs of increasing ventilatory insufficiency and patient distress, and discontinue or modify the process if they appear, persist, or worsen:

(a) Increasing tachypnea: (*eg,* beyond a total rate of 30 to 35 breaths/min) associated with patient distress.

(b) Agitation, panic, diaphoresis, or tachycardia, unrelieved by reassurance and adjustment of the mechanical ventilation system.

(c) Acidemia: acute drop in pH to <7.25 to 7.30, associated with an increasing $PaCO_2$.

2. Excessive imposed work of breathing from demand valves or circuits, as indicated by substantial decreases in airway pressure during patient efforts, should be avoided during attempts at discontinuation of ventilatory support whether using a T tube or the ventilator circuit.

3. If intermittent mandatory ventilation (IMV) is used, the rate and degree of withdrawal of ventilatory support should be guided by pH, PCO_2, total respiratory rate, heart rate, and signs of patient distress. There is considerable difference between mandatory and spontaneous breaths.

4. If pressure support ventilation (PSV) is used:

(a) The rate of reduction of the PSV level should be guided by total respiratory rate rather than by tidal volume. As a rule, respiratory rate should not exceed 30 breaths/min.

(b) If a patient can maintain adequate gas exchange and comfort level on a low level of PSV (*eg,* 5 cm H_2O), it is not necessary to reduce this to zero before extubation.

5. In patients in whom ventilatory support cannot be withdrawn successfully over a short period, a systematic approach should be taken to identify and treat contributing factors, such as the following:

(a) *Imposed loads* in the apparatus that increase work of breathing (*eg,* demand valves, small-diameter ET tubes).

(b) *Respiratory factors* (*eg,* bronchospasm, excessive secretions, pharmacologic depression of ventilatory drive, persistence of underlying disease, etc).

(c) *Nonrespiratory factors* (*eg,* cardiovascular dysfunction, increased metabolic rate, acid-base problems, hypophosphatemia, malnutrition, anxiety, etc).

6. In “difficult-to-wean” patients in whom discontinuation of ventilatory support occurs gradually over several days, it may be desirable to increase the level of support at night to enable the patient to rest effectively, as demonstrated by the ability to sleep.

7. Successful extubation requires the ability to protect the upper airway and clear secretions adequately in addition to successful discontinuation of ventilatory support. These factors should be considered and addressed both prior and subsequent to extubation.

Underlying Principles

Section 4: Physiologic Principles Relevant to Mechanical Ventilation

A. Patient-Related Physiologic Principles

The response to mechanical ventilation is governed by several physiologic relationships. Two cardinal rules apply:

1. Although the qualitative response of a given physiologic variable to manipulation of ventilator settings may be predictable, the quantitative response is highly variable and patient specific. Thus, an increase in PEEP or level of ventilation usually improves PaO_2 at a given FIO_2. However, the extent of improvement in any given patient may be large or small, and the short-term improvement may produce complications due to the higher pressures (barotrauma). Likewise, an increase in $\dot{V}E$ may be expected to result in a lower $PaCO_2$, but the amount of reduction may be large or small and must be balanced against the potential complications relating to the increase in ventilatory pressures.

2. A ventilator manipulation designed to improve one relation or variable may have undesirable effects on other equally important relations or variables. For example, an increase in PEEP may improve PaO_2 but adversely affect cardiac output, thereby negating the improvement in PaO_2 at the tissue level. Likewise an increase in $\dot{V}E$ to reduce PCO_2 may result in greater auto-PEEP or adverse effects on cardiac output. The extent of negative side effects of a given ventilator manipulation is, again, highly variable and patient specific. The physiologic relations that are most important to consider are as follows:

1. *Ventilation Perfusion ($\dot{V}/\dot{Q}$ Relations):* The PaO_2 obtained at a given FIO_2 is a function of the uniformity (homogeneity) of distribution of ventilation and perfusion to different lung units. Units with relatively low ventilation and high perfusion are associated with incomplete oxygenation and, hence, a low PO_2 for a given FIO_2. Units with no ventilation at all but which continue to receive perfusion contribute to shunting, which is one extreme of $\dot{V}/\dot{Q}$ inequality.

The distribution of ventilation among different units depends on the following:

(a) Whether the unit is aerated or not at end expiration; units that are air free at end expiration (due to atelectasis or fluid filling) require very high inspiratory pressures if they are to receive any ventilation at all.

(b) For aerated units, the distribution of ventilation is determined principally by regional compliance and resistance. Lung disease is invariably nonuniform, and this is the basic reason for the existence of serious $\dot{V}/\dot{Q}$ mismatching and for difficulty in oxygenation. On theoretical grounds, when the regional differences in mechanics are principally in resistance, distribution of ventilation should become more uniform with long inspiratory times (TIS) and with decelerating flow patterns. Conversely, where the nonhomogeneity involves regional compliances, shorter TIS and square flow pattern should result in more uniformity. Whether equalizing regional ventilation is good or bad for $PaCO_2$ is not entirely predictable, since improving $\dot{V}E$ of a poorly ventilated unit may or may not be beneficial, depending on the state of perfusion of that unit. It is also evident that by rendering the $\dot{V}E$ distribution more uniform, some units (usually the healthy ones) will receive less ventilation as the poorly ventilated units receive more. If perfusion is preferentially distributed to the former, overall $\dot{V}/\dot{Q}$ may worsen as distribution of $\dot{V}E$ is improved.

The regional distribution of perfusion is determined principally by regional resistances in blood vessels supplying and draining different units. Regional resistances are related to gravity (dependent units have lower resistance), vasomotor tone, and mechanical factors (*e.g.,* lung volume and anatomic narrowing). Regional alveolar pressure (more appropriately, regional transpulmonary pressure) plays a critical role. When transmitted regional alveolar pressure is higher than pulmonary artery pressure (PAP), perfusion is arrested (West zone 1).

For a given degree of $\dot{V}/\dot{Q}$ mismatching and FIO_2, PaO_2 is critically affected by mixed venous O_2 saturation (SvO_2); lower venous O_2 saturation is associated with lower PaO_2; SvO_2 is, in turn, dependent on cardiac output, metabolic rate, and hemoglobin.

From the standpoint of oxygenation, the primary beneficial effect of increased distending pressure (PEEP or greater tidal volume VT) is the recruitment of nonfunctional or very poorly ventilated units. The benefit can be enhanced or mitigated through secondary and unpredictable effects on the following: (1) the distribution of ventilation to other units (by upward displacement of these units to more favorable or less favorable segments of pressure volume curve); (2) the distribution of perfusion (through effects on the relation between Palv and PAP and on lung volume [and hence vascular dimensions]); and (3) on mixed venous PO_2 (through effects on metabolic rate [less or more fighting, less or more work of breathing] or cardiac output).

2. *Relation Between Ventilation and $PaCO_2$:* The relation between minute ventilation ($\dot{V}E$) and $PaCO_2$ is:

$$PaCO_2 = 0.863\ \dot{V}CO_2/[\dot{V}E(1 - VD/VT)]$$

where $\dot{V}CO_2$ is CO_2 production in milliliters per minute STPD, a reflection of the metabolic activity of tissues, and VD/VT is equal to the tidal volume dead space ratio.

This equation emphasizes the fact that $PaCO_2$ is determined by the relationship between metabolic rate and ventilation, and not solely by the absolute level of $\dot{V}E$. The bracketed term reflects the fact that not all breathed gas ($\dot{V}E$) is useful for CO_2 exchange; only the fraction (1–VD/VT) is effective.

In normal subjects, much of the dead space (VD) is due to the volume of the conducting airways (anatomic VD). Since this volume changes little with VT, VD/VT tends to decrease as VT increases and VD/VT rarely exceeds 0.3 (*i.e.,* 30 percent of VT). In ventilated patients, particularly those with intrinsic lung disease, VD/VT can reach extremely high values (*eg,* 0.7 to 0.8) and VD is principally related to ventilated but poorly perfused lung regions (alveolar VD). In such cases VD/VT need not decrease with an increase in VT since the higher alveolar pressure required to generate the larger VT may increase alveolar VD (increasing the amount of zone 1, see above). Thus, quantitatively, the change in $PaCO_2$ as VT is increased may not be predictable. The response of PCO_2 to changes in $\dot{V}E$ is further confounded by possible effects of changes in ventilator settings on metabolic rate (see above equation). Thus, a patient may become more relaxed (and hence lower $\dot{V}CO_2$) or more agitated as ventilator settings are adjusted. Increases in VD/VT and $\dot{V}CO_2$ should be considered whenever a change in $\dot{V}E$ does not result in the expected change in $PaCO_2$ or whenever a large $\dot{V}E$ is required to maintain a reasonable $PaCO_2$.

The above equation also emphasizes the effectiveness of accepting a higher $PaCO_2$ (permissive hypercapnia) or of lowering metabolic rate (*eg,* by sedation, paralysis) in reducing the required level of ventilation. A lower ventilation is usually associated with lower distending pressures. To the extent that high distending pressures contribute to barotrauma and negative hemodynamic consequences, permissive hypercapnia and reductions in metabolic rate may help reduce the complications of ventilatory support.

3. *Thoracic Pressures and Cardiovascular Function:* Blood returns to the thorax along a pressure gradient from peripheral vessels to the right atrium (RA). To the extent that intrathoracic pressure affects RA pressure, it may alter the gradient for venous return. Since cardiac output cannot be different from venous return, an increase in intrathoracic pressure will, all else being equal, tend to reduce cardiac output. This effect is enhanced in the presence of hypovolemia.

Right ventricular (RV) output can also be affected by changes in RV afterload. The latter is affected in a complex way by lung volume; an increase in lung volume tends to increase the resistance of alveolar vessels while decreasing the resistance of extra-alveolar vessels. The net effect on total resistance is unpredictable. Changes in RV afterload can aggravate or minimize the effect of changes in intrathoracic pressure on RA pressure.

Changes in intrathoracic pressure also affect LV function; a higher intrathoracic pressure acts to reduce LV afterload. Where poor LV function is limiting cardiac output, an increase in intrathoracic pressure may result in better LV emptying, with secondary consequences on RV afterload (decrease) and venous return (increase). Changes in lung volume may also reflexly affect peripheral vascular tone.

It is clear that effects of changes in ventilator settings on hemodynamics are complex. However, in general, cardiac output is adversely affected by increases in intrathoracic pressure; the actual response varies.

4. *Tissue Oxygenation:* One of the main objectives of ventilatory support is to ensure that tissues are provided with their O_2 requirements. The rate at which O_2 is delivered to the tissues (O_2 delivery [$\dot{D}O_2$]) is a function of cardiac output ($\dot{Q}T$), hemoglobin (Hgb), and O_2 saturation (SaO_2). Thus:

$$\dot{D}O_2 = 1.39\ Hgb \infty SaO_2 \infty Qt + 0.003 \infty PaO_2$$

DO_2 represents the theoretical maximum for O_2 consumption by the tissues. In practice, tissues cannot extract all the delivered oxygen. As DO_2 is reduced, the tissues are capable of increasing the fraction extracted such that their O_2 needs are met. A point is reached, however, where the fraction of O_2 that can be extracted reaches a maximum level. As O_2 delivery decreases below this critical level, the O_2 needs of the tissues cannot be met. This state (O_2 extracted < amount warranted by metabolic activity) may ultimately result in tissue damage and can, theoretically, account for multisystem failure in critically ill patients.

This critical level of O_2 delivery is related to the metabolic activity of tissues (the higher the activity, the higher the critical $\dot{D}O_2$) and to the maximum fraction that tissues can extract from delivered O_2. In disease, metabolic rate (*i.e.,* $\dot{V}O_2$) is often high. There is also evidence that in some clinical states (notably sepsis), the maximum fraction that can be extracted is reduced. Both factors tend to raise critical $\dot{D}O_2$.

Mechanical ventilation affects two of the main determinants of $\dot{D}O_2$, namely SaO_2 and $\dot{Q}T$. Often, ventilator measures that are aimed at increasing one variable cause an opposite change in the other (*eg,* PEEP may improve SaO_2 but concomitantly may reduce $\dot{Q}T$). Where maintaining a reasonable PaO_2 (*eg,* >60 mm Hg) requires PEEP in excess of 10 cm H_2O, it is important to consider whether the net effect (after allowing for possible adverse effects on $\dot{Q}T$) is beneficial (*i.e.,* increase in $\dot{D}O_2$), whether the remaining benefit, if any, warrants the extra risk of high PEEP or FIO_2, and whether the same improvements in $\dot{D}O_2$ /$\dot{V}O_2$ relation may not be accomplished less dangerously through reduction in metabolic rate or by increasing hemoglobin.

5. *Respiratory Mechanics:* The pressure required to produce a given tidal volume (VT) in a given time (TI=inspiratory time) is a function of the elastic and resistive properties of the respiratory system. The elastic properties are defined by the static pressure-volume (P-V) relation. This relation is sigmoidal with the system being most compliant (lowest elastance) in the midvolume range and becoming substantially stiffer near the upper (total lung capacity [TLC]) and lower volume extremes. Normally, end-expiratory volume is at about 40 percent of vital capacity (VC), and tidal changes in volume occur in the middle, most compliant, range. In ventilated patients, the VT may be located near the upper or lower extremes of the P-V curve where the system is naturally stiff. The former situation—VT encroaching on TLC—occurs under two conditions: (1) when VC is extremely small as a result of severe intrinsic lung disease; here, the flat portion of the P-V curve might lie within the target VT; and (2) in the presence of high PEEP (external or intrinsic) which increases FRC.

Tidal volume occurs in the stiff range near residual volume (RV) under two conditions: (1) with obesity and abdominal distention which force end-expiratory volume (the starting position for the next breath) to be in the low range of VC. In this case, the increased stiffness is partially related to chest wall stiffness and partially to alveolar collapse. A variable portion of the applied pressure is, therefore, dissipated across the chest wall and not the lung (*i.e.,* less likelihood of barotrauma for the same distending pressure); and (2) when airway or alveolar closure occurs at higher than normal volumes. In this situation, airway closure may occur within the VT range, and this derecruitment causes the lung to appear stiffer.

When measured compliance (VT/[plateau pressure minus end-expiratory pressure (total PEEP)]) is too low, it is important to ascertain whether this is in part due to the VT cycling near one of the volume extremes and, if so, whether cycling is occurring near TLC or RV. Ventilator "cycling" refers to the mechanism by which the phase of the breath switches from inspiration to expiration. This has important implications both in terms of identifying the underlying pathophysiology (is the increased stiffness due to structural changes) and in defining the ventilator strategy to be used to minimize barotrauma. This distinction can be made with simple manipulations of ventilator output while monitoring airway pressure.

One simple approach is to decrease VT for one to two breaths and then assess compliance with the smaller VT. An increase in compliance as VT decreases suggests that lung volume is near the stiff upper part of the P-V curve. An unchanged compliance indicates that VT is cycling in the linear midrange. Conversely, if compliance decreases as VT is lowered, volume is likely cycling near the stiff lower range of the P-V curve. In this case, addition of PEEP should improve the operating compliance (by raising end-expiratory volume toward the more compliant range). This should be helpful, particularly during weaning.

The conventionally measured compliance reflects the elastic properties of both chest wall and lung. A low measured compliance may be due to stiff lungs and/or a stiff chest wall or to as small fraction of the lung being ventilated (*eg,* ARDS). Increased lung stiffness is the predominant mechanism with intrinsic lung disease, with auto-PEEP, and where there is airway or alveolar closure in the VT range (see above). Increased chest wall stiffness may be the predominant cause of decreased compliance with primary chest wall disease (*e.g.,* kyphoscoliosis) or where obesity or abdominal distention causes VT to cycle near RV where the chest wall is naturally stiff (see above).

Determination of the contribution of lung and chest wall to decreased compliance is possible only by concurrently (with airway pressure [Paw]) estimating pleural pressure using an esophageal catheter. Thus, if Paw during an inspiratory hold is 40 cm H_2O higher than end-expiratory pressure (PEEP) while the corresponding value from pleural pressures (Ppl) (*i.e.,* Ppl during plateau minus Ppl at end expiration) is 30 cm H_2O, then the lung contributes a quarter of the stiffness while the chest wall contributes three quarters, and so on. Where increased lung stiffness is the major reason for decreased compliance, the lung receives the brunt of the distending pressure and, all else being the same, the risk of barotrauma will theoretically be greater.

The other component to the distending pressure is that related to resistance. In ventilated patients, total resistance is made up of two components, ET tube resistance and resistance of the patient's airways. In many cases, ET tube resistance is the major component of total resistance. The resistance of the ET tube is not constant, but increases with flow rate. With the exception of obstructive diseases, the patient's resistance normalized for lung volume (specific resistance) is normally very small. In obstructive diseases, resistance is high and is often volume dependent, being higher at low lung volume. It should be remembered that the measured resistance value is determined by the size of the aerated compartment; thus, in ARDS (a condition in which the aerated capacity may be only one third of normal), the measured resistance value may be high, while the specific resistance is normal or low.

Whereas total elastance and total resistance determine the total distending pressure required to attain a given V_T in a given T_I, regional differences in respiratory mechanics can importantly influence the distribution of inhaled volume within the lungs. This may result in some lung regions becoming relatively overdistended even though total V_T may be reasonable. The regional distribution of V_T is affected in a complex way by the underlying reason for nonhomogeneity (*i.e.,* regional differences in resistance or compliance), T_I, and flow pattern.

The total distending pressure applied at any instant (Ptot) is the sum of pressure applied to overcome elastic recoil (Pel, a function of volume above passive FRC and the P-V curve), and the pressure applied to overcome resistive elements (Pres, a function of flow rate and resistance):

$$Ptot=Pel+Pres$$

In paralyzed or apneic patients Ptot is entirely supplied by the ventilator and Paw=Ptot. When the elastic and resistive properties are known, Ptot can be estimated. Any difference between Paw and Ptot is a reflection of the pressure generated by the patient. This approach can thus be utilized to assess the extent of patient effort and, hence, adequacy of ventilatory support.

6. *Dynamic Hyperinflation and Auto-PEEP:* Dynamic hyperinflation (DH) is defined as failure of lung volume to return to passive FRC (volume at which elastic recoil equals external PEEP) prior to the onset of the next inspiration. Whenever this happens, alveolar pressure remains higher than external PEEP throughout expiration, and unless airways completely collapse, expiratory flow continues until the onset of the next inspiration. Hysteresis is the tendency of the lungs, due to surfactant , to exist at higher volumes in expiration than in inspiration.

Auto-PEEP is the difference between alveolar pressure and external airway pressure at end expiration. A difference (*i.e.,* auto-PEEP) will always exist whenever expiratory flow continues until the end of expiration (this is the gradient for flow). In the passive patient, such a gradient can only result from dynamic hyperinflation since alveolar pressure in this case reflects only passive elastic recoil; a gradient thus means lung volume did not return to passive FRC. In the patient with active expiratory muscles, alveolar pressure is also affected by the pressure generated by expiratory muscles. A gradient (auto-PEEP) can therefore exist even though volume is at, or even below, passive FRC. Auto-PEEP in the active patient does not necessarily

signify the presence of DH and its magnitude is not an index of the magnitude of DH. This is important to recognize, since the physiologic consequences and management of auto-PEEP due to dynamic hyperinflation and to expiratory activity differ (see below). Expiratory muscle activity frequently exists in the presence of high respiratory drive and/or high expiratory resistance.

Dynamic hyperinflation develops in the setting of high expiratory resistance or expiratory flow limitation and is influenced by the compliance of the respiratory system, the volume from which exhalation begins, and the expiratory time. In ventilated patients, the delay in emptying may be patient related (obstructive diseases) or, very commonly, may be due to "plumbing" problems (narrow ET tube, kinking or water clogging of exhalation tube, poor exhalation valve, etc). Measurement of Paw during expiration helps to distinguish between problems intrinsic in the patient and ET tube, from problems within the external circuit (high Paw during exhalation points to the latter causes).

Auto-PEEP (DH, air trapping, intrinsic PEEP) has been described in many conditions (*e.g.,* COPD, asthma, ARDS) and can occur whenever $\dot{V}E$ is relatively high. Dynamic hyperinflation most commonly occurs, however, in the setting of severe airflow obstruction. Here, the ventilation requirements may be modest, but expiratory resistance is often severalfold greater than its inspiratory counterpart.

The consequences of DH are related to the associated changes in lung volume and pleural pressure. (1) It causes V_T to cycle closer to TLC where compliance is low (see above). More distending pressure is required to attain the same V_T. (2) It interferes with triggering in the assisted mechanical ventilation or pressure support modes; the patient has to generate enough pressure to offset auto-PEEP plus trigger sensitivity before triggering occurs. (3) Because of 1 and 2, it increases the work of breathing during weaning attempts. (4) It affects hemodynamics in a manner similar to external PEEP. (5) It can cause overestimation of the pressure difference required for tidal ventilation and subsequent underestimation of the true compliance of the respiratory system.

Auto-PEEP in the absence of DH (*i.e.,* end-expiratory lung volume at or below passive FRC) does not cause V_T to cycle near TLC (in fact, it may have an opposite effect by reducing end-expiratory lung volume below passive FRC), has little effect on triggering, does not increase work of inspiratory muscles (in fact, it may spare inspiratory muscle work through sharing of total work between inspiratory and expiratory muscles), and does not result in underestimation of true compliance. In fact, where volume begins below passive FRC due to expiratory muscle activity, the opposite (overestimation of compliance) may occur. Administration of external PEEP under these circumstances serves no purpose and may make it more difficult for expiratory muscles to reduce lung volume.

In the passive state, and for a given degree of expiratory obstruction, the two variables that are most critical in determining extent of DH are total $\dot{V}E$ and the inspiratory/expiratory (I:E) ratio. A higher $\dot{V}E$ will cause more DH whether the high $\dot{V}E$ is the result of a large V_T or high f. In the former case (larger V_T), more time is required to return lung volume to the passive FRC, whereas in the latter case (high f), less time is available to empty the same V_T. It is for this

reason that reduction in $\dot{V}E$ (through permissive hypercapnia or reduction in ventilatory demand) is one of the most effective ways of reducing DH. At a given f, the I:E ratio determines the time for delivery of the VT. Higher values tend to increase auto-PEEP.

Measurement: Auto-PEEP should be suspected in all patients with airways obstruction or whenever the flow tracing demonstrates persistent flow at end exhalation. During *passive* ventilation (patient is not making respiratory efforts), auto-PEEP can be measured by comparing the end-expiratory airway occlusion pressure (easily measured in only a few currently available ventilators) with the set level of PEEP or by observing the amount of positive airway pressure required to initiate inspiratory flow. A helpful method for executing end-expiratory port occlusion in the passive patient uses the ventilator's inflation onset as the timing mechanism with a Braschi valve. Auto-PEEP can also be measured using plateau pressures. Operationally, plateau pressure is first recorded during a single cycle of volume-controlled ventilation at the usual ventilating frequency. (To avoid further hyperinflation, the end-inspiratory pause should not be applied for more than a single cycle.) Inflation is then prevented for approximately 20 s by a marked reduction in frequency, after which a single end-inspiratory plateau pressure is remeasured. The difference in plateau pressures is auto-PEEP.

Another similar method is first to measure the additional (dynamically trapped) volume released when a single routine tidal inflation is delayed by 20 to 30 s, and then to divide the measured trapped volume by respiratory system compliance. Under passive conditions, compliance is perhaps best judged by dividing the difference in static end-inspiratory ("plateau") pressures observed at two distinctly different VT into that volume difference. This, however, is valid only if compliance is constant throughout VT (*i.e.,* VT is fully within the linear segment of the P-V curve).

In a passively ventilated patient, the effect of PEEP on lung and chest volumes can be accurately assessed by observing the peak dynamic or static airway pressures. Failure of these pressures to rise in response to adding PEEP indicates dynamic airway compression, flow limitation, and potential benefit to the addition of PEEP.

Finally, when auto-PEEP results from dynamic airway compression, the least PEEP increment required to evoke a detectable increase in lung volume or peak cycling pressure is sometimes considered to be the pressure required to counterbalance the original level of auto-PEEP. This technique is invalid, however, when expiration is not flow limited (*i.e.,* when DH is due to a simple increase in resistance and not to flow limitation). Moreover, the applied pressure needed to counterbalance the "critical" pressure approximates only 75 percent to 85 percent of the auto-PEEP determined by expiratory port occlusion.

In patients with active respiratory efforts (*i.e.,* assist mode, PSV, etc) auto-PEEP can not easily be approximated by the end-expiratory occlusion, since expiratory muscle activity can influence the measured value. It can also be estimated as the esophageal pressure deflection required to initiate inspiration or to terminate expiratory flow. Interestingly, auto-PEEP estimated by this method is usually less than that measured by end-expiratory port occlusion. Although the reason for this disparity remains unclear, it has been argued that gas begins to flow into the lung when auto-PEEP in the least affected units has been overcome.

As indicated above, the auto-PEEP measured in patients with active respiratory efforts need not reflect dynamic hyperinflation. To our knowledge, there are currently no accepted methods of assessing the extent of DH in patients with active respiratory efforts. The use of external PEEP in these cases should be based more on clinical response to graded external PEEP (*i.e.,* more or less distress) than on the measured value of auto-PEEP.

7. *Respiratory Muscle Output and Endurance:* Laboratory studies have shown that inspiratory muscles fatigue when forced to generate pressures in excess of critical levels.[35] The critical pressure output above which fatigue occurs is a function of inspiratory muscle strength (*eg,* maximum inspiratory pressure). It is not clear whether fatigue occurs outside the laboratory setting, where inspiratory muscle output is spontaneously selected by the patient and not imposed (as in the laboratory). Nevertheless, the laboratory results point out the limited capacity of inspiratory muscles to sustain relatively high pressure outputs.

It is very difficult to assess the potential for fatigue in the ventilated patient. Whereas actual muscle output can be estimated through measurement of work of breathing, the pressure generated by muscle contraction or Pmus (see above), the denominator (maximum possible output) is difficult to determine. Furthermore, the critical fractions developed in the laboratory (*eg,* tension-time index >0.15) need not apply in the critically ill patient. So far, the best indication of whether the patient's muscles are overstressed remains the clinical impression of respiratory distress.

8. *Control of Breathing:* There is tremendous interindividual variability in the level of ventilation desired by patients (*i.e.,* ventilatory demand). The range extends from a few liters per minute (as in patients with chronic CO_2 retention) to >30 L/min (*eg,* in sepsis). High levels of ventilatory demand are related to high metabolic rate (muscle activity, fever), excessive V_D/V_T, or to a lower CO_2 set point where the patient targets a subnormal P_{CO_2} (metabolic acidosis, neural reflexes, central problems). In the presence of high $\dot{V}_E$ demand, high $\dot{V}_E$ output by the ventilator is required in order for the patient to feel comfortable. In turn, high $\dot{V}_E$ output by the ventilator translates into greater distending pressure (more volume and flow) and greater tendency for auto-PEEP.

In patients with high ventilatory demands, every effort should be made to identify the mechanism and, if possible, correct it. Should the distending pressures required to maintain comfort remain excessive, forced reduction in ventilatory drive through sedation and, if necessary, paralysis (to reduce metabolic rate) may be appropriate. At present, there are insufficient data to indicate the precise pressures (or volumes) that must be avoided (see complications, Section 5-B).

B. Ventilator-Related Physiologic Principles

During spontaneous unassisted breathing, contraction of the diaphragm and other accessory muscles of inspiration results in a decrease in intrathoracic pressure, followed by a corresponding decrease in alveolar and airway pressures. These decreased pressures cause an increase in thoracic volume and the movement of a V_T into the lungs. Relaxation of ventilatory muscles returns these pressures and volumes to their resting levels. That is, the elastic recoil of the

thoracic cage and of the lung increases intrathoracic pressure, causing an increase in alveolar and airway pressure, allowing exhalation of the V_T. During mechanical ventilatory assistance, the magnitude and the direction of these pressures may be grossly altered with potential adverse effects as outlined elsewhere in this text. As a result, the appropriate selection of gas delivery settings and monitoring of system pressures is likely important.

1. *Ventilator Settings:*

(a) *Volume.* In volume-targeted (*i.e.,* volume-cycled) ventilation, a machine-delivered V_T is set to be consistent with adequate gas exchange and patient comfort. The V_T selected in adults normally varies from about 5 to 15 ml/kg of body weight. Numerous factors, such as lung/thorax compliance, system resistance, compressible volume loss, oxygenation, ventilation, and barotrauma, are considered when volumes are selected. Of critical importance is the avoidance of localized overdistention. This can generally be accomplished by selecting V_T that remain on the steep aspect of the P-V curve of the patient-ventilator system and by ensuring that peak airway and alveolar pressures do not exceed a maximum target. Although controversy exists regarding specific target levels, many would agree that a peak alveolar pressure greater than 35 cm H_2O raises concern regarding the development of barotrauma and ventilator-induced lung injury increases. With pressure-targeted (in pressure-limited) ventilation, delivered V_T varies depending on target pressure selected, system impedance, and the patient's spontaneous ventilatory pattern.

(ii) *Respiratory Rate.* Setting of mandatory ventilator gas delivery rate is dependent on the mode of ventilation selected, the delivered V_T, dead space to tidal volume ratio, metabolic rate, target $PaCO_2$ level, and level of spontaneous ventilation. With adults, set mandatory rate normally varies between 4 and 20/min, with most clinically stable patients requiring mandatory rates in the 8 to 12/min range; in patients with either acute or chronic restrictive lung disease, mandatory rates exceeding 20/min may be necessary, depending on desired $\dot{V}_E$ and the targeted $PaCO_2$. Along with $PaCO_2$, pH, and comfort, the primary variable controlling the selection of mandatory rate is the development of air trapping and auto-PEEP. As with the selection of most ventilator settings, development of air trapping should be avoided because of its effect on $\dot{V}/\dot{Q}$ matching, work of breathing, and barotrauma.

(iii) *Flow Rate.* The selection of peak inspiratory flow rate during volume-targeted ventilation is primarily determined by the level of spontaneous inspiratory effort. The inspiratory flow rate determines how quickly the breath is delivered. The time required to complete inspiration is determined by the tidal volume delivered and the flow rate: Ti = VT/Flow Rate.

In patients triggering volume-targeted breaths, patient effort, work of breathing, and patient ventilator synchrony depends on the selection of peak inspiratory flow. Peak inspiratory flows should ideally match patient peak inspiratory demands. This normally requires peak flows to be set at 40 to 100 L/min, depending on $\dot{V}_E$ and drive to breathe. During controlled ventilation, peak flows may be set lower than 40 L/min in order to establish a specific T_I. With pressure-targeted ventilation, the peak inspiratory flow is determined by the interaction of the set pressure, respiratory resistance, and patient effort. The specifics of how *quickly* the pressure target is reached is defined by the manufacturer.

(iv) *Inspiratory Time/I:E Ratio.* The selection of a specific TI and I:E ratio is generally based on hemodynamic response to ventilation, oxygenation status, and level of spontaneous breathing. In spontaneously breathing patients, gas delivery should be coordinated with patient inspiratory effort to ensure synchrony. This normally requires about 0.8 to 1.2 s and an I:E of about 1:2 to 1:1.5. During controlled mechanical ventilation, TI or I:E ratios may be lengthened in order to elevate mean airway pressure (MAP) and enhance oxygenation. When lengthening TI and I:E ratios, the impact of these alterations on the cardiovascular system must be carefully monitored. The primary factors limiting increases in both TI and I:E ratios are patient discomfort, the need for sedation, the development of auto-PEEP, and hemodynamic compromise.

(v) *Flow Profile.* Few data identifying differing physiologic responses to inspiratory flow profiles are available when adjusted for the same VT and TI/TTOT.[45,46] Essentially no differences exist among square, decelerating, and sine wave delivery profiles in terms of gas exchange or work of breathing, and these approaches appear to be equally acceptable in the majority of patients requiring ventilatory support, provided that the mean flow rate is adequate. To our knowledge, no data supporting the use of an accelerating flow pattern are currently available. Selection of flow profile is available only in volume-targeted approaches to ventilation. With all pressure-targeted modes, an exponentially decelerating pattern is normally established as the ventilator attempts to rapidly achieve the pressure target set and to maintain the target constant throughout the inspiratory phase.

(vi) *Sensitivity.* Since mechanical ventilators and artificial airways impose a resistive load on the spontaneously breathing ventilator-assisted patient, ventilator-trigger sensitivity should be set at the most sensitive level that prevents self-cycling. Generally, this is –0.5 to –1.5 cm H_2O. Recently introduced flow cycling systems are generally more efficient than pressure cycling approaches, but the clinical significance of this is unclear. These systems should also be set at maximum sensitivity (1 to 3 L/min).

(vii) *FIO_2.* The selection of the FIO_2 is dependent on the target PaO_2, PEEP level, MAP, and hemodynamic status. In general, as a result of concerns regarding the effect of high FIO_2 s on lung injury, the lowest acceptable FIO_2 should be selected. However, the effect of FIO_2 on lung injury must be balanced by the effect of airway and alveolar pressures on lung injury. In those patients who are most difficult to oxygenate, FIO_2 can be minimized by optimizing PEEP and MAP, by deep sedation with or without pharmacologic paralysis, and by lowering the minimally acceptable SaO_2 to <90 percent while ensuring adequate cardiac output.

(viii) *PEEP.* Positive end-expiratory pressure is applied to recruit lung volume, elevate MAP, and improve oxygenation. PEEP may decrease venous return and preload of the LV, as well as decreasing the triggering work of breathing caused by auto-PEEP. The optimal level of PEEP depends on the desired physiologic response. In ARDS, PEEP level is established in conjunction with FIO_2 and TI settings to establish a target PaO_2 (SaO_2) or O_2 delivery. Although it is difficult to identify an upper limit for PEEP in this setting, most would agree that the lower limit should be at or above the lower inflection point on the P-V curve in the early phase of acute lung injury. This is generally a PEEP level of about 8 to 12 cm H_2O. As with any pressure, avoidance of high PEEP levels is desirable.

2. *Pressure Measurements:* During the delivery of a positive pressure breath, system pressure can be measured in a number of locations (internal to the ventilator, at the airway opening, and at the carina). The farther away the measurement is from the alveoli, the greater the possible difference from actual alveolar pressure. During patient triggering, alveolar pressure is more negative than carinal pressure, which is more negative than airway opening pressures, which are more negative than internal ventilator pressures. Because of resistance to gas flow during a positive pressure breath, pressure measured internal to the ventilator is greater than airway opening pressure, which is greater than carinal pressure, which is greater than alveolar pressure.[52] Pressure measured at all of these locations is only equal during periods of zero flow.

(i) *Peak.* Peak pressure is the maximum pressure obtainable during active gas delivery. In volume-targeted ventilation, peak pressure is dependent on both compliance and airways resistance, as well as V_T, peak flow, and flow pattern. For a given compliance and airway resistance, higher peak flow results in higher PAP. Generally, with all other variables equal, an accelerating flow profile results in a higher PAP than any other profile, since the highest flows are delivered with this pattern at end inspiration. With pressure-targeted ventilation, the peak inspiratory pressure is approximately equal to the target pressure. However, because of the high initial flow and the decelerating flow pattern in pressure-targeted ventilation, the initial system pressure may exceed the pressure target by about 1 to 3 cm H_2O.

(ii) *Plateau.* This is normally defined as the end-inspiratory pressure during a period of at least 0.5 s of zero gas flow. It should be measured on the first breath after the setting of an inflation hold and requires passive ventilation. The plateau pressure is the pressure required to counter-balance end inspiratory forces and roughly approximates the average peak alveolar pressure. With pressure-targeted ventilation, the pressure target approximates to the plateau (alveolar) pressure if a period (0.5 s) of zero delivered gas flow is observable.

(iii) *Mean.* The system pressure averaged over the entire ventilatory period is defined as the MAP. Because expiratory resistance usually exceeds inspiratory resistance, MAP as displayed on monitoring devices almost always underestimates mean alveolar pressure (MalvP) to some extent. The MalvP can be estimated from the MAP by the following formula:

$$MalvP=(\dot{V}E/60)(R_E–R_I)+MAP$$

Where R_I and R_E are inspiratory and expiratory resistances, respectively. Provided sufficient PEEP is applied to seek out recruitable lung units, oxygenation and MAP demonstrate a predictable and quantifiable direct relationship.

(iv) *End-Expiratory.* This is the airway pressure at the termination of the expiratory phase, normally equal to atmospheric or the applied PEEP level. However, in patients with prolonged expiration or short expiratory times, end-expiratory alveolar pressure may be further elevated as a result of the development of auto-PEEP. Alveolar and airway pressures are not the same unless periods of no-flow are established. That is, end-inspiratory airway pressure normally exceeds alveolar pressure because of resistance to gas flow, whereas end-expiratory alveolar pressure may exceed airway pressure because of the development of auto-PEEP and MalvP always exceeds MAP.

3. *Machine Problems:*

(i) *Demand Valves.* In all assisted modes of ventilation, the patient must activate gas delivery. This requires a pressure differential sufficient to trigger the ventilator. Once triggering occurs, sufficient gas flow must be provided to meet inspiratory demand. Both of these processes impose work on the patient. The amount of effort required to activate any given ventilator varies greatly and is generally greater with pressure triggering than flow triggering. The addition of PEEP or CPAP may increase imposed work because of the adjustments the ventilator must make to maintain PEEP/CPAP during spontaneous breathing. Although the work imposed by these systems is generally minimal, total imposed work of breathing can be significant when demand valves are considered in series with the work imposed by ET tubes and humidifier systems.

(ii) *Humidifiers.* Three different types of humidifiers are used during mechanical ventilation: bubble-through, passover, and artificial noses. Of these, the passover humidifier is the only one that does not have the potential to impose added work of breathing. Bubble-through humidifiers have minimal effect on imposed work of breathing if the machine sensing of patient effort is on the expiratory side of the circuit or at the circuit "Y." However, if patient effort is sensed on the inspiratory side, imposed work of breathing may be markedly increased because of the need to create a pressure gradient across the humidifier.

Artificial noses represent a resistance load placed in series with the ET tube and demand valve. Their effect on work of breathing is dependent on length of use, patient ventilatory drive, and design of the valve. In general, artificial noses should not be used in patients with marked ventilatory muscle dysfunction, particularly if small-sized ET tubes are used and if the development of auto-PEEP is an ongoing issue. Regardless of humidifier used, it should be able to establish at the carina a temperature of 30° to 32°C with an absolute humidity of 30 mg.

(iii) *Apnea Ventilation.* Many ventilators do not incorporate backup apnea ventilation during pressure support or CPAP breathing. As a result, careful setting of low respiratory rate, low V_T, and low minute volume alarms are critical for the safe application of these modes of ventilation. In ventilators where apnea ventilation is available, apnea of definable time periods results in the provision of a backup control mode volume-targeted approach to ventilation. Resumption of spontaneous breathing or practitioner intervention re-establishes the original ventilatory mode.

C. Patient-Ventilator Interactions

The use of a mechanical ventilator often superimposes a clinician-selected pattern of ventilation on the patient's natural breathing rhythm. A mechanical ventilator is a machine that generates a controlled flow of gas into a patient's airways.

Under these circumstances, certain interactions between patient and ventilator will occur. These fall into two categories: (1) the response of the mechanical breath delivery system to patient efforts, and (2) the response of patient efforts to ventilator settings.

1. *Response of the Breath Delivery System to Patient Efforts:* If the ventilatory demands of the patient do not coincide with the quantity (or quality) of ventilation provided by the ventilator, patient-ventilator dyssynchrony can impose an inspiratory muscle load. This, in turn, leads to increased oxygen consumption of the respiratory muscles and patient discomfort, which is diagnosed as the patient "fighting" the ventilator. This detrimental patient-ventilator interaction may be due to the patient's "inappropriately" high ventilatory drive or to inappropriate ventilator settings or circuits.
2. If the clinician determines that this problem is due to "inappropriate" patient demands, it may be appropriate to use sedation or paralysis to eliminate patient respiratory effort. Conversely, the problem may relate to the ventilatory mode or ventilator setup being used. These detrimental interactions between patient and ventilator can occur during any of the following phases of breath delivery.

(i) *Triggering.* Triggering is the initiation of gas delivery. Significant imposed ventilatory muscle loads can be imposed by insensitive or unresponsive triggering systems. In addition, the presence of auto-PEEP, narrow ET tubes, obstructed airways, and stiff parenchyma will serve to magnify the insensitivity or unresponsiveness of the triggering system. Attempts to maximize trigger sensitivity and responsiveness through demand valve adjustments or through the counterbalancing of auto-PEEP with applied PEEP are appropriate. Unfortunately, oversensitive valves can result in spontaneous ventilator cycling independent of patient effort.

(ii) *Gas Delivery.* Gas flow from the ventilator is governed (or limited) by a set flow (flow limited) or set pressure (pressure limited) on most ventilators. Any patient effort during flow-limited breaths will only result in a decrease in airway pressure since additional flow above what is set is not available. This can produce a significant imposed load on the ventilatory muscles. In contrast, any patient effort during a pressure-limited breath will result in an increase in flow but no change in airway pressure. A patient's flow demands are thus theoretically more readily met by a pressure-limited breath strategy. The capability of adjusting the rate of rise of airway pressure during pressure-limited breaths further enhances control of this variable.

(iii) *Cycling.* On current systems, gas delivery can be terminated by set volume, set time, or set flow. With volume or time cycling, continued patient effort is ignored by the machine and this can lead to the patients pulling against a closed inspiratory flow valve. Conversely, active expiratory efforts by the patient with volume or time cycling result in elevation in airway pressure that can result in automatic breath termination in association with a high pressure alarm limit. Flow cycling gives the patient more control than breath cycling.

Usually flow cycling is set to occur at 25 percent of peak flow. Increasing patient effort can thus delay cycling such that more gas is delivered. Conversely, decreasing efforts (or even initiation of expiratory efforts) cause these flow criteria to be met earlier and thus a smaller volume of gas is delivered over a shorter period. Synchrony between end of patient effort and end of pressure limiting is, however, not assured even with flow cycling. Depending on the strength of patient efforts vs. set pressure on respiratory mechanics and on the flow level at which cycling occurs, pressure may continue beyond the patient's inspiratory effort (thereby interfering with expiration) or may terminate prematurely before end of patient effort.

Response of Patient Efforts to Ventilator Settings: Ventilator settings, such as V_T, flow rate, and pattern with flow-limited volume-cycled breaths, or the pressure level and rate of pressure rise with pressure-limited breaths are capable of the following: (1) altering the activity of mechanoreceptors in the airways, lungs, and chest wall; (2) altering blood gas tensions; and (3) eliciting respiratory sensations in conscious or semiconscious patients.

In turn, these can alter the rate, depth, and timing (T_I, T_E) of respiratory efforts through neural reflexes, chemical control (chemoreceptors), and behavioral responses. These changes in patient effort may modify expected responses to changes in ventilator settings. These modifications may take several forms, including the following: (1) failure of $\dot{V}_E$ or V_T to change in the expected direction or to the expected magnitude as ventilator settings are changed to increase or decrease these variables; (2) periodic breathing, with periods of apnea, as P_{CO_2} cycles around the CO_2 set point; (3) loss of synchrony between patient and ventilator; and (4) changes in ventilatory demand (increase or decrease) as a result of altered level of consciousness (following a change in ventilator settings) or of changes in mechanoreceptor output (acting via reflex or behavioral mechanisms). There are very few systematic studies on the effect of various changes in ventilator settings on pattern of patient effort. This information is needed particularly with the current shift in emphasis from controlled ventilation methods to patient-interactive methods (*eg,* PSV).

Section 5: Complications of Mechanical Ventilation

Although mechanical ventilation offers vital life support, its use can result in untoward or life-threatening side effects. Many such hazards can be modified or avoided by appropriate attention to the technique of implementation. Interventions associated with mechanical ventilation include airway intubation, application of positive pressure to the respiratory system, provision of supplemental oxygen, imposition of unnatural breathing patterns, and the administration of sedative or paralytic agents.

A. Complications of Airway Intubation

Endotracheal intubation is usually performed transnasally or transorally. The nasal route provides a more stable artificial airway and allows mouth closure, improving comfort in some patients. However, a bleeding diathesis is a contraindication to nasal intubation. Moreover, because a nasal tube is generally smaller than its oral counterpart, it presents greater flow resistance and may impede extraction of retained secretions from the central airways. Sinusitis is a potential complication of ostial occlusion and impeded drainage by the nasal tube.

The artificial airway allows potential pathogens to enter the trachea from the external environment, dramatically increasing the risk of nosocomial pneumonia. Moreover, disruption of the coughing mechanism and mucociliary escalator encourages retention of airway secretions. Endotracheal tubes prevent aspiration of gross particulates, but permit pharyngeal secretions to

enter the trachea via the interstices of the balloon cuff, frequently resulting in tracheal colonization and increasing the risk of nosocomial pneumonia.

Tube misplacement and dislocation occur frequently. Although intubation of the right or (less frequently) left main bronchus most commonly occurs at the time of intubation, head movement may cause the tube orifice to migrate 2 cm in either direction from its neutral position along the tube axis. Overdistention of the ventilated lung and hypoventilation or atelectasis of the nonintubated lung are especially likely to occur in the heavily sedated patient receiving PPV. Hypoxemia, barotrauma, and cardiovascular compromise may result. Inadvertent extubation is among the most dangerous complications associated with mechanically ventilating a physiologically unstable patient; consequently, disoriented and uncooperative patients should be made as comfortable as possible, but they should be securely restrained.

Glottic injury often occurs during unusually difficult or emergency intubation. Glottic edema and minor erosive lesions of the vocal cords occur commonly during prolonged intubation. Postextubation glottic dysfunction and lasting damage to the vocal cords may occur, especially among women and among those patients in whom large tubes are placed. Risk factors for tracheal erosion, glottic stenosis, tracheal dilatation, and tracheomalacia are not precisely defined; however, cuff pressures that exceed capillary perfusion pressure (≅25 cm H_2O) are likely to cause ischemic ulceration and more advanced forms of mucosal damage.

In the absence of reliable guidelines to indicate optimal timing, most practitioners reserve tracheostomy for those patients who are not making clear and steady progress after 2 to 3 weeks of therapy or for those with suspected abnormality of the upper airway. Tracheostomy affords reduced V_D, partially restores glottic function, improves secretion clearance, enhances comfort, and holds the potential to allow both oral feeding and verbal communication. It may be associated, however, with life-threatening complications such as tracheal erosion, tracheo-innominate artery fistula (hemorrhage), and extraluminal migration of the tube orifice in the early postoperative period. Stomal granulation or stenosis are frequent problems after decannulation.

B. Complications of PPV

During PPV, the lungs and chest wall distend, intrathoracic pressure rises, and the lungs are often exposed to high inspired fractions of oxygen. In the setting of ARDS, for example, PaO_2 is improved by providing high fractional concentrations of inspired O_2 and by raising mean and end-expiratory alveolar pressures. Each of these interventions has an associated risk-benefit ratio. Although considerable experimental data have been accumulated, detailed clinical information is not yet available regarding which oxygen concentrations, pressures, and ventilation patterns are safe to apply for extended periods.

1. *Barotrauma:* For adult patients, flow-limited, volume-cycled ventilation using large V_Ts (10 to 15 ml/kg), rapid inspiratory flow rates, and PEEP when needed to adjust lung volume has previously been the standard of practice in managing most problems of ventilatory support.

Widely held objectives of ventilation have given priority to “normalizing” arterial blood gas values and ensuring adequate oxygen delivery. Until recently, respiratory system pressures have been monitored, but not tightly constrained.

There is little doubt that high ventilating pressures and excessive *regional* lung volumes are damaging. All forms of barotrauma that have been described previously in the pediatric literature, including interstitial and subcutaneous emphysema, pneumomediastinum, pneumoperitoneum, pneumopericardium, pneumothorax, tension cysts, systemic gas embolism, and damage similar to bronchopulmonary dysplasia, have now been recognized in adult patients, as well. Susceptibility to barotrauma may vary with the stage of the disease process; pressures well tolerated during the earliest stage of illness may prove excessive later on. Many forms of barotrauma occur with increased frequency after ventilation for extended periods, especially in patients with ARDS.

It has been shown in a variety of animal models that ventilation with high VTs or high PAP can induce or extend acute lung injury. In previously normal lungs, such damage is characterized by granulocyte infiltration, hyaline membranes, and increased vascular permeability. Fibroblast proliferation follows over a period of days. Such lung injury occurs with peak inspiratory pressure (PIP) as low as 30 cm H_2O in normal sheep, and with PIP as low as 20 cm H_2O in rabbits whose lungs have been lavaged with saline solution. There are also suggestions from the experimental literature that ventilatory pattern influences the incidence and severity of injury, but further evidence is needed, and an optimal pattern has not been defined.

Although ARDS has previously been considered a problem of *diffuse* lung injury and a generalized increase of tissue recoil, it now appears that the radiographic, densitometric, and mechanical consequences of ARDS are heterogeneous. In severe cases, the inflation capacity of the lungs may be less than one third of normal. The compliance and fragility of tissues comprising the aerated compartment in patients with ARDS may be closer to normal than previously envisioned, especially in the earliest phase of this disease.

It is currently believed that ventilatory patterns that apply high transalveolar stretching forces cause or perpetuate tissue edema and damage. Some experimental data suggest that large VTs, themselves, may also extend tissue edema, independent of the maximal pressure to which the alveolus is exposed. It is not clear, however, that large VTs are injurious when peak alveolar pressures are kept below 35 cm H_2O and sufficient PEEP is used to prevent widespread alveolar collapse and thereby maximize respiratory system compliance. Use of small VTs that avoid tissue overdistention and the acceptance of any consequent elevation of $PaCO_2$ (permissive hypercapnia) have been suggested to minimize the risk of barotrauma in patients with asthma and ARDS.

Although it remains unproved, some data suggest that periodic inflations with a relatively large volume may be needed to avert collapse of unstable lung units when very small VTs and low levels of PEEP are used. In animal models, recruitment of lung volume by adequate amounts of PEEP can substantially reduce the ventilator-associated lung injury resulting from high PIP.As yet, such application has not been shown to have a similar benefit in patients.

Strategies that prevent the exposure of the lung to high pressures (limiting overdistention) and those that lower the $\dot{V}E$ requirement may be associated with less ventilator-induced tissue injury and improved outcome. Such approaches include permissive hypercapnia, pressure-controlled ventilation, and pressure-limited, volume-cycled ventilation. Based on the experimental literature, maximal transalveolar pressure should not exceed 30 to 35 cm H_2O during each tidal cycle. (This usually corresponds to 35 to 45 cm H_2O end-inspiratory static [plateau] pressure, depending on chest wall compliance.)

In certain experimental settings, even pressures lower than this have been associated with tissue injury, especially when applied for extended periods. It may be desirable to allow spontaneous ventilatory efforts whenever it is possible to do so without incurring an excessive breathing workload or unbalancing the $\dot{V}O_2$ /$\dot{D}O_2$ relationship. These modes, which include intermittent mandatory ventilation (IMV), pressure support, airway pressure release ventilation (APRV), bi-level airway pressure, intermittent mandatory pressure release ventilation (IMPRV), CPAP, and various modes applied without airway intubation (noninvasive ventilation), tend to reduce maximal transalveolar pressure but do not guarantee a reduced incidence of barotrauma.

Adjunctive measures to conventional ventilation aimed at enhancing tissue oxygen delivery or increasing CO_2 removal (extracorporeal [$ECCO_2R$] or intracaval [IVOX] gas exchange tracheal gas insufflation, high-frequency ventilation [HFV]) decrease the exposure of the lung to high pressures (and volumes) and may, at times, allow better gas exchange than is otherwise possible. The success of these hazardous methods depends heavily on the skill with which they are applied. At the present time, their utility for general medical practice must be considered unproved.

2. *Oxygen Toxicity:* High fractions of inspired O_2 (FIO_2) are potentially injurious when applied over extended periods. In the laboratory setting, tissue injury depends on the FIO_2 and the duration of exposure. Because alveolar injury is an exponential function of inspired oxygen concentration (FIO_2), even modest reductions in FIO_2 over the range of 0.6 to 1.0 may attenuate tissue damage. There is no convincing evidence that sustained exposure to $FIO_2 \leq 0.5$ causes tissue injury, and for practical purposes, most clinicians do not attempt aggressive measures to reduce FIO_2 (*e.g.,* vigorous diuresis, inotropic pharmacotherapy, high levels of PEEP, experimental modes of ventilation, or adjunctive support) until the inspired O_2 concentration exceeds approximately 0.60. The combinations of O_2 concentration and duration of exposure that produce significant damage have not been firmly established in the setting of critical illness, and may well vary with disease type, severity, and individual susceptibility.

3. *Cardiovascular Complications:* Ventilatory support can help restore the balance between DO_2 and O_2 consumption when it alleviates an intolerable breathing workload. Conversely, PPV often impairs cardiac output by disturbing the loading conditions of the heart, as described earlier in Section 4, A-3.

Mean lung volume or MalvP correlates best with the tendency of a given ventilatory pattern to cause hemodynamic compromise. *Under conditions of passive inflation,* MAP (as a clinically measurable reflection of MalvP) relates fundamentally to oxygen exchange, cardiovascular performance, and fluid retention. The importance of each effect varies greatly in different

patients. Mean airway pressure can be raised by adding PEEP, by extending the inspiratory time fraction (increasing I:E ratio), or by increasing $\dot{V}E$. The tendency for an elevation of MAP to compromise hemodynamic performance is heightened by impaired cardiovascular reflexes and depletion of intravascular volume.

The proportion of the alveolar pressure transmitted to the pleural space is determined by the relative compliances of the lung and chest wall:

$$\Delta Ppl = \Delta Palv \infty (Cl/[Cl+Cw]).$$

Therefore, the hemodynamic effect of a given increment in MalvP will be accentuated when the lungs are relatively compliant and/or the chest wall is stiff. Hemodynamic consequences are predictably less when the patient makes spontaneous breathing efforts. For these reasons, dynamic hyperinflation occurring in a passively ventilated patient with severe airflow obstruction produces auto-PEEP that is particularly likely to cause hemodynamic compromise. Auto-PEEP can be attenuated by reducing $\dot{V}E$ or by reducing I:E ratio, thus increasing expiratory time.

Limiting tissue demand for oxygen and maintaining effective cardiovascular function are essential to an effective ventilation strategy. Recent studies suggest that survival in overt sepsis and sepsis syndrome correlates with oxygen delivery. At the time of this writing, however, it is not clear whether therapeutic interventions that attempt to maximize O_2 delivery (when it is already in the normal range and there are no overt signs of tissue hypoxia) or avoid depression of cardiac output improve outcome. Maximizing O_2 delivery may require expansion of intravascular volume, an intervention that has been associated with adverse outcomes in patients with acute lung injury.

4. *Breathing Effort and Patient-Ventilator Asynchrony:* The use of mechanical ventilation superimposes a clinician-selected pattern of ventilation on the patient's natural breathing rhythm. Circuits that impose substantial resistance and machines that respond poorly to the flow demands or cycling cadence of the patient may result in dyspnea and an unnecessary breathing workload. Factors that have been shown to increase the breathing workload during partial ventilatory support include ET tube resistance, excessive triggering threshold or response delay, insufficient flow capacity of the ventilator to meet peak patient demands, and the development of dynamic hyperinflation. The latter gives rise to auto-PEEP, which depresses the *effective* functional triggering sensitivity and may contribute to intermittent failure of the machine to respond to patient effort.[98] Inappropriately slow inspiratory flow rates may cause the patient to enter the expiratory phase of the tidal cycle before the volume-cycled breath from the machine has been completed, often resulting in conflict between the natural and the imposed breathing rhythms. The magnitude and importance of these effects is a direct function of $\dot{V}E$.

Pressure-limited modes of ventilation (*eg,* pressure support) are theoretically unlimited with respect to meeting maximal flow demands; however, this ideal is seldom accomplished in practice. Such modes often present important problems of their own for the vigorously breathing patient. Pressure support, for example, although invaluable for overcoming ET tube resistance and for assisting the patient with moderate ventilatory requirements, is not well suited to the

needs of a patient breathing at a rapid cycling frequency, one with variable ventilatory requirements, or one who has a very high airway resistance (especially from a very small ET tube). Tidal volume can fall dramatically as frequency increases, especially in patients with airflow obstruction. Moreover, a fixed level of pressure support cannot compensate for a change in ventilatory impedance or the development of auto-PEEP. A patient who requires a high level of pressure support when breathing at a rapid rate may find the applied level of pressure excessive when the ventilatory requirement abates.

C. Adverse Effects of Sedation and Paralysis

Sedation and paralysis are often required to allow patient comfort and to facilitate the imposition of ventilatory patterns (*eg,* inverse ratio ventilation [IRV]) which would otherwise conflict with the patient's own ventilatory pattern. Sedation may result in vasodilation that contributes to hypotension and reduced cardiac output. Paralytic agents immobilize the patient, encouraging secretion retention, atelectasis, and muscle wasting. With breathing efforts prevented, such patients are totally dependent on the ventilation set by the clinician and provided by the machine. Inadvertent disconnection of the ventilator circuit can prove rapidly catastrophic in this setting. Certain neuromuscular-blocking agents have been recently associated with neuromuscular weakness that persists long after withdrawal of drug treatment. At present, it is not certain whether such effects relate primarily to the nature, dose, or duration of the drug, to synergism with corticosteroids, or to the depth of paralysis itself.

D. Other Complications

Among the wide variety of noncardiopulmonary complications that have been described for mechanical ventilation, perhaps the most important involve mental distress and dysfunction of the renal, gastrointestinal, and central nervous systems. Psychological distress during mechanical ventilation is exceedingly common, for reasons that include (but are not limited to) sleep deprivation and impaired sleep quality, pain, fear, inability to communicate, and the use of drugs (*eg,* benzodiazepines) with dissociative properties.

Renal dysfunction during PPV is believed to be a consequence of reduced circulating blood volume. This tends to alter perfusion of the renal parenchyma, redistribute intrarenal blood flow, release antidiuretic hormone, or inhibit atrial natriuretic peptides. In any event, reduced free water clearance and generalized fluid retention are commonplace during ventilation with high pressures.

Gastrointestinal consequences of mechanical ventilation include gut distention (due to air swallowing), hypomotility, and obstipation (due to pharmacologic agents and immobility), vomiting (due to pharyngeal stimulation and motility disturbances), and mucosal ulceration and bleeding. Serious dysfunction of the liver that occurs as a direct consequence of mechanical ventilation is rare. However, PEEP has been associated with hyperbilirubinemia and mild

elevations of liver enzyme levels in the serum, possibly related to altered hepatic perfusion and impeded venous and biliary drainage.

Increased intrathoracic pressure can elevate jugular venous and intracranial pressures, and thereby reduce cerebral perfusion pressure. Such effects assume particular importance in the setting of reduced mean arterial pressure and reduced intracranial compliance resulting from head injury or surgical intervention. The risk of intracranial hypertension in patients ventilated with high MAPs is attenuated by conditions that limit transmission of alveolar pressure to the pleural space (low ratio of lung to chest wall compliance).

Section 6: Specific Modes of Ventilation

A. Standard Modes

1. *Introduction:* Assisted modes of ventilation are those in which part of the breathing pattern is contributed or initiated by the patient. The work of breathing performed by the patient is never abolished, and one of the difficulties for the physician is to determine the appropriate settings to match the patient's demand, both in terms of gas exchange and work of breathing. The main reasons for using assisted modes in the ICU are the following: (1) to synchronize patient and ventilator activity; (2) to reduce the need for sedation; (3) to prevent disuse atrophy of the respiratory muscles; (4) to improve hemodynamic tolerance of ventilatory support; and (5) to facilitate the weaning process.

Significant differences exist among ventilators concerning demand-valve sensitivity and opening delay, the algorithm for pressure support (PS) (rise in pressure, mechanism, and criteria for cycling from inspiration to exhalation, plateau pressure), flow-impedance characteristics of the expiratory circuit, and equipment. A great deal of data obtained experimentally and in patients suggest that these differences may alter the work of breathing. Many of the proposals listed below may be modified by the quality of the equipment used.

2. *Assist-Control (A/C):*This is a mode of ventilation in which every breath is supported by the ventilator. A backup control ventilatory rate is set; however, the patient may choose any rate above the set rate. Until recently, most ventilators using this mode delivered breaths that were volume cycled or volume targeted. Using volume-targeted, A/C ventilation, the V_T, inspiratory flow rate, flow waveform, sensitivity, and control rate are set. Most of the data in the literature concerning A/C were obtained with this mode. Pressure-limited or pressure-targeted A/C in which pressure level, T_I, control rate, and sensitivity are set is now available on several ventilators.

Advantages. Assist-control ventilation combines the security of controlled ventilation with the possibility of synchronizing the breathing rhythm of patient and ventilator, and it ensures ventilatory support during every breath.

Risks or Disadvantages. (1) Excessive patient work occurs in cases of inadequate peak flow or sensitivity setting, especially if ventilatory drive of the patient is increased (volume-targeted A/C);[25,42,101] (2) it may be poorly tolerated in awake, nonsedated subjects and can require sedation to ensure synchrony of patient and machine cycle lengths; (3) it may be associated with respiratory alkalosis; (4) it may potentially worsen air trapping in patients with COPD; and (5) if pressure-targeted A/C is used, there is risk of variable (and potentially markedly decreased) VT during changes in lung impedances, patient ventilatory drive, or patient-ventilator dyssynchrony.

Patient work of breathing or effort during volume-targeted A/C is dependent on sensitivity, flow rates (flow rate lower than 40 L/min should probably be avoided), and respiratory drive of the patient. This is dependent on many stimuli, including fever, anemia, hypoxia, pain, hypovolemia, level of consciousness, etc. Since patient work is dependent on the ability of the ventilator to rapidly recognize patient effort and provide sufficient flow to meet inspiratory demand, the set-up of the ventilator may play an important role in the patient's tolerance of this mode.[63,101] With pressure-targeted A/C, the ventilator, once triggered, provides sufficient flow to allow the set pressure plateau level to be achieved rapidly. As a result, concern with excessive patient work is potentially minimized in pressure-targeted A/C.

3. *Synchronized Intermittent Mandatory Ventilation (SIMV):* Synchronized IMV is a mode of ventilation and a mode of weaning that combines a preset number of ventilator-delivered mandatory breaths of predetermined VT with the facility for intermittent patient-generated spontaneous breaths.[102,103] When SIMV is used, the patient receives different types of breath, including: controlled (Mandatory) breath, Assisted (synchronized) breaths, and Spontaneous breaths, which can be pressure supported. Similar to A/C, several ventilators offer the possibility of delivering pressure-targeted breaths instead of volume-targeted breaths during mandatory cycles. Mandatory breaths can be patient triggered with SIMV; however, if patient effort is not sensed within a specific period, the ventilator delivers a mandatory breath. Pressure support (see below) may be applied during nonmandatory breaths.

Set-up Parameters. These include VT, flow rate and/or TI, frequency of controlled breaths, and sensitivity. When pressure-targeted breaths are used, pressure level and TI must be set.

Advantages. (1) The patient is able to perform a variable amount of respiratory work and yet there is the security of a preset mandatory level of ventilation; (2) SIMV allows for a variation in level of partial ventilatory support from near-total ventilatory support to spontaneous breathing; and (3) it can be used as a weaning tool.

Risks. With IMV, there are risks of dyssynchrony between the patient effort and machine-delivered volume.

With SIMV the risks are as follows: (1) hyperventilation and respiratory alkalosis are possible, similar to A/C. (2) Excessive work of breathing due to the presence of a poorly responsive demand valve, suboptimal ventilator circuit (its impedance will vary with the particular ventilator used), or inappropriate flow delivery could occur. In each case, extra work is imposed on the patient during spontaneous breaths. This work can be minimized or abolished with the

addition of pressure support. (3) Worsening dynamic hyperinflation has been described in patients with COPD.

The total work (or power) performed by the patient is dependent on the number of mandatory breaths. It was initially thought that the effort performed by the patient was virtually zero during mechanical breaths. However, recent data suggest that the muscular effort of the dyspneic patient during machine-assisted breaths does not vary substantially from the unassisted cycles on a breath-to-breath basis, *i.e.,* at the same overall level of ventilator support, effort is more or less independent of whether or not the breath is assisted. The work of breathing may also vary with the addition of pressure support during spontaneous cycles and with the use of pressure-targeted mandatory breaths. There has been no demonstrated advantage of using IMV over T-piece trials in terms of reducing weaning duration.

4. *Pressure Support Ventilation (PSV):* Pressure support ventilation is a pressure-targeted, flow cycled, mode of ventilation in which each breath must be patient triggered. It is used both as a mode of ventilation during stable ventilatory support periods and as a method of weaning patients. It is primarily designed to assist spontaneous breathing and therefore the patient should have an intact respiratory drive.

With PSV, at the onset of inspiration, the pressure rises rapidly to a plateau that is maintained for the remainder of inspiration. The patient and ventilator work in synchrony to achieve the total work of each breath. On most ventilators, termination of inspiration occurs when a flow threshold is reached during the decelerating phase of inspiratory flow. That is, the breath is flow cycled to exhalation (this has been made possible by incorporating pneumotachographs in the ventilators). To avoid confusion, it should be stressed that the difference with pressure-targeted A/C, described above, is that termination of inspiration is different: it is time cycled for pressure-targeted A/C (*i.e.,* the T_I is fixed), whereas it is flow cycled for pressure support, *i.e.,* airway pressurization always stops before reaching the zero flow, and inspiratory duration is dependent on patient's effort.

Set-up Parameters. These include pressure level and sensitivity. No mandatory PS rate is set; however, many ventilators incorporate volume-targeted back-up modes in the event of apnea. In some ventilators, it is possible to adjust the rate of rise in pressure at the beginning of inspiration or to adjust the flow threshold for cycling from inspiration to expiration.

Advantages. (1) As a result of the patient having significant control over gas delivery, overt dyssynchrony is less likely than with A/C or SIMV. When the PS level is chosen appropriately, this mode is generally regarded as comfortable for most (but not all) spontaneously breathing patients. (2) Pressure support ventilation reduces the work of breathing roughly in proportion to the pressure delivered and is associated with a decrease in respiratory frequency and increase in V_T with increasing levels of PS. These breathing pattern characteristics may be useful in selecting the appropriate PS level. (3) Pressure support ventilation can be used to compensate for the extra work produced by the ET tube and the demand valve. (4) It allows for a wide variation in the level of partial ventilatory support from nearly total ventilatory support (high pressure levels) to essentially spontaneous breathing. (5) Pressure support ventilation may be useful in patients who are “difficult to wean.” Preliminary data suggest either no difference when

compared with other modes or a shorter weaning duration and a higher success rate in selected patients using pressure support.

Disadvantages. Tidal volume is not controlled and is dependent on respiratory mechanics, cycling frequency, and synchrony between patient and ventilator. Therefore, careful monitoring is recommended in unstable patients; a back-up $\dot{V}E$ seems necessary for safety; hypoventilation may develop during continuous-flow nebulizer therapy. Pressure support ventilation may be poorly tolerated in some patients with high airway resistances because of the preset high initial flow and terminal inspiratory flow algorithms. This may be improved, however, with adjustment of initial flow rates which is possible on new systems.

Work of Breathing. Increasing PS levels decrease respiratory effort as indicated by a number of changes in breathing pattern. This mode can be combined with SIMV and has been used to compensate for the additional work of breathing due to the ET tube and the demand valve, adding PS (5 to 10 cm H_2O) during the nonmandatory breaths. Higher levels of PS can also be used, combining mandatory and supported breaths. At the present time, clinical studies are lacking to demonstrate the superiority of one mode of partial ventilatory assistance over others. Pressure support has been widely accepted in many ICUs and for some physicians, it seems to be the most useful modality for delivering assisted ventilation either as full ventilatory support or as a mode for gradually withdrawing mechanical ventilation. For others, it is a useful adjunct to existing modes.

5. *Continuous Positive Airway Pressure (CPAP):* Continuous positive airway pressure is a mode designed to elevate end-expiratory pressure to levels above atmospheric pressure to increase lung volume and oxygenation. A constant positive airway pressure is supplied by the ventilator throughout the ventilatory cycle; all breaths are spontaneous. It is also proposed as a means of reducing the pressure gradient between the mouth and the alveoli in patients with air trapping. It is designed to assist spontaneously breathing patients and therefore requires an intact respiratory drive.

Until recently, two main types of CPAP systems were used. Those offered by most mechanical ventilators work via a demand valve that needs to be opened to deliver the gas to the patient. This demand valve is pressure triggered or flow triggered. Other specially designed systems work on the principle of a continuous high flow of pressurized gas in the external circuit from which the patient can breathe spontaneously. The advantage of the first system is that ventilator monitoring is still available but the major drawback is that work of breathing is increased by the presence of a demand valve.

A continuous-flow system is incorporated in some mechanical ventilators in an attempt to combine the advantages of the two previous systems. In this mode, an adjustable, constant flow of gas is continuously delivered in the external circuit during the expiratory phase. Both the inspiratory flow and the expiratory flow are measured and compared by the machine. A difference between these two flow rates indicates to the ventilator that inspiration or expiration is occurring, leading to an adjustment in the delivered flow rate. Flow of gas is calculated in liters per minute. The normal flow pattern of gas moving in and out of the lungs is sinusoidal.

Set-up Parameters. These include pressure level and sensitivity: level of negative pressure (demand valve system) or flow threshold and basal flow rate (continuous-flow systems and/or flow-triggered systems).

Advantages. CPAP offers the benefits of PEEP to spontaneously breathing patients. It will improve oxygenation if hypoxemia is in part secondary to decreased lung volume; it may recruit collapsed lung units, minimizing the work of breathing and improving oxygenation. It may help to reduce the work of breathing in patients with dynamic hyperinflation and auto-PEEP.

Recent data suggest that the work of breathing is reduced with systems incorporating a continuous-flow system in comparison to demand valve systems.

Risks. Hyperinflation and excessive expiratory work may result if excessive CPAP levels are used. Poor clinical tolerance may increase inspiratory work of breathing, if hyperinflation is produced or if nonthreshold PEEP devices are used. There will be increased expiratory work if hyperinflation is produced or if PEEP devices with large flow resistances are used. The use of demand valves with intubated patients receiving CPAP may lead to patient ventilator dyssynchrony.

This mode can be used in intubated patients as well as nonintubated patients (*eg,* patients with sleep apnea). Although inspiration is not really assisted, modern ventilators deliver a small level of pressurization, *i.e.,* a 1 to 3 cm H_2O level of pressure support, to avoid negative airway pressure relative to the end-expiratory level during inspiration. It is not clear, however, whether this has a significant clinical effect.

6. *Servo-Controlled Modes:* Servo-controlled modes are used both for ventilation and for weaning patients. The basic principle is the use of a feedback system to control a specific variable within a given narrow range.[115-117] The ventilatory mode is either SIMV or PSV. The targeted parameter is set by the physician and can be either $\dot{V}E$ or a component of the breathing pattern (respiratory rate, V_T).

Examples of servo-controlled modes include the following:

(i) *Mandatory Minute Ventilation (MMV).* MMV relies on a patient's spontaneous breathing to meet a predetermined $\dot{V}E$. If this goal is not met, mechanical breaths at predetermined volume are delivered with a rate sufficient to supply the required $\dot{V}E$. Here the targeted parameter is $\dot{V}E$. In some ventilators used to implement MMV, the basic mode is SIMV; in others, it can be PSV.

(ii) *Servo-Controlled PSV (With the Exception of MMV).* The underlying mode is PSV and the targeted parameter is respiratory rate or V_T. If the targeted value is not met, the ventilator can either modify the pressure target level or the way the breath is cycled. The regulation of the targeted variable varies among ventilators. It can take different forms: volume-assured PS, pressure augmentation, volume support, pressure-supported breaths, and volume-assisted breaths.

(iii) *Knowledge-Based Systems.* More complex systems have been implemented in microcomputer-driven ventilators and are being studied or are already used for the routine treatment of patients in specialized centers.[9,118-120] These systems have been proposed to help in the treatment of patients with ARDS or to automatically wean patients from mechanical ventilation. At the present time, none of these systems is commercially available.

Advantages include adaptation of the ventilator to the needs of the patient. These systems try to combine the advantages of a partial ventilatory support with the variability in the needs of the patient.

Disadvantages include the following: (1) The algorithm may induce nonphysiologic breathing patterns. (2) The adequate target value can be difficult to adjust (*e.g.,* adequate level of $\dot{V}E$ for MMV). (3) Measurement of $\dot{V}E$ may give false information if breathing pattern is not considered (rapid shallow breathing may go unrecognized). The published data and clinical experience with these modes are minimal.

B. Alternate Modes of Ventilation

1.Introduction: During the last decade, a new concept has emerged regarding acute lung injury. In severe cases of ARDS, only a small part of the lung parenchyma remains accessible to gas delivered by the mechanical ventilator. This is widely known as the *"baby lung"* concept. As a consequence, V_T of 10 ml/kg or more may overexpand and injure the remaining normally aerated lung parenchyma (See Section 5, B-1) and could worsen the prognosis of severe acute respiratory failure by extending nonspecific alveolar damage. Because lung volumes and airway pressures relationships are determined by the respiratory P-V curve, and because the apparent "stiffness" of ARDS lung appears related to the fraction of aerated lung, rather than to a generalized increase in elastic recoil, *the specific compliance of the remaining aerated lung parenchyma may be nearly normal.*

High airway pressures may result in overdistention and local hyperventilation of more compliant parts of the ARDS lung. Over-distention of lungs in animals has produced diffuse alveolar damage. There are also data in the literature suggesting that ventilation using relatively low end-expiratory pressures (less than the inflection point [opening pressure] of the pressure-volume curve) causes progression of lung injury in animal models of ARDS. This is the reason why alternative modes of mechanical ventilation—all based on a reduction of end-inspiratory airway pressures and/or V_T delivered to the patient and some based on ventilation between the lower and upper inflection points of the P-V curve—have been developed and are clinically used by many physicians caring for patients with severe forms of acute respiratory failure. Three of them, HFV, IRV, and airway pressure release ventilation (APRV), will be described in this section. Since, to our knowledge, there are no data demonstrating the superiority of these nonconventional ventilatory modes in terms of morbidity and mortality, only their physiologic rationale and their putative advantages and disadvantages will be presented.

2. High-Frequency Ventilation (HFV): High frequency ventilation is the administration of small V_T—1 to 3 ml/kg—at high frequencies—100 to 3,000/min. Because it is a mode of mechanical

ventilation based on a marked reduction in V_T and airway pressures, it has the greatest potential for reducing pulmonary barotrauma. Mechanisms of gas transport change from conventional bulk flow ($V_A = fx(V_T - V_D)$ to other types when $V_T \leq V_D$. Proposed mechanisms include coaxial flow, Taylor dispersion, pendelluft, and augmented molecular diffusion. Under these conditions, the fxV_T product is usually much higher than during conventional mechanical ventilation and V_A appears to be more influenced by V_T than f.

There are a number of different types of HFV. The three most common are high-frequency oscillation, high frequency PPV, which is used in anesthesia, and high-frequency jet ventilation (HFJV), which is used both in anesthesia and in critically ill patients with acute respiratory failure. HFJV is the only high-frequency mode routinely used to ventilate patients with ARDS, mainly in Europe. Convincing comparative data concerning the advantages of HFJV over conventional mechanical ventilation (CMV) have been presented in the following limited number of clinical studies. There is no agreement, however, that HFJV is better than CMV in these situations: in ARDS patients with circulatory shock, in cardiac patients with low cardiac output state, and in patients with tracheomalacia, BPF, and tracheoesophageal fistula.

In one well-controlled multi-centered clinical trial (the HIFI trial), high-frequency oscillation was found not to be superior to CMV in ventilation of neonates with infant respiratory distress syndrome, but this study has been criticized because of the lack of a volume recruitment protocol. In a number of animal studies, ventilation above the inflection point is required for the beneficial effects of HFV; HFV may need to be implemented early in the course of the disease to be effective.

Because of the risk of gas trapping related to expiratory flow limitation, HFV is generally contraindicated for asthma and COPD. Although some European groups routinely use HFJV in combination with low V_T conventional ventilation to treat patients with severe forms of ARDS (8 to 10 tidal volumes per minute of 3 to 4 ml/kg superimposed on HFJV), there are no convincing data demonstrating the superiority of this method of mechanical ventilation in terms of pulmonary barotrauma and mortality. It must be pointed out that the only prospective randomized study comparing HFJV with conventional ventilation which was performed in a nonhomogeneous population of cancer patients with ARDS did not demonstrate any significant advantage for one or the other method. HFJV can be safely administered in the clinical setting of the ICU according to the following guidelines:

(i) Clinicians must be very familiar with the technique (ventilatory settings, types of injection, humidification).

(ii) Like all other forms of HFV, HFJV, when administered for periods longer than 8 h, requires adequate humidification of delivered gases or severe necrotizing tracheobronchitis can occur. Because the pressure drop across the injection system is very large (*the operating pressures are between 1 and 3 atmospheres whereas MAP is between 1 and 30 cm H_2O*), gas expansion occurs within the trachea causing cooling. Therefore, specially designed devices for providing adequate humidification during HFJV are required. One such device is a specially constructed high-temperature vaporizer. Many detrimental effects of HFJV are, in fact, due to inadequate humidification.

(iii) The effects of ventilator parameters related to airway pressures and V_T are reasonably well understood. Respiratory effects of changing ventilatory settings are markedly influenced by the patient's respiratory mechanics. Increasing I:E ratio and driving pressure increase FRC and V_T. Increasing respiratory frequency markedly decreases V_T volume and increases $PaCO_2$ and has minimum effect on FRC in patients with stiff lungs.[132] The more compliant the respiratory system, the larger the increase in FRC induced by increasing I:E ratio, driving pressure, and respiratory frequency.

(iv) Mean airway pressure should be continuously monitored using an intratracheal catheter located at least 5 cm below the injection site. It has been demonstrated in experimental and clinical conditions that MAP measured during HFJV is a reasonably good reflection of MalvP in patients *without* significant obstruction.

(v) There is a large body of evidence in various animal models that HFV is most effective in diseases with stiff lungs when applied following a volume recruitment maneuver. The aim is to ventilate the lung at a pressure above the inflection point, yet at pressures sufficiently low not to cause high pressure (or volume) damage to the lung. This approach has been used in neonates with success, but to date, these clinical trials have been relatively small.

Potential Risks. Due to the large flow rates used and the fact that gas transport is less well understood, HFV is inherently more dangerous than CMV. Outflow obstruction can rapidly lead to increases in lung volume and attendant hemodynamic compromise and barotrauma. Air trapping due to the high flow rates is always of concern, especially in patients with compliant lungs and airways obstruction. Air trapping can be assessed by measuring airway opening pressure under static conditions after airway occlusion, by monitoring esophageal pressure, or by measurements of lung volume obtained at the chest wall (*eg,* inductive plethysmography). Inadequate humidification can induce severe necrotizing tracheobronchitis as described above.

3. Inverse Ratio Ventilation (IRV): Inverse ratio ventilation is the use of I:E ratio >1:1 during CMV. There are two different types of IRV: pressure-controlled (pressure-limited) IRV, where the ventilator generates a servo-controlled square wave of pressure to the airways via a decelerating inspiratory flow, and volume-cycled IRV, where the ventilator generates a predetermined V_T via a constant or a decelerating inspiratory flow. Flow profiles of appropriate length of inspiratory “holds” or “pauses” are applied as necessary for the desired I:E ratio. Pressure-controlled IRV is more widely used than volume-cycled IRV in patients with ARDS. Since MAP is a major determinant of PaO_2, a major part of the rationale for using IRV in ARDS is to maintain MAP relatively high, but to hold peak alveolar pressure within a safe range.

The second theoretic concept underlying IRV is the prolongation of inspiration to allow for recruitment of lung units with long time constants. If air trapping does not develop, MAP will increase without a change in PAP or V_T. On the other hand, if T_I is so prolonged that air trapping does develop, the resulting auto-PEEP will either raise PAP (volume-cycled IRV) or decrease V_T (pressure limited or pressure-controlled IRV). Indeed, it appears that many of the reported advantages of IRV in improving PaO_2 are related to air trapping (auto-PEEP), and that similar beneficial effects on oxygenation or O_2 transport may be obtained by using conventional I:E ratios with sufficient PEEP to obtain the same increase in mean lung volume. Deep sedation

and/or paralysis are nearly always required. At present, there is a lack of convincing data to support the advantage of IRV over conventional ventilation. To our knowledge, no study has evaluated the outcome or the comparative incidence of pulmonary barotrauma in ARDS patients treated with IRV as opposed to conventional ventilation.

Nevertheless, if IRV is used, it can be safely implemented in the critically ill with ARDS, according to the **following guidelines**:

(i) Volume-controlled IRV may be more easy to implement than pressure-controlled IRV since volume-cycled modes are often more familiar to many clinicians. This ventilatory mode guarantees a preset V_T and is available on all ICU ventilators.

(ii) Deep sedation is required in most patients under IRV to avoid dyssynchrony with the ventilator.

(iii) Careful monitoring of PAP and end-inspiratory plateau pressure is required during volume-controlled IRV. The high pressure alarm should be set at 10 cm H_2O above the intended PAP.

(iv) Careful monitoring of $\dot{V}_E$ is required during pressure-controlled IRV because V_T is markedly dependent on the patient's respiratory mechanics.

(v) The auto-PEEP level, which may develop as the I:E ratio increases, should be regularly measured (see Section 4, A-6).

(vi) Hemodynamic status should be assessed using a Swan-Ganz catheter when IRV is implemented.

These guidelines should help minimize the two major complications associated with the use of IRV: pulmonary barotraumas and hemodynamic deterioration.

4. *Airway Pressure Release Ventilation (APRV):* Airway pressure release ventilation increases alveolar ventilation by intermittently releasing continuous positive pressure generated by the ventilator. In passive patients, APRV is identical to pressure-controlled IRV; however, the patient's ability to breathe spontaneously during APRV creates a markedly different intrapleural pressure waveform. The rationale for APRV is to limit PAPs, thereby limiting barotrauma. APRV is not intended for patients with severe airflow obstruction.

There are two types of pressure release ventilation: APRV during which pressure release time is preset, and IMPRV. Both are specifically designed for assisting spontaneously breathing patients. In these modes, ventilatory assistance is provided by intermittent changes in FRC related to changes in PEEP. Comparative experimental and clinical studies have shown that APRV and IMPRV can improve alveolar ventilation of animals and humans breathing with CPAP, without a deterioration in arterial oxygenation or an increase in PAP. When compared with CMV, APRV was shown to produce similar hemodynamic effects at similar MAP in

patients with acute respiratory failure. Whether this type of ventilatory support has any advantage over CMV with PEEP in terms of pulmonary barotrauma is not known. APRV can be provided by a CPAP breathing circuit in which the CPAP level can be modified by opening or closing a release valve connected to a timer.

IMPRV, which has been integrated in an ICU ventilator, provides end-expiratory pressure changes according to the patient's spontaneous breathing activity. Respiratory monitoring and alarms are available and each spontaneous inspiration can be assisted by PS. If the patient's respiratory frequency increases above 30/min, auto-PEEP becomes a limiting factor and IMPRV is no longer an efficient method of ventilatory support.

During APRV, the following respiratory parameters are preset: upper and lower airway pressure levels, frequency of pressure release, and pressure release time. During IMPRV, the following respiratory parameters are preset: upper and lower PEEP levels, frequency of PEEP changes and sensitivity of the trigger. Ventilatory assistance is maximum when PEEP is changed in each of two spontaneous respiratory cycles and can be progressively decreased by spacing PEEP changes (PEEP release every 2, 3, 4, 5, 6 cycles, *etc.* … spontaneous expiration). Whether this type of ventilatory assistance can facilitate weaning of patients with acute respiratory failure is not known.

Section 7: Discontinuation of Mechanical Ventilation

A. What Is It, and When Does It Begin?

Weaning has been defined as the process whereby mechanical ventilation is *gradually* withdrawn and the patient resumes spontaneous breathing. Within the daily vernacular of the ICU, most clinicians do not employ the term weaning in the strict sense, but rather they use it to include the overall process of discontinuing ventilator support. To enhance communication between investigators and clinicians, it may be wise to drop the term *weaning,* and replace it by a term such as discontinuation of mechanical ventilation. This, in turn, could be subdivided into different categories depending on the pace of the discontinuation process—these terms could replace older, less precise terminology such as the “fast wean” and “slow wean.” Alternatively, the term *discontinuation* could be used to describe disconnecting the patient from the ventilator over a short, predefined time limit, while *weaning* refers to the more gradual process; unfortunately, the dividing line between these two processes is arbitrary with no obvious basis.

It has become increasingly difficult to define the precise time at which the discontinuation process commences. It was relatively easy to define this time in the past when volume-cycled A/C ventilation and T-tube trials were the sole or predominant method of treating patients. With the widespread use of IMV and PSV in modern ICUs, it has become increasingly difficult to define the precise time at which these modes are no longer being used as the primary mode of ventilator support and are being adjusted to assist with the discontinuation process. In an ICU setting, ventilator support is typically initiated because of an episode of acute respiratory failure.

In general, most clinicians would consider it imprudent to start a discontinuation process until there is evidence of significant resolution of the initial precipitating illness. Unfortunately, rigorous physiologic or clinical indices have never been proposed to help to define this time. This largely relates to the lack of data characterizing the changes in respiratory function from the time that ventilator support is instituted until the time that it can be safely withdrawn. Until this time can be defined in clear-cut objective terms, it is going to be extremely difficult to conduct trials comparing the efficacy of different techniques of discontinuing mechanical ventilation.

B. Relative Importance of Pathophysiologic Determinants of the Discontinuation Process

There are four major factors that determine the ability to discontinue ventilator support: (1) respiratory load and the capacity of the respiratory neuromuscular system to cope with this load; (2) oxygenation; (3) cardiovascular performance; and (4) psychological factors.

To our knowledge, systematic studies have never been conducted to determine the relative importance of these pathophysiologic mechanisms. However, many clinicians and investigators suspect that respiratory muscle dysfunction resulting from an imbalance between respiratory neuromuscular capacity and load is the most important determinant. Unfortunately, measurements of each of the components included in this balance have not been systematically obtained in patients at the time that ventilator support is being discontinued. Measurements of respiratory center output indicate that a depressed respiratory drive is rarely responsible for the inability to discontinue ventilator support. Phrenic nerve function is usually satisfactory, except for a small proportion of patients who develop problems following coronary artery bypass surgery. Respiratory muscle strength is reflected by measurements of maximal inspiratory pressure. Available evidence suggests that this, *on its own,* is not an important determinant of the ability to resume and sustain spontaneous ventilation after a period of mechanical ventilation.

A number of techniques can be used to assess respiratory muscle endurance or fatigue in a research laboratory. None of these has ever been reliably applied in ventilator-supported patients. Thus, we do not know if respiratory muscle fatigue ever occurs in patients who are unable to resume spontaneous ventilation and, if it occurs, how important it is in determining clinical outcome or patient treatment. Respiratory muscle fatigue has been defined in dichotomous terms (present or absent), but the impairment in contractility is more likely to exist in the form of a continuum. Thus, it is quite conceivable that “mild fatigue” *per se* may not seriously interfere with the process of discontinuing ventilator support. This is an area where additional research is sorely needed.

The load on the respiratory system is primarily determined by an increase in respiratory resistance, a decrease in respiratory compliance, and the presence of auto-PEEP, which poses an additional threshold load. Each of these factors could produce a marked increase in respiratory work and interfere with the process of discontinuing the ventilator. Although measurements of respiratory work have been obtained in patients at the time of discontinuing mechanical ventilation, most studies contain significant methodologic flaws. Even allowing for these limitations, it is doubtful that a single threshold value of respiratory work can reasonably discriminate between patients who are able to successfully sustain spontaneous ventilation and

those requiring continued ventilator support. In particular, there is a tremendous need for research defining the precise interplay between respiratory load and respiratory muscle performance in such patients. Such knowledge would be important not only in elucidating the mechanisms responsible for the inability to resume spontaneous ventilation, but it would also help in guiding optimal ventilator support prior to the discontinuation attempts.

Although mechanical ventilation is commonly instituted because of problems with oxygenation, this is rarely a cause of difficulty at the time that mechanical ventilation is being stopped, largely because ventilator discontinuation is not contemplated in patients who display significant problems with oxygenation.

Research into the discontinuation of ventilator support has primarily focused on factors affecting the respiratory system. Although impaired cardiovascular performance has significant impact on the respiratory system (decreasing O_2 supply to the respiratory muscles, increasing respiratory work secondary to pulmonary edema, and hypoxemia as a result of a low mixed venous O_2 tension), remarkably few studies have examined the role of cardiovascular performance as a determinant of the ability to resume successful spontaneous ventilation.

In patients with known coronary artery disease, significant cardiovascular impairment reflected by a marked increase in pulmonary artery occlusion pressure has been documented at the time of resuming spontaneous ventilation. This is another area where much research is required, both in patients who require ventilator support primarily because of cardiovascular problems, and in patients without obvious underlying cardiovascular disease. In particular, it is important to document the time course of significant decompensation in such patients and to determine if this differs significantly from the pattern occurring in patients with a primary pulmonary disorder.

Psychologic factors are a major determinant of outcome in some patients, especially in those patients who require prolonged ventilator support. Minimal research has been conducted into this important issue, and, thus, it is difficult to state its relative importance in determining the ability to resume spontaneous ventilation.

C. Predictive Indices

A wide variety of physiologic indices have been proposed to guide the process of discontinuing ventilator support. Traditional indices include the PaO_2/FIO_2 ratio, the alveolar-arterial PO_2 gradient, maximal inspiratory pressure, VC, $\dot{V}E$, and maximum voluntary ventilation. Newer indices include pressure measured 0.1 s after occlusion of the airway, the f/V_T ratio, and integrative indices such as CROP (an integrative index which includes compliance, rate, oxygenation and pressure[1]), the pressure-time index, and V_E40. In general, these indices evaluate a patient's ability to sustain spontaneous ventilation. They do not assess a patient's ability to protect his/her upper airway. Indices of upper airway function have been developed for treating postoperative patients, but similar indices have not been evaluated in critically ill patients.

There are enormous discrepancies in the literature on the accuracy of indices in predicting successful discontinuation of mechanical ventilation. Discordance is due, at least in part, to

methodologic problems and differences among studies. These include the following: (1) characteristics of the patient population; (2) the method of making the measurements; (3) reproducibility of the measurement; (4) the method of selecting the threshold value of an index; (5) the method of testing the accuracy of an index; and (6) definition of end points in the evaluation study.

As currently employed, predictive indices are most commonly used in evaluating a patient for extubation. Measurement of these physiologic indices may suggest to a physician that ventilator support can be discontinued at an earlier time than he/she might otherwise have thought possible. This may help in decreasing the risk of complications associated with mechanical ventilation. When an index suggests that resumption of sustained spontaneous ventilation is unlikely to be successful, it can provide important information regarding the patient's underlying pathophysiologic state. However, there is no evidence to suggest that a particular set of physiologic indices is helpful in guiding the selection of a particular technique to hasten the process of discontinuing ventilator support. Accordingly, at this time, it is impossible to say precisely if, and how, such physiologic indices should be used in clinical decision making or in the treatment of a patient who is still requiring ventilator support.

It is important to remember that the condition of ventilator-dependent patients can vary considerably from day to day. Thus, a patient's ability to successfully resume and sustain spontaneous ventilation should be evaluated on a recurrent basis.

D. Techniques of Discontinuing Ventilator Support

The major techniques of discontinuing ventilator support include T-tube trials, IMV, and PSV.

There is considerable variation among clinicians in the manner of applying T-tube trials.[141,150] Some clinicians continue maximal ventilator support (*eg,* CMV with neuromuscular blockade, or A/C) up until the point at which they believe that a patient has a reasonable chance of extubation. This decision is usually based on clinical examination and measurement of physiologic indices. At this point, the ventilator is stopped and the patient breathes through a T-tube system. The duration of such a T-tube trial has never been standardized, and it varies from about 30 min to several hours. During the trial, a decision is made to extubate the patient (provided that problems with upper airway protection are considered unlikely) or to reinstate ventilator support. Some clinicians do not attempt another T-tube trial for $\geq$24 h after an unsuccessful attempt. Other clinicians employ intermittent T-tube trials of gradually increasing duration (from 5 to 60 min) intermittently (*eg,* 3 to 4 h apart); this is conducted on an empirical basis.

Intermittent mandatory ventilation was the first alternative approach to T-tube trials. IMV involves a gradual reduction in the amount of support being provided by the ventilator and a progressive increase in the amount of respiratory work being performed by the patient. The pace of decreasing the IMV rate is generally based on clinical assessment and measurement of arterial blood gas values, but precise guidelines do not exist. As discussed elsewhere in this consensus

conference, breathing through the demand valve of an IMV circuit can produce a marked increase in the work of breathing (see Section 4, B-3).

Pressure support ventilation can also be used to gradually decrease the level of ventilator support. The level of PSV is gradually decreased so that a patient becomes increasingly responsible for a larger proportion of overall ventilation. It is commonly assumed that the level of PSV can be decreased to a low level that will compensate for the resistance of the ET tube and circuit, and that the patient can then be extubated at that level of PSV. Unfortunately, there are no simple parameters than can predict the level of PSV that compensates for this resistance in an individual patient.

A gradual approach to the discontinuation of mechanical ventilation (*i.e.,* IMV or PSV vs abrupt T-tube trials) has two theoretical advantages: (1) the use of less positive pressure (since these are modes of partial rather than full assistance), and, thus, a potential for fewer pressure-related complications, and (2) performance of some level of respiratory work should prevent the development of respiratory muscle atrophy—this is mainly an advantage when contrasted with a patient receiving CMV with neuromuscular blockade, since patients being treated with A/C (with or without intermittent T-tube trials) probably perform sufficient work to prevent significant deconditioning.

In addition to the independent use of T-tube trials, IMV, or PSV as approaches to discontinuing ventilator support, these techniques are frequently integrated and specific protocols defined for a given patient in an attempt to establish the most optimal approach. That is, PSV and IMV have been combined, with both gradually decreased, or the level of one technique kept constant while the other is gradually decreased. T-tube trials have also been integrated with PSV and IMV.

At the present time and to our knowledge, no study has been published that has compared the optimal use of the three major techniques of discontinuing mechanical ventilation.

E. Treatment of the Difficult Patient

Discontinuation of mechanical ventilation poses considerable difficulty in a significant proportion of patients. These patients account for a disproportionate amount of healthcare costs, and they pose enormous clinical, economic, and ethical problems. In addition to the factors already discussed, several other issues need to be considered in these patients. Increasing ventilator support at night to ensure maximal rest is recommended. In a recent study, the institution of a ventilator-management team was shown to decrease the number of ventilator and ICU days. Nutritional support needs to be considered since these patients are dependent on a ventilator for a relatively long time. Uncontrolled, retrospective studies suggest that nutritional supplementation can expedite the discontinuation of mechanical ventilation, but this has yet to be examined prospectively. Likewise, respiratory muscle training appeared promising in an uncontrolled study, but its benefit has yet to be evaluated prospectively. Finally, a prospective study of biofeedback was shown to be particularly beneficial. However, skilled practitioners are not widely available, and the initial beneficial findings in this study need to be confirmed.

Review of Mechanical Ventilators

Introduction

There are three fundamentally different modes of ventilation available in the NICU: "pressure ventilators", "volume ventilators", and high frequency ventilators. They all serve to support adequate **ventilation** and **oxygenation**, but each has it's own particular niche.

Ventilation (CO_2 removal) is a function of minute ventilation which is respiratory rate (RR) multiplied by tidal volume (Vt).

MINUTE VENTILATION = RATE x TIDAL VOLUME

Arterial Oxygenation improves when either the fraction of inspired oxygen concentration (FiO_2) and/or mean airway pressure (MAP) are increased.

The first step in managing a patient on a ventilator is to choose appropriate goals for **ventilation** and **oxygenation** (i.e. blood gases). These goals depend on the patient's disease state. An otherwise healthy term infant intubated for choanal atresia might have as a goal pH = 7.40, $PaCO_2$ = 40, PaO_2 = 60. In a small preterm infant (<1000g), to minimize lung injury due to mechanical ventilation, a strategy of mild permissive hypercapnea may be followed. In a patient with severe chronic lung disease gases with $PaCO_2$ of 60-65 torr and SaO_2 >88% may be acceptable. In contrast, a patient with persistent pulmonary hypertension of the newborn might have as a goal pH>7.45, $PaCO_2$<30, PaO_2>100 in an attempt to attenuate hypoxic pulmonary vasoconstriction.

Ventilation goals can be a range of pH values and/or a range of $PaCO_2$ values. Extreme acidosis (pH<7.10) is to be avoided but otherwise mild acidosis alone appears to be relatively well tolerated. Of greater concern are wide swings in $PaCO_2$ which can have significant effects on cerebral blood flow. Also of concern is identifying and, if appropriate, treating the underlying cause of the acidosis. Ventilation can best be monitored using arterial blood gases. Capillary blood gases (and even more so venous) tend to give low values for pH (~0.05-0.1 lower depending on perfusion). The difference between arterial and capillary or venous pH is variable over time and between patients. As an estimate of ventilation, it does not work well in older infants with BPD, infants with hydrops, and other conditions that impair transcutaneous passage of capillary gas. If using a conventional ventilator (not High Frequency), end tidal CO_2 monitoring (capnography) is another valuable non-invasive method of **estimating** ventilation though it may give inaccurate readings with chronic lung disease (e.g. BPD).

Oxygenation goals can either be a range of arterial oxygen saturation or PaO_2 values. Oxygen saturation (SaO_2) best reflects arterial blood oxygen content (SaO_2 x Hemoglobin x 1.34) and thus is of direct physiologic interest. PaO_2 better reflects degree of shunt, and is more accurate than SaO_2 at the lower range. Oxygenation can best be monitored by pulse oximetry or arterial blood gases. Capillary and venous blood gases are never useful measures of arterial PaO_2.

The appropriateness of initial ventilator support needs to be **rapidly** confirmed by checking a blood gas (within 15-20 minutes if possible) and making adjustments accordingly. Initial ventilator settings for pressure ventilators are typically chosen based on what types of pressures and rates were required when hand bagging. Initial settings on volume ventilators are usually chosen based on typical minute ventilation requirements (e.g. rate of around 20-30 breaths per minute with tidal volume of ~4-6 mL/kg). When switching from conventional ventilation to high frequency ventilation, a rule of thumb is to choose a mean airway pressure (MAP) for the high frequency ventilator that is 2 cmH_2O greater than the MAP on the conventional ventilator. Amplitude starting point is chosen such that there is adequate shake (a rule of thumb being the umbilical line should be shaking slightly; alternative rule of thumb is to start at twice the MAP and back down from there until shake appears appropriate).

Blood Gases: When evaluating a blood gas, first determine your goals for pH, $PaCO_2$, and PaO_2. Secondly, determine the type of specimen to decide if any correction for capillary or venous specimen is in order. Finally, evaluate the blood gas to see if any changes in inspired oxygen concentration or ventilator settings are needed. Before analyzing the blood gas it is worth determining whether the gas is significantly different from previous gases and, if so, why (i.e. was a ventilator wean made, is the patient extubated, is there a pneumothorax, is the patient showing signs of sepsis, are there signs of persistent pulmonary hypertension of the newborn, is this an expected change given the patients diagnosis). If PaO_2 is low then FiO_2 or MAP need to be increased. If the pH is low, one should determine if the acidosis is respiratory ($PaCO_2$ high) and/or metabolic (calculated HCO_3 low). Increasing ventilation is merely a temporizing act **until the cause of the acidosis is determined**. If the pH is low and/or the $PaCO_2$ is high, indicative of a respiratory acidosis, then ventilation needs to be increased by increasing rate and/or tidal volume - how this is accomplished again varies from ventilator to ventilator.

Abbreviations:

ETT: Endotracheal Tube
PEEP: Positive End Expiratory Pressure
CPAP: Continuous Positive Airway Pressure, PEEP with no rate
PIP: Peak inspiratory pressure
MAP: Mean Airway Pressure
RR: Respiratory Rate
Ti,Te: Inspiratory and expiratory times
I:E: Ratio of inspiratory to expiratory time
Vt: Tidal Volume, volume of each breath
SaO_2: arterial oxygen saturation determined by arterial blood gas analysis
SpO_2: arterial oxygen saturation determined by pulse oximetry
FiO_2: Fractional inspired oxygen
HFV: High Frequency Ventilation
HFOV: High Frequency Oscillatory Ventilator/Ventilation
Amplitude: (aka Delta P) Setting on HFV. Difference between maximum and minimum airway pressure

Volume Ventilators

Historically, volume ventilators (time cycled, volume regulated, volume limited) were used in anesthesia (a bellows of defined tidal volume pumped at a given rate) and as pediatric and adult intensive care evolved. Initially these ventilators were not used in the NICU due to the difficulty achieving consistent small volumes (5-7 mL/kg in a 1200g infant!). Current volume ventilators are able to deliver small volumes consistently. In the past, triggering was inconsistent and increased the work of breathing. The latest generation (Siemens 300) has resolved these problems. In the NICU their use has been primarily in larger infants with chronic lung disease (partly because SIMV was only available on volume ventilators until recently) or perioperatively (tradition - likely related to familiarity of operating room personal with volume ventilators). Their use in the acute NICU setting has extended into the micropremie population.

PROS: Stable minute ventilation with known tidal volume. Simpler models available for use outside hospital setting. Control or SIMV modes are available. Home ventilators currently available are typically "volume ventilators".

CONS: Tidal volume is maintained at the expense of peak airway pressure. If lung compliance falls by 50% (i.e. ETT slipping down right mainstem) then to maintain tidal volume, peak airway pressure doubles, possibly increasing the risk of volutrauma or barotrauma. Since these ventilators do not have constant flow, to breathe spontaneously the infant always has to trigger a valve to allow airflow. Large leaks around the ETT can be problematic due to difficulty maintaining tidal volume and "triggering" (patient cycling) of the ventilator causing frequent alarming.

Adjusting ventilation/oxygenation:

To increase alveolar ventilation: **Increase Rate** or **Increase Tidal Volume**

MINUTE VENTILATION = **RATE** x **TIDAL VOLUME**

To increase oxygenation: **Increase FiO_2**, or **Increase PEEP**, or **Increase Tidal Volume**

OXYGENATION is improved by increasing **MAP** and/or **FiO_2**

/ \

Vt PEEP

High Frequency Ventilators

This is a radical innovation in ventilator design. The rate in "high frequency" is the Hz (range 3-15 Hz) (i.e. 180-900 breaths per minute). Since the tidal volume generated by these ventilators approximates dead space, simple pulmonary mechanics, physics, and physiology are inadequate to explain their operation. Gas exchange occurs by enhanced diffusion.

HFOV (High Frequency Oscillatory Ventilation): (Sensormedics 3100A) uses a piston with a diaphragm unit to actively move gas in and out of the lung. This type of ventilator requires a special non-compliant breathing circuit. Indications for use of high frequency ventilation are unclear but include:

1. Initial and subsequent ventilatory support in very low birth weight infants with respiratory distress syndrome.
2. Air leak (pneumothorax, pulmonary interstitial emphysema).
3. Failure of conventional ventilation (pre-ECMO step) particularly in persistent pulmonary hypertension of the newborn, meconium aspiration syndrome, pneumonia, pulmonary hemorrhage.
4. To reduce the risk of volutrauma and barotrauma when conventional ventilator settings are very high.

PROS: May allow gas exchange when conventional ventilation has failed.

CONS: Unclear which patients will respond and there is some risk involved in "just trying". Switching ventilators on an unstable patient who is failing conventional ventilation may result in clinical deterioration. The high airway pressures often seen with high frequency ventilation can be transmitted to the heart (particularly with compliant lungs) and result in impaired cardiac output requiring inotropes and/or volume boluses. HFOV makes turning patients, taking x-rays, or performing ultrasounds more complex due to the heavy, non-flexible tubing. Stopping HFOV for suctioning or administering nebulized medications may negate its benefit.

Adjusting ventilation/oxygenation: Ventilation is dependent on amplitude much more than rate. In larger infants paradoxically lowering Hz improves CO_2 removal on the Sensormedics. This is *postulated* to occur by increased tidal volume (more inspiratory time) and better gas escape (more expiratory time). Mean airway pressure primarily effects oxygenation. MAP can also influence ventilation: too high a MAP and ventilation may drop due to decreased compliance from overdistention, too low a MAP and hypoventilation may occur secondary to atelectasis.

To increase minute ventilation: **Increase Amplitude,** (although changes in MAP and Hz can *sometimes* have significant effects on CO_2 also)

MINUTE VENTILATION = RATE x TIDAL VOLUME
/ / \
Hz **AMPLITUDE** MAP

To increase oxygenation: **Increase FiO_2**, or **Increase MAP,** (change in Hz may *sometimes* effect oxygenation also). Note, however, over-distention can impair oxygenation. When in doubt, a chest x-ray is indicated.

OXYGENATION is proportional to **MAP** x **FiO_2**

Pressure Ventilators

These are the most frequently used ventilators in the NICU. Traditional "pressure ventilators" are constant flow, time cycled, pressure-limited devices. Constant flow implies that there is a constant flow of gas past the top of the endotracheal tube. Pressure limited means that once the pre-set PIP has been reached, it is maintained for the duration of the inspiratory cycle. Time cycled implies that breaths are given at fixed intervals, independent of the infant's respiratory efforts. Newer "pressure ventilators" can sense infant's breaths and synchronize to them. There may be some added work of breathing due to the need to trigger breaths - this has been hard to quantify and remains controversial.

PROS: The constant flow permits the infant to easily take spontaneous breaths. Simple, reliable mechanical design. Pressure limitation prevents sudden changes in PIP as compliance changes (i.e. on a pressure ventilator if compliance falls by 50% PIP does not change - though tidal volume drops, for example ETT slipping down right mainstem).

CONS: Variable tidal volume as lung compliance changes. Should lung compliance worsen then Vt will drop (if the ETT plugs Vt drops to zero, but the ventilator does not sense it). Should compliance improve (following surfactant for example) this may result in over-distention. If the child is exhaling during a non-synchronized ventilator breath, then the breath is ineffective.

Adjusting ventilation/oxygenation: Key determinants of oxygenation (MAP) and ventilation (tidal volume) are not directly adjustable, but are derived from related parameters (Bold faced). Adjustments are thus less straightforward than with either "High Frequency" or "Volume Ventilators". Furthermore, there are interactions between the various parameters. Driving pressure conceptually is similar to "amplitude" on high frequency ventilation, but is not directly adjustable: it is proportional to the difference between PIP and PEEP. To increase ventilation: **Increase Rate**, or **Increase PIP**, or **Increase Inspiratory Time**, or **Decrease PEEP** (rarely done)

MINUTE VENTILATION= RATE x TIDAL VOLUME
/ | \ / \
Ti Te I:E Driving Time
Pressure Constant
| / \
PIP-PEEP Resistance Compliance

To increase oxygenation: **Increase FiO_2**, or **Increase MAP** (see below)-- OXYGENATION is proportional to MAP x **FiO_2**

$$\mathbf{MAP} = \frac{(Ti \times PIP)+(Te \times PEEP)}{Ti + Te}$$

This equation **assumes** Pressure vs. Time is a square wave.

Ways to increase MAP (See figure 1):

1. Increase PEEP
2. Increase PIP
3. Increase Ti
4. Increase RR
5. Increase Flow

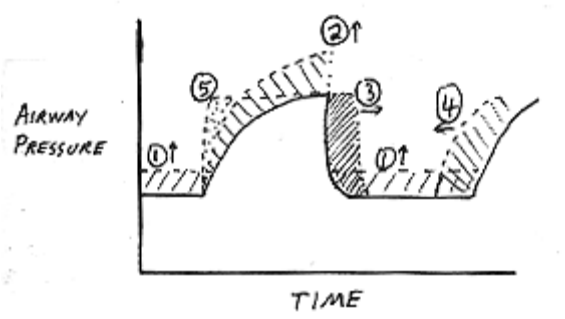


Figure 1: Pressure vs. time

Ventilator Modes

IMV: Intermittent Mandatory Ventilation. Intermittent breaths (fixed PIP or Vt) at a fixed rate. **Not synchronized** to patient. Beyond the set rate, the infant is on his/her own.(See figure 2-IMV) Standard on most ventilators, but infrequently used. Generally good for small premature infants but when rates are high (>60) or large infant "fighting" ventilator (exhaling during ventilator's inspiratory cycle), the lack of synchronization may impair ventilation.

CON: No synchronization.

SIMV: Synchronous Intermittent Mandatory Ventilation. Like IMV but **synchronized** (senses infant's spontaneous breaths). Beyond the set rate the infant is on his/her own (See figure 2-SIMV). Since it is synchronized to the patient's effort, it is the preferable mode. It will function exactly like IMV if the infant is apneic or the trigger/synchronization fails. Typically used in infants who can reliably trigger demand valve and those fighting a preset rate. Usually lower rates/pressures since at higher spontaneous rates (>60) may get inadvertent PEEP and air trapping. Also good for older ventilator dependent patients.

PRO: Synchronized to patient effort.

CON: None, at worst is like IMV.

AC: Assist/Control. **Synchronized** (senses infant's spontaneous breaths) but with **mandatory minimum set rate, all breaths** the infant takes are a **full assisted ventilator breaths** (See figure 2-AC) Used in more active ventilator dependent infants not aggressively being weaned.

PRO: Infant can increase minute ventilation easily on demand, based on need.

CON: When weaning can't wean rate, only PIP or Vt.

SIMV-PC with PS (Pressure Support): Term used on Servo 300 ventilators to describe an SIMV "pressure ventilator" with set PIP/PEEP. Beyond a set background ventilator rate, spontaneous breaths are augmented (supported) with pressure - usually relatively low values (+4 to +8 cmH_2O) (See figure 2-PC/PS). Uses: 1) To provide mandatory backup breaths (conceptually large sighs to prevent gradual progressive atelectasis) while allowing amount of PS to be weaned slowly to "train" respiratory muscles 2) As a means of providing intermediate respiratory support (less than conventional modes but more than CPAP or extubation) 3) Pressure support just enough to overcome resistance of ETT and ventilator circuit and maintain minimum adequate spontaneous ventilation. Uses as above, but in "pure pressure" support mode. (If set PIP and PS pressures are the same then essentially you have pressure AC mode).

NOTE ON WRITING PS: Pressure support is above PEEP. At the University of Washington NICU, writing an order for "PS 5, PEEP of 4" yields inspiratory pressures for assisted breaths of 5+4=9.

SIMV-VC with PS (Pressure Support): Term used on Servo ventilators to describe SIMV with set Vt. Beyond the set rate, spontaneous breaths are augmented (supported) with pressure - usually relatively low values. The difference between this mode of ventilation (VC/PS) and the mode described above (PC/PS) is that in VC/PS the SIMV breaths are volume breaths and in PC/PS mode the SIMV breaths are pressure breaths.

PC: Pressure Control. Term used on Siemens Servo ventilator to describe AC mode with a set PIP/PEEP ("pressure ventilator" AC mode).

VC: Volume Control. Term used on Siemens Servo ventilator to describe AC mode with a set Vt ("volume ventilator" AC mode).

PRVC: Pressure regulated volume control. In this mode, a volume is set and the delivered pressure self adjusts to achieve the set volume. With the Servo 300, the pressure will stairstep up over 5 breaths until the set volume is met. If apnea occurs, the ventilator sounds an audible alarm and switches to the PC backup mode. The therapist must manually change back to PRVC mode.

PSVG: Pressure support volume guarantee. This mode is available on the Drager Babylog 8000. This mode is pressure limited with a set tidal volume. The pressure will stairstep up to meet the set tidal volume. There are two sets of values: Set (ordered) and Measured (spontaneous). Set values include tidal volume (4-8 mL/kg), inspiratory time, inspiratory pressure limit (PIP), rate, and PEEP. The set values are utilized when the infant is apneic. Otherwise, the infant regulates their own PIP to meet the set tidal volume. As infant's compliance improves, the PIP needed to deliver set tidal volume decreases.

Pro: Adjusts for compliance automatically, compensates for ETT leaks, no need to correct for tubing volume

Con: Weaning mode only. If infant needs increasing support, switch to another mode on ventilator.

SIMV + VG: With the Drager Babylog 8000, the addition of VG (volume guarantee) to SIMV allows one to control the inspiratory time. The PIP still adjusts to meet the set tidal volume, but the inspiratory time is set by the therapist.

Pro: More supportive and more control of ventilation than with PS + VG.

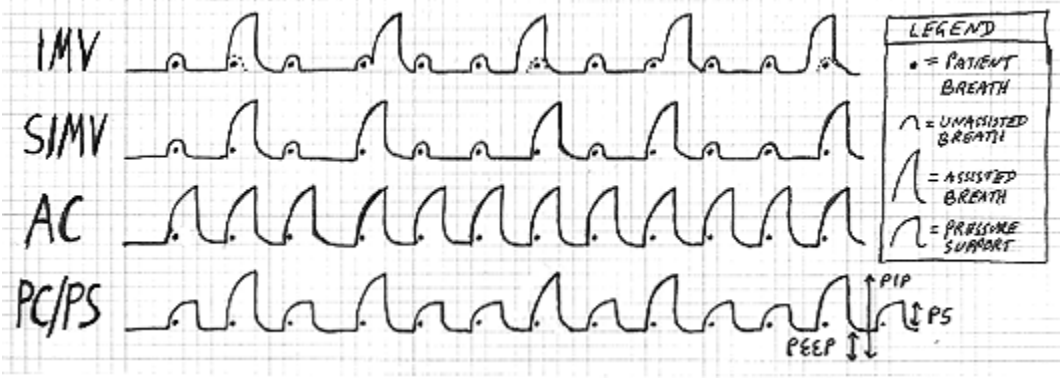
Con: Less control over PIP, infant is still doing most of the work of breathing.

CPAP: Continuous Positive Airway Pressure (like PEEP). Primarily used to maintain airway distending pressure; major effect is to help to maintain lung volume and improve oxygenation. Can be administered via ETT or nasal prongs. Uses: 1) To prevent alveolar collapse in mild HMD (perhaps avoiding intubation) 2) In mild chronic lung disease (perhaps avoiding reintubation) 3) In severe apneic spells to avoid reintubation. Sometimes used in infants as prelude to extubation to ensure adequate respiratory drive. If done for a prolonged time, infants tire out breathing through the relatively high resistance of a 2.5-3.5 ETT.

PRO: Improve oxygenation by maintaining functional residual capacity.

CON: Impair ventilation by increasing FRC and increasing work of breathing (exhaling against pressure).

FIGURE 2: RESPIRATORY PATTERN UNDER DIFFERENT MODES



Specific Ventilators

NOTE: *Each ventilator manufacturer has utilized specific names for mode functions of their specific machine that may not be identical with other machines. For example, PSV of the Servo 300 is not the same as PSV of the Drager Babylog 8000.*

Drager Babylog 8000: This ventilator is specifically designed for infants up to 10 kilograms (22 pounds). It is capable of both volume and pressure ventilation. A flow sensor at the patient wye accurately measures tidal volume and senses air flow initiated by the patient allowing triggering of the ventilator cycle. The sensor is able to compensate for small ETT leaks. The Drager Babylog 8000 provides the following modes: AC, SIMV, PSV (pressure support ventilation), Volume guarantee (VG), and independent Expiratory Flow (VIVE). VG is often used with SIMV, PS, and AC. The most important and commonly used modes are SIMV, PSV, VG, and CPAP.

Sensormedics 3100A: High frequency oscillatory ventilator with active inhalation/exhalation driven by a moving piston and diaphragm. Requires special stiff non-compliant ventilator circuit. Can be utilized for a wide weight range of infants. Some preliminary work using it in smaller infants suggests that it may result in less barotrauma than conventional ventilation.

Siemens Servo 900C: Either a volume or pressure ventilator, **no gas flow from ventilator between breaths.** Volume ventilator with IMV, SIMV, AC and pressure modes PC/PS. Has pediatric settings for alarm limits, but no specific **infant** modes. Used when primarily volume ventilator needed in larger term infant and often for home ventilatory support. Currently, used only in the IICU.

Siemens Servo 300: Either a volume or pressure ventilator, **low bias flow from ventilator between breaths.** Does not have continuous high flow through circuit and requires some effort

on the part of the infant to trigger significant flow. A "bias" flow of 0.5 LPM is present and the trigger is a 30% disruption in this flow rate sensed at the ventilator. Alternatively it can sense a drop in airway pressure instead, but this requires more effort on the infant's part. The major advantage over Siemens 900C is that it has **infant** ranges for Vt, flow, pressures, and alarms. Has extensive list of modes: PC, VC, SIMV-VC+PS, SIMV-PC+PS, CPAP and PRVC (pressure regulated volume controlled). Uses include 1) Volume ventilation of small infants, 2) Synchronized/mixed modes, 3) Overcoming resistance of circuit & ETT with PS, 4) Facilitation of weaning by allowing

Review:
Ventilatory Management/ Modes of Support

The goals of mechanical ventilation are to relieve respiratory distress, decrease the O_2 cost of breathing, improve pulmonary gas exchange, reverse respiratory muscle fatigue, enable the lungs to heal, and avoid complications. The most common modes of mechanical ventilation are **assist-control (AC), intermittent mandatory ventilation (IMV), and pressure-support ventilation (PSV).**

- In the **AC mode**, the ventilator delivers a breath either when triggered by a patient's inspiratory effort, or independently if such an effort does not occur in a preselected time.

- **Characteristics:** preset rate and tidal volume (sometimes PIP), either on the patient's initiative or at the set interval a full mechanical breath is delivered.
- **Uses:** for patients who have a very weak respiratory effort, allows synchrony with the patient but maximal support. Not a weaning mode, as at any rate they are getting complete mechanical support.
- **Contraindications:** none in particular
- **Advantages:** a fairly comfortable mode, providing a lot of support
- **Disadvantages:** can lead to hyperventilation if not closely monitored, not able to wean in this mode.

- In the **IMV mode**, a patient receives periodic positive-pressure breaths at a preset volume and rate from the ventilator, and, in addition and unlike in AC, can breathe spontaneously. Spontaneous breathing is achieved through a demand valve that can produce a considerable increase in the work of breathing.

- **Characteristics:** set breath delivered at a fixed interval. No patient interaction, pressure or volume modes
- **Uses:** commonly in neonates on the Sechrist, can be a weaning mode
- **Contraindications:** none really, unfriendly to older patients
- **Advantages:** regular guaranteed breath
- **Disadvantages:** does not allow patient to breath with the ventilator except by chance. Does not work with the patient
- **Ventilators:** Sechrist, most others can do this as well.

• In the **PSV mode**, a fixed amount of pressure augments each breath. Airway pressure is maintained at a preset level until the patient's inspiratory flow falls to a certain level (for example, 25% of peak flow). Tidal volume is determined by the level of PSV, patient effort, and pulmonary mechanics. Newer ventilator modes such as inverse-ratio ventilation, airway pressure release ventilation, and extracorporeal CO_2 removal are experimental.

- **Characteristics:** supports each spontaneous breath with supplemental flow to achieve a preset pressure. Gives a little push to get the air in, so to speak.
- **Uses:** In the spontaneously breathing patient this helps overcome the airway resistance of the endotracheal tube. Usually use 5 for older patients and 10 for smaller (smaller ETT has higher resistance, more impediment to flow). Can be very helpful for weaning.
- **Contraindications:** patient who is not spontaneously breathing, i.e. on muscle relaxants
- **Advantages:** helps overcome resistance of tube, making spontaneous breathing easier
- **Disadvantages:** the flow rate is very high on the Servo 900C, which can make pressure support uncomfortable for some small patients. This is hard to predict.

Here is a partial list of other modes, in addition to the ones above. Unless otherwise mentioned these modes are all in volume control, meaning that you set the tidal volume, rather than the peak inspiratory pressure. Some newer ventilators, particularly the Servo 300 can do these modes in either pressure or volume control.

⇒ **SIMV (Synchronous IMV)**

- **Characteristics:** set breath delivered within an interval based on the set respiratory rate. Ventilator spends part of the interval waiting for spontaneous breath from the patient, which it will use as a trigger to deliver a full breath. If not sensed it will automatically

give a breath at the end of the period. Any other breaths during the cycle are not supplemented.

- **Uses:** commonly used in many settings. Can be a weaning mode (see also with PS)
- **Contraindications:** none in particular
- **Advantages**: allows work with the patient, somewhat more friendly.
- **Disadvantages:** Any other breaths during the cycle are not supplemented
- **Ventilators:** all but the Sechrist

⇒ **SIMV/PS**

- **Characteristics:** combination of the previous two modes. Extra breaths in the cycle are supplemented with pressure support.
- **Uses:** useful in most circumstances, including weaning.
- **Contraindications:** none in particular.
- **Advantages:** allows both synchrony with the patient and help in overcoming the resistance in the endotracheal tube, to allow easier spontaneous breathing.
- **Disadvantages:** none in particular. PS does not add anything in the patient who is not spontaneously breathing. Sometimes patients will have difficulty with the pressure support on some ventilators.
- **Ventilators:** all but the Sechrist

⇒ **PC (Pressure Control)**

- **Characteristics:** basically IMV, where the breath is controlled by the Pmax or Swing pressure and not the set tidal volume
- **Uses:** in neonates, or in patients with high airway pressures(such as ARDS) to avoid barotrauma
- **Contraindications:** none in particular, not a friendly mode in an awake patient
- **Advantages:** Pressure limited, decreases the risk of barotrauma
- **Disadvantages:** no guaranteed tidal volume
- **Ventilators:** all.

⇒ **PRVC (Pressure Regulated Volume Control)**

- **Characteristics:** a volume controlled Assist-Control mode, that adjusts the flow rate of the delivered air to deliver the set tidal volume at or below the set maximum pressure.
- **Uses:** in patients with high airway pressures, but can be used in any patient
- **Contraindications:** none in particular
- **Advantages:** gives you a guaranteed tidal volume but minimizes barotrauma. Some authors feel this is a more friendly mode for awake patients.
- **Disadvantages:** new, no particular disadvantages.
- **Ventilators:** only available on the Servo 300

⇒ **CPAP (Continuous Positive Airway Pressure)**

- **Characteristics:** just as it says. This is the same as PEEP.

- **Uses:** for patients with upper airway soft tissue obstruction or tendency for airway collapse. As a final mode prior to extubation in some patients.
- **Contraindications:** any patient without spontaneous respiratory effort. Not a good idea in a patient with obstructive pulmonary disease (like asthma, COPD)
- **Advantages:** simple, easy to use
- **Disadvantages:** provides no supportive ventilation.
- **Ventilators:** all.

Where to start?

Every patient is different and it is hard to know exactly what a patient will need in terms of ventilatory support until they are actually on the ventilator. So many of us a have preset ideas as to where to start any patient and then adjust the ventilator afterwards to achieve the desired ventilation effect.

- **Pressure vs. Volume:** Choose Volume to start.

Why? generally a more friendly method, more to choose from, also you have the benefit of the guaranteed tidal volume which is important, especially early

- **Mode:** SIMV with or without Pressure Support.

Why? again a patient friendly mode. Pressure support is only helpful if the patient is going to be spontaneously breathing. Would use PS of 5 in big patient, 10 in small.

- **Rate:** 20

Why? A good place to start. You can always adjust later. For small children this is lower that their usual spontaneous rate but with the larger tidal volumes that are delivered this increases the minute ventilation.

- **PEEP:** 5mm Hg

Why? a little above physiologic. Not so high as to cause problems.

- **FiO_2:** 100%

Why? You can start to wean once you are certain everything is stable. Allows maximal pre-oxygenation in case anything happens.

- **Tidal Volume:** 10ml/kg

Why? Above physiologic, gives good distention without significant barotrauma. 10-12ml/kg is the standard range.

- **Inspiratory Time:** somewhere from 0.5 to 1 second

Why? physiologic. Longer for bigger kids. But this will vary on the situation. Asthmatics for example merit very short I-times to allow maximal time for exhalation.

Early things to worry about

Peak Pressures: You would like to keep these under 40 if at all possible. If they start climbing into the higher 40's to 50's you should consider changing to Pressure control ventilation.

Oxygenation: Inability to wean the FiO_2 should be a concern. Once on the ventilator the goal should be to get the FiO_2 under 60%. If you are unable to do this it implies shunting either from lack of airway recruitment (PEEP too low) or alveolar inflammation or disease (like ARDS). This is where increasing the Mean Airway Pressure will be of benefit.

Ventilation: Am I over or under ventilating this patient based on his needs. Remember a patient who is being intubated because of an upper airway problem may have an excellent respiratory drive and not need much support. While a patient in shock with profound metabolic acidosis may need a higher rate to help compensate. Keep in mind the reason you are putting the patient on the ventilator. Obtaining a blood gas early after intubation (15-20 minutes after being on the ventilator) will help you decide if you are moving in the right direction.

Blood Gases

The simplest way to look at mechanical ventilation is as a way to keep the blood gases normal.
So what makes up a Blood Gas?

- **pH** hydrogen ion concentration
- **pCO_2** partial pressure of carbon dioxide
- **pO_2** partial pressure of oxygen

(Note: for more information on blood gases, see course number: 990614, Oxygen Transport and Interpretation of Blood Gas Data, or course number: 990711, Interpretation of Arterial Blood Gases.)

You get several other values, but many of these are calculated and/or not reflective of pulmonary function which is what you are controlling with MV.

- **pH** and **pCO_2** are closely related and are affected by minute ventilation.
- **pO_2** is governed by oxygen delivery and ventilation and perfusion (V and Q) match.

Minute Ventilation

- Very simply is RRxTV, respiratory rate times tidal volume.
- Because CO_2 rapidly diffuses across the alveolar space the more air you can move into and out of the lungs the more rapidly the CO_2 can be removed.

Oxygen Delivery and **VQ** match - is controlled by,

- Your FiO_2 (fraction of inspired oxygen)
- And is related to your airway recruitment. Airway recruitment is indirectly reflected in your mean airway pressure (MAP). By increasing your mean airway pressure you can increase your airway recruitment (although this is not a linear relationship) MAP is a function of the PEEP (positive end expiratory pressure) and a fraction of the PIP (peak inspiratory pressure or Pmax).

Quick review

• So, to control **pH** and **pCO_2**, you manipulate the minute ventilation, ergo the respiratory rate and tidal volume.

• To control **pO_2**, you manipulate the oxygen delivery and the VQ match, ergo you adjust the FiO_2 and the mean airway pressure (PEEP and PIP)

Ventilator settings are based on a patient's size and condition and are repeatedly reassessed. FiO_2 is initially set at 0.9 to 1.0, and then adjusted to achieve a PaO_2 of 60 to 90 mm Hg. Tidal volume is commonly set at 10 to 15 mL/kg; recent evidence, however, suggests that large volumes may induce or aggravate lung injury, and a lower volume (7 to 8 mL/kg) may be preferable. Ventilator rate depends on the mode of ventilation: with AC, the back-up should be 2 to 4 breaths/min below the total rate; with IMV, it should initially be high and then gradually decreased in accordance with the patient's tolerance; and with PSV, the rate is not set. An inspiratory flow rate of 60 L/min is optimal for most patients, but a higher value may be preferred in patients with COPD to decrease work of breathing and improve gas exchange.

PEEP usually improves oxygenation in patients with acute diffuse pulmonary infiltrates who have hypoxemia associated with reduced lung volume. It raises end-expiratory lung volume by opening airways and recruiting collapsed alveoli, thus enabling inspiration to occur on the steep portion of the pressure-volume curve. These changes combined with the redistribution of excess fluid within the lungs reduce intrapulmonary shunt. The goal of PEEP is to alleviate life-threatening hypoxemia and decrease exposure to a potentially toxic FIO_2. Titration of PEEP should not be guided solely by PaO_2, because improvement in PaO_2 may be offset by a simultaneous fall in cardiac output. Instead, optimal PEEP is achieved by maximizing O2 delivery at the lowest FIO_2 setting. In general, this can be achieved with a PEEP of 5 to 15 cm H_2O.

Positive-pressure ventilation and PEEP may produce various complications. A decrease in cardiac output is primarily due to a reduction in venous return. Additional factors include an increase in pulmonary vascular resistance from the stretching of intra-alveolar vessels, and a decrease in left ventricular compliance as a result of leftward movement of the interventricular septum and an increase in juxta-cardiac pressure from the distended lung.

Conversely, in patients with poor myocardial function, positive-pressure ventilation may increase cardiac output due to a decrease in left ventricular afterload. The development of extra-alveolar air in the form of pneumothorax, pneumomediastinum, or subcutaneous emphysema is termed barotrauma. However, this is probably a misnomer because alveolar overdistention rather than high peak airway pressure appears to be the primary mechanism.

The classic apicolateral location of a pneumothorax is less common in ventilator-dependent patients (largely due to their posture), with the result that the diagnosis is often missed. If an air leak persists for longer than 24 hours after a chest tube has been inserted, a bronchopleural fistula probably exists. The consequences of such a fistula include failure of lung expansion; loss of tidal volume and PEEP, with worsening gas exchange; unwanted cycling of the ventilator because chest-tube suction pressure may be transmitted to the airway and trigger the machine; and pleural infection.

Pneumonia occurs in approximately 30% of patients receiving ventilator support; the risk increases with the duration of ventilator support, at a rate of about 1% per day. Ventilator-associated pneumonia is associated with a high mortality; in a series of patients receiving mechanical ventilation for more than 72 hours, 71% of patients developing pneumonia died versus 29% of those without pneumonia.

A**n important aspect of Mechanical Ventilation is knowing how to avoid complications for the patient on the ventilator. Here we present you with that information:**

How to Ward off Complications of Mechanical Ventilation

If you care for a patient who relies on mechanical ventilation, the therapy itself poses a threat. The trade-off for this lifesaving treatment may be a serious complication.

Here we review problems that can develop even in well-managed ventilator patients--pneumonia, atelectasis, barotrauma, oxygen toxicity, and weaning problems--spells out measures you can use to minimize your patient's risk of developing them.

Inroad to Pneumonia

Mechanical ventilation itself helps lay an inroad to pneumonia: The endotracheal (ET) tube or tracheostomy bypasses the airways' first lines of defense, the nasopharynx and oropharynx. Bacteria can enter through the suctioning tube or migrate around the ET tube. Compounding the risk is the fact that patients on ventilators are acutely or chronically ill and may have little ability to resist infection.

Besides the inherent risks of tracheal intubation, the following problems can also increase your patient's risk of pneumonia:

- *Immunosuppression* may occur with chronic disease or if he receives broad-spectrum antibiotics, steroids, or chemotherapeutic agents.
- *Altered consciousness, pain,* and *bed rest* can decrease his ability to move and cough, making him more likely to retain secretions that could consolidate in his lungs. In the warm, moist lung environment, secretions are an ideal breeding ground for bacteria.
- *Chronic disease* and *malnutrition* can decrease his immune response and hamper healing.

Signs of pneumonia may be mild and nonspecific, such as low-grade fever or increased respiratory rate, or more overt, such as refractory hypoxemia. Changes in lung sounds are an early red flag: Secretions pooling in a dependent area decrease breath sounds in that area. You may also hear crackles, especially after your patient coughs or takes several deep breaths, because these maneuvers help reopen smaller airways and alveoli. If you detect abnormal breath sounds or crackles, act quickly to reduce the possibility of pneumonia.

Prevention tips

- Perform chest physiotherapy: vibration; palpation; assistive cough techniques; deep breathing; positioning; and forced expiration, which is done by exhaling once or twice from a normal lung volume (not a deep breath). This technique mobilizes secretions but causes less intrathoracic pressure than coughing and less risk of airway closure that could trap secretions.
- Meticulously wash your hands. Wear sterile gloves for open-system suctioning, clean gloves for closed suctioning.
- Manage your patient's pain so he doesn't guard his breathing.
- Use aspiration precautions, such as elevating the head of his bed to reduce the potential for bacteria to migrate from the esophagus and stomach.

When things get heavy

If your patient's secretions continue to increase, his larger airways become blocked and he coughs more. Because of the heavy sputum production, you may hear gurgles when you auscultate his lungs. At this point, he needs aggressive pulmonary intervention: Maximize humidity, increase his fluid intake, turn and position him to mobilize secretions, and suction frequently to prevent further airway obstruction.

Continued airway blockage causes atelectasis, or collapsed alveoli. As a result, your patient develops hypoxemia, his tissue perfusion decreases, and his sympathetic nervous system tries to compensate by stimulating heart rate, breathing, and blood pressure (BP). However, if hypoxemia progresses to critical levels (SpO_2 less than 90%), the myocardium becomes hypoxic, cardiac output decreases, and BP falls.

Certain diagnostic tests can confirm the presence of pneumonia. Your patient's white blood cell (WBC) count may be elevated as bacterial invasion triggers inflammatory and immune responses. A chest X-ray or computed tomography scan can reveal lung infiltrates. A sputum culture can help identify the pathogen and appropriate antibiotics.

If testing indicates that your patient has pneumonia, continue prevention measures, such as positioning, suctioning, and monitoring, and administer antibiotics as ordered. Monitor for adverse medication effects, including renal and hepatic toxicity. Assess for desired effects as well, keeping in mind that antibiotics generally don't take effect for 24 hours or more. Signs that the therapy is working include a change in sputum characteristics, thinning secretions, resolving fever, improved arterial blood gases or oxygen saturation, and loosening breath sounds (coarser crackles, less wheezing, and diminished sounds).

Atelectasis:

A case of collapsePreventing atelectasis is key, especially if your patient has pneumonia or he's recently undergone surgery. Atelectasis, the collapse of alveoli, can occur as a result of inadequate tidal volume or secondary to pneumonia. When the alveoli are under-inflated or partially blocked by secretions, gas exchange decreases. A decreased oxygen saturation level is an early sign; breath sounds are diminished in the area of alveolar collapse. Other signs include

reduced lung compliance, as indicated by high airway pressures and frequent high-pressure ventilator alarms, and refractory hypoxemia even when $F_I O_2$ and tidal volume are increased.

A chest X-ray can help pinpoint areas of atelectasis. An increased WBC count may indicate trapping of infectious secretions that can lead to pneumonia.

Prevention tips

- Your patient in coughing, deep breathing, and forced expiration.
- Perform suctioning as needed.
- Manage his pain.
- Change his position every 1 to 2 hours. If possible, help him sit in a chair.
- Consider using positive end-expiratory pressure (PEEP), which helps keep alveoli inflated. However, use it cautiously because moderate to high pressures can injure lung tissue.

Barotrauma: When treatment injures

Injury to the lung tissue from mechanical ventilation occurs because of excessive pressure or excessive tidal volume. (See *The Push and Pull of Breathing*.) Generally referred to as barotrauma, the damage can range from ruptured alveoli and blebs to a pneumothorax (an injury that occurs in 4% of all ventilator patients). Often, the damage occurs because the lungs are already diseased or the patient has secretion buildup in his airways.

Chronic obstructive pulmonary disease plus mechanical ventilation add up to a high risk of barotrauma: Chronic disease weakens lung structures and makes them more susceptible to injury. Asthma, which leads to air trapping and increased airway pressures, also increases the risk of injury.

Continuous positive airway pressure (CPAP) and PEEP may trigger barotrauma by keeping the alveoli inflated under pressure. Anxiety may also contribute: If your patient becomes anxious and hyperventilates while on the ventilator, the increased rate, pressure, and tidal volume may contribute to increased airway pressure.

Subtle signs of barotrauma include changes in sputum quantity and color. More prominent signs include diminished breath sounds, increased respiratory rate, and a decreased oxygen saturation level. With pneumothorax, your patient may have chest pain or pain on inspiration, restlessness, anxiety, diminished or absent breath sounds, dyspnea, tachycardia, hypoxemia, and increased peak inspiratory pressure. A chest X-ray or computed tomography scan can help establish a differential diagnosis.

Treatment for barotrauma includes lower airway pressures or alternative forms of mechanical ventilation, such as pressure-support ventilation or high-frequency jet ventilation using low tidal volumes and a rapid respiratory rate. For a pneumothorax, the physician may insert a chest tube.

Prevention tips

- Maintain oxygenation with minimal positive pressure, using low levels of PEEP and pressure support.
- Prevent hyperventilation. Relaxation exercises, music therapy, and sedation can help reduce your patient's anxiety and, as a result, his airway pressure.

Oxygen Toxicity: Too much of a good thing

A patient who receives oxygen at concentrations greater than 70% for as little as 16 to 24 hours is susceptible to oxygen toxicity. The problem begins when prolonged hyperoxygenation triggers production of excess oxygen free radicals, active compounds that normally help destroy bacteria. Coach But in the presence of oxygen and inflammation, oxygen free radicals oxidize fatty acids in cell membranes, impairing cellular metabolism. Gas diffusion and surfactant activity decrease, and the patient's risk of alveolar fibrosis and pulmonary edema increases.

A patient suffering from oxygen toxicity may experience fatigue, lethargy, weakness, restlessness, nausea and vomiting, anorexia, coughing, and dyspnea. Later signs include severe dyspnea, tachypnea, tachycardia, decreased breath sounds, crackles, and cyanosis. Risk factors for oxygen toxicity include acute respiratory distress syndrome, sepsis, shock, hypoxemia, and ischemia.

Prevention tips

- Choose the lowest oxygen setting possible to prevent hypoxemia.
- Use PEEP with pressure support ventilation to increase oxygen diffusion.
- If your patient's $F_I O_2$ ventilator setting is greater than 40%, use continuous pulse oximetry to monitor SpO_2. If his SpO_2 level falls below 94%, obtain arterial blood gases to monitor for pH imbalances.
- Perform aggressive pulmonary hygiene, which helps minimize the need for oxygen support.

Weaning difficulty: Trouble backing off

Even if your patient's primary problem improves, he may have trouble decreasing dependence on mechanical ventilation, especially if his cardiovascular status is unstable. For example, consider what can happen if you attempt weaning a patient with heart failure: Extubation causes his intrathoracic pressure to quickly change from positive to negative, pulling blood from the peripheral vessels into the vena cava and possibly overwhelming his heart. The sudden volume overload also causes fluid to back up into his lungs, causing pulmonary edema and requiring continued mechanical ventilation.

Other problems that may interfere with your patient's ability to tolerate weaning are underlying respiratory disease, poor nutrition, or neuromuscular dysfunction. Or he simply may not be emotionally ready.

Prevention tips

- Begin weaning soon after mechanical ventilation is initiated, using minimal support. Synchronous intermittent mandatory ventilation will allow your patient to initiate breaths on his own to help maintain respiratory muscle strength. To help him progressively breathe more independently, decrease the respiratory rate. Some ventilators can be set to deliver CPAP to partially support ventilation and maximize oxygen delivery before extubation.
- If your patient has cardiovascular disease, optimize his cardiac output before weaning attempts: Maintain fluid balance, decrease cardiac workload, and attempt weaning when his medications are at peak effectiveness.
- Provide adequate nutrition so he'll have the muscle mass and energy he needs to resume the work of spontaneous breathing. Tube-feeding or parenteral nutrition helps meet his nutritional requirements for successful weaning.
- Minimize use of sedating medications, such as opioids and long-acting benzodiazepines. Although sedation helps prevent self-extubation and reduce anxiety, sedatives can also have long-term effects on respiratory drive.
- Provide psychological support. Teaching the patient and his family and staying with him to coach breathing help decrease anxiety associated with weaning.

Getting ready

Respiratory distress and hypoxemia signal weaning difficulty, so when you attempt to wean your patient, monitor for tachycardia, dyspnea, a respiratory rate greater than 30, crackles on auscultation, and an SpO_2 level below 92%. Be on guard for changes in his cardiovascular status, especially if he has compromised heart function: an increase in heart rate of 10 to 20 beats/minute, a decrease in BP of more than 10 mm Hg, and decreased cardiac output evidenced by slow capillary refill, cyanosis, tachycardia, hypotension, and decreased urine output. If he develops these signs, stop the weaning effort and optimize his hemodynamic status before further weaning attempts.

In spite of your best efforts, chronic disease or setbacks may cause your patient significant difficulty weaning from mechanical ventilation. If he repeatedly fails weaning attempts, he may benefit from a long-term program designed to implement gradual weaning over 3 to 4 weeks. The respiratory care, social service, or discharge-planning department can help him get into the program.

Lifesaving Support

Mechanical ventilation supports your patient's respiratory system during acute failure. Although it's a lifesaving intervention, it can also cause serious complications. With prevention, early detection, and effective management of these problems, you can improve your patient's outcomes and his chance of survival.

LEARNING THE LINGO

Selecting the appropriate ventilator settings and modes helps reduce the risk of complications during mechanical ventilation.

Settings: adjusting the controls and alarm conditions

- respiratory rate (RR): breaths per minute
- tidal volume (V_T), or volume of gas delivered: milliliter per breath, liter per minute
- flow rate: the speed at which air enters the lungs
- pressure setting: maximum airway pressure delivered on inspiration (cm H_2O)
- oxygen (O_2): percentage of oxygen delivered with each breath ($F_{I\,O\,2}$, the fractional measure, is written as a decimal. For example, O_2 = 70%; $F_{I\,O\,2}$ = 0.70.)

Modes: adapting the ventilator's functions to the patient

Standard modes

- *continuous mandatory ventilation.* The patient receives a preset V_T at a preset RR; the cycle doesn't adapt to his spontaneous efforts.
- *assist/control ventilation.* This mode replaces continuous mandatory ventilation on newer ventilators. The ventilator adapts to the patient's spontaneous breaths and supports inspiration if his spontaneous breathing rate doesn't reach preset levels.

Expanded modes

- *intermittent mandatory ventilation (IMV).* The patient can increase RR, but each spontaneous breath receives only the V_T he generates. Mechanical breaths are delivered at preset intervals and a preselected V_T, regardless of the patient's efforts.
- *synchronized intermittent mandatory ventilation (SIMV).* This method is similar to IMV except the ventilator won't trigger while the patient is exhaling, so the potential for barotrauma is reduced.
- *pressure support ventilation.* This adjunct to SIMV applies a pressure plateau to the airway throughout patient-triggered inspiration to decrease resistance by the tracheal tube and ventilator tubing.
- *positive end-expiratory pressure (PEEP).* Used in conjunction with other modes, PEEP helps prevent premature closing of alveoli by maintaining a constant positive pressure.
- *continuous positive airway pressure (CPAP).* This mode produces a result similar to PEEP by augmenting spontaneous ventilation with increased end-expiratory pressure.

- *bilevel positive airway pressure (BiPAP).* This is CPAP with two preset pressure levels: one for inspiration, one for expiration.
- *inverse-ratio ventilation.* This mode recruits more alveoli by reversing the normal inspiration/expiration ratio of 1:2 to 2:1 or 4:1, making the inspiratory phase longer.

High-Frequency Modes

- *high-frequency jet ventilation.* Using a special ventilator, this method delivers RRs of 240 to 660 breaths/minute.
- *high-frequency oscillation ventilation.* A specialized ventilator vibrates (oscillates) an air column at a frequency of 480 to 1800 cycles/minute.

PREVENTING PROBLEMS

Take these precautions against ventilator-related complications:

- Get the patient moving. Turn and position him at least every 2 hours and help him sit in a chair at least twice daily. If possible, begin ambulation with a portable ventilator. If he can't get out of bed, use pulmonary physiotherapy. This intervention alone discourages development of pneumonia by moving secretions and preventing consolidation.
- Clear the ventilator tubing frequently to reduce resistance and minimize the medium for organism growth. Empty the tubing circuits to prevent water buildup and use self-sealing water traps to prevent 'breaking' the circuit. Minimize circuit change and opening of circuits. Use closed suction systems.
- Monitor the hemodynamic effects of mechanical ventilation because changes in support affect cardiac activity. To optimize your patient's fluid volume status, maintain fluid balance and administer diuretics (when ordered) before weaning attempts.
- Suction only as needed to eliminate secretions. Increase the ventilator oxygen level, but not the respiratory rate, before suctioning. Limit suctioning time to 10 seconds, and use no more than three passes to decrease the threat of trauma to the airways and suctioning-induced hypoxia.
- Avoid using 0.9% sodium chloride solution when suctioning or use it only for thick, tenacious secretions. It pushes secretions back down the endotracheal tube or tracheostomy and can trigger coughing and bronchospasm that cause hypoxia and barotrauma.
- Whenever you suction, assess and document the quantity, thickness, color, and odor of sputum.
- Ensure proper nutrition. Tube-feeding is preferred, but parenteral nutrition is also an option. Start nutritional support as early as possible.

- Provide adequate hydration to keep secretions thin and mobile.

THE PUSH AND PULL OF BREATHING

Spontaneous breathing: The lungs pull in air

Contraction of the diaphragm and respiratory muscles creates negative pressure in the pleural space. As a result, the lungs are pulled outward, enlarging and creating a vacuum that draws in air. The negative pressure also augments venous return to the heart.

Mechanical ventilation: Air is pushed in

Mechanical ventilation uses positive pressure to force air into the lungs. The positive pressure can damage lung tissue, particularly the alveoli, and decrease venous return to the heart.

An important aspect of Mechanical Ventilation is knowing how/and when to "wean" a patient off of the ventilator. While we just discussed some aspects of that, here we present you with more information, including a case study:

Weaning Patients from Mechanical Ventilation

Since more patients today are being weaned off mechanical ventilation in settings outside the intensive care unit. Here's what you need to know to make the process go smoothly.

Although most patients requiring mechanical ventilation remain on a ventilator for 7 days or less, some require support for several weeks or more. To control costs, institutions are increasingly inclined to move stable ventilator-dependent patients out of the intensive care unit (ICU) to other units or facilities for weaning from ventilator dependence.

The trend makes sense clinically as well as financially. Recent studies suggest that most patients on long-term mechanical ventilation don't need ICU care. In fact, continued placement in the ICU may increase infection risks and interfere with recovery.

Successful weaning is most likely when:

- the patient is highly motivated, medically stable, and able to participate in activities of daily living.
- the patient and family fully understand available treatment options.
- the family actively participates in care.
- a multidisciplinary team, including experienced respiratory care professionals, is available to provide support and direction. The last criterion explains why, as a rule, weaning is best undertaken in an acute or intermediate care setting with consistent caregivers.

In this section, we'll follow a ventilator-dependent patient transferred from the ICU to your four-bed medical respiratory care unit for weaning and discuss how you'd prepare for and manage his care.

Getting Organized

Douglas Johnson, 52, was transferred 2 weeks ago from a community hospital to your hospital's medical intensive care unit (MICU). He's made some progress with weaning, is hemodynamically stable, and no longer requires intensive care. He'll be transferred to your unit tomorrow and you'll be his primary respiratory care giver.

Preparing for his arrival, you call the MICU to obtain an advance report and arrange to stop by to introduce yourself to Mr. Johnson and his family before the transfer. To help coordinate care, you decide to organize the data you collect into a daily weaning log for the benefit of other caregivers who'll play important roles in Mr. Johnson's weaning. This log should include Mr.

Johnson's ventilator settings, breathing treatments, weaning mode and settings, time off the ventilator each day, reason for weaning termination, recent lab results, and the results of any procedures and diagnostic tests.

Next, call respiratory therapy to set up the ventilator for Mr. Johnson's arrival. Then, prepare his room. Gather all the equipment you'll need, including:

- suction supplies (in-wall suction for tracheal and possibly gastric suctioning, a manual resuscitation bag, and high-flow oxygen setup). A closed-suctioning system is preferred, but also have single-use suction catheters and sterile water available for emergencies. Have a tonsil suction device at the bedside for oral secretions.
- pulse oximeter
- blood pressure (BP) equipment
- spare tracheostomy tube (same size and type the patient has in place).

History Lessons

During report, you learn the following about Mr. Johnson:

- *Medical history*. Mr. Johnson was admitted for exacerbation of his chronic obstructive pulmonary disease (COPD) complicated by a left lower lobe pneumonia. He required intubation and mechanical ventilator support soon after hospital admission. His medical history includes a 25-year smoking history and COPD, which was diagnosed 5 years ago. Before this hospitalization, he'd been treated with beta-agonists and ipratropium inhalers. He's been receiving daily physical therapy and can transfer with a walker and assistance from bed to chair twice daily.

- *Weaning history*. Despite several attempts at extubation, he hasn't been able to remain extubated longer than 24 hours. Consequently, he received a tracheotomy 8 days ago. His physician has chosen pressure support ventilation (PSV) as the mode of weaning. Mr. Johnson is receiving PSV of +8 cm H_2O pressure and +5 cm H_2O continuous positive airway pressure (CPAP) during his weaning trials. Yesterday, he spent 10 hours and 50 minutes on this mode-his longest period off full support. His 'resting mode' is assist/control. He continues to receive beta-agonists and anticholinergic nebulizer treatments every 4 hours to maintain airway patency.

  Secretions suctioned from his airway have been clear and not excessive. His breath sounds are distant with intermittent expiratory wheezes bilaterally.

  Mr. Johnson's weaning trials have been terminated in the past because of tachypnea (respiratory rates greater than 35), tachycardia (heart rate greater than 120) and oxygen desaturation to the 91%-to-93% range from his range of 96% to 98% on assist/control.

He's receiving lorazepam, a benzodiazepine, 2 mg every 8 hours, p.r.n., for anxiety and agitation.

- *Nutritional status*. Mr. Johnson has lost 10 pounds (4.5 kg) since his initial hospital admission. He's 10% under his ideal body weight, with low serum protein and albumin levels. Since his tracheotomy was performed, he's been on a regular diet. A calorie count has revealed that his caloric intake is below his estimated needs. After a swallowing evaluation, the speech therapist believes that Mr. Johnson is at high risk for aspiration because of physical deconditioning and a weak cough, so he's receiving a percutaneous endoscopically placed gastrostomy (PEG) feeding tube today. He'll start enteral feedings in the near future.
- *Lab values*. Mr. Johnson has needed potassium and magnesium supplementation throughout his hospitalization.
- *Emotional support*. He has a very supportive wife who visits after work each day. He has many close friends and an accommodating boss in his position as a county supervisor. When you visit Mr. Johnson in the MICU, he's friendly and can communicate with an electronic larynx and letter board.

You now have a clear idea of Mr. Johnson's needs. As you return to your unit, you think about how you can meet them.

Team Effort

Weaning requires a collaborative multidisciplinary approach. Usually the physician (often a pulmonologist) leads the group and decides on the ventilator mode and weaning protocol. (For more details, see *Understanding Weaning Modes*.)

Understanding Weaning Modes

One of several ventilator modes may be used during weaning. So far, research hasn't established which is best.

Assist/control (A/C). A/C may be used as the resting mode for patients undergoing weaning trials. This mode provides full ventilatory support by delivering a preset volume and rate; if the patient initiates a breath, the ventilator delivers the preset volume.

Synchronized intermittent mandatory ventilation (SIMV). This mode delivers a preset tidal volume and number of breaths per minute. Between ventilator-delivered breaths, the patient can breathe spontaneously with no assistance from the ventilator on those extra breaths. As the patient's ability to breathe spontaneously increases, the preset number of ventilator breaths is decreased and the patient does more of the work of breathing.

Pressure support ventilation (PSV). This weaning mode augments the patient's efforts during inspiration by generating a preset amount of pressure in the ventilatory circuit, so the patient

receives a larger tidal volume with less work. The patient determines the timing of breaths, tidal volume received, and rate of breathing. Pressure support levels can be systematically reduced as the patient assumes more of the work of breathing.

Continuous positive airway pressure (CPAP). This mode allows the patient to breathe spontaneously, while applying positive pressure throughout the respiratory cycle to keep the alveoli open and promote oxygenation. Providing CPAP through the ventilator circuit during spontaneous breathing also offers the advantage of an alarm system and may reduce patient anxiety during early trials if the patient has been taught that the machine is keeping track of his breathing. However, CPAP may increase resistance for spontaneous breathing, so it's often used in conjunction with PSV.

T-piece/tracheostomy mask. Weaning trials using a T-piece are normally conducted with the patient disconnected from the ventilator, receiving humidified oxygen only, and performing all work of breathing. Patients who don't have to overcome the resistance of the ventilator circuit may find this mode more comfortable-or they may become anxious as they breathe with no support from the ventilator. During T-piece trials, provide extra encouragement and monitoring.

Other important team members include:

- *the respiratory therapist*, who helps assess whether your patient is ready for a weaning trial, monitors his response to the trial, and helps tailor the weaning protocol to his needs
- *a speech therapist*, who helps the patient communicate using special devices, assesses his ability to swallow, and directs retraining as he resumes eating
- *occupational and physical therapists*, who help restore mobility and increase participation in activities of daily living
- *the dietitian*, who makes sure the patient gets enough calories to meet the high metabolic demands of weaning
- *the patient and his family*, whose support and input are essential during weaning. Include the patient and family in the plan of care daily to help create the support system he'll need during this difficult process.

As the primary respiratory care giver, you coordinate all activities. If possible, schedule at least weekly team meetings to help coordinate care activities and modify goals. Keep team members informed about the patient's progress orally and in writing; for example, through the daily weaning logbook.

Designating a primary respiratory care giver in these kind of situations promotes consistency. As 'head coach,' you can facilitate communication among the staff and help maintain a consistent schedule. Consistent communication with the patient builds and maintains rapport. Praise the patient for a job well done when appropriate and help him deal with setbacks.

He'll be less anxious if he trusts his caregivers, so check in at reasonable intervals, especially during weaning trials; make sure his call light is easily accessible; and answer the light as soon as possible.

You can also minimize his anxiety by establishing a consistent daily routine that's workable for the staff, acceptable to Mr. Johnson, and flexible enough to work during weaning trials that will change in length. Post the daily schedule in his room as a reminder for all.

Timing is important during weaning trials. Allow Mr. Johnson periods on full ventilator support to rest his respiratory muscles. Facilitate adequate sleep, and start weaning trials early in the morning after he's had a good rest.

Mr. Johnson says he prefers to sit in a chair during weaning trials-but he could also lie in a high-Fowler position in bed. Offer him a fan to provide a continuous stream of cool air during weaning trials.

Work with the physical and occupational therapy staff to schedule their time with Mr. Johnson when it's best for him and not merely convenient for them. Work Mr. Johnson's personal hygiene care in at a time other than during weaning trials.

Finally, build in some free time to spend with his family, read, watch television, and take part in other relaxing activities. He can also use these distractions during trials to take his mind off the work of breathing.

Suction the airway only when needed. Routine (every 2 hours) suctioning is no longer recommended, nor is instilling saline down the tracheal tube before suctioning. Frequently assess breath sounds, breathing pattern, oxygen saturation, and ability to mobilize secretions to determine the need for suctioning. Work with Mr. Johnson on improving his ability to clear his airway by coughing.

Getting Started

Tuesday morning, Mr. Johnson arrives in your unit at 9 a.m. and is ready to begin his weaning trial at 11 a.m. Physical assessment reveals that his vital signs are within normal limits while he's on ventilatory support. You note that he has scattered expiratory wheezes and you ask the respiratory therapist to give Mr. Johnson a p.r.n. albuterol treatment before he starts his weaning trial. You review his complete blood cell count and note that his hemoglobin and hematocrit values are normal.

Mr. Johnson starts his weaning trial on PSV +8 cm H_2O, CPAP +5 cm H_2O, and 40% oxygen. One hour into the trial, you observe that his spontaneous tidal volume is less than 300 ml and that his respiratory rate is over 30. His minute volume is approaching 10 liters/minute.

You assess his breath sounds and find that the wheezing has resolved, but his breath sounds are especially diminished at the bases. Mr. Johnson appears anxious and says he feels very short of breath. You ask him to rate his dyspnea and he puts it at an 8 on a scale of 0 (not short of breath) to 10 (worst shortness of breath imaginable). His heart rate is in the 120s and slightly irregular. His other vital signs are within normal limits. Based on the assessment, you and the respiratory therapist decide to stop the weaning trial and put him on assist/control until his respiratory status

stabilizes (see *When Should You Call it Quits* ?). You notify the physician of the weaning failure, and he modifies the weaning plan.

When Should You Call it Quits?

Discontinue a weaning trial if the patient exhibits any of the following symptoms.

Pulmonary

- Paradoxical abdominal movement
- Intercostal retractions
- Presence of or increased use of accessory muscles
- Tachypnea (respiratory rate >35 or a change of 10)
- Shallow breathing (tidal volume <5 ml/kg)
- Increased dyspnea
- Cyanosis
- Spo_2 less than 90% or 94% (depending on patient baseline)
- pH <7.30
- Pao_2 <60 mm Hg or >20 mm Hg decrease
- $Paco_2$ change of +/- 10 mm Hg

Cardiovascular

- Pulse +/- 20 beats/minute change or >120 beats/minute (sustained for 5 minutes)
- BP +/- 20 mm Hg change from baseline systolic; +/- 10 mm Hg change from baseline diastolic (sustained for 5 minutes)
- Diaphoresis
- New cardiac arrhythmia
- Angina

Central nervous system/psychologic

- Decrease in level of consciousness
- Increasing anxiety
- Apprehension
- Irritability or agitation
- Exhaustion

Managing an 'Athlete'

Was Mr. Johnson ready for weaning? To answer that question, think of your patient as an athlete in training. Effective communication, a consistent training schedule, and support for continued 'training' will allow your patient to achieve his weaning goals. Assess the following factors to determine his fitness for weaning.

Respiratory Status. To meet the increased demands of weaning, the patient needs adequate BP, fluid volume, and hemoglobin to oxygenate the tissues. His hemoglobin level should be 8 grams/dl or higher.

Arterial blood gases (ABGs), valuable for assessment of oxygenation, ventilation, and blood pH, may be indicated under certain circumstances. When a patient experiences a dramatic change in condition or lack of progress for no obvious reason, ABGs may provide insight and help dictate appropriate interventions.

Pulse oximetry, which is now more common than ABGs for monitoring oxygen saturation, has the advantage of being continuous and noninvasive. However, it doesn't provide information about the patient's ventilation; you'll need an ABG analysis to determine the patient's $Paco_2$, Pao_2, and HCO^{-3} . The $Paco_2$ level affects blood pH (normally 7.35 to 7.45).

Remember that attempting to normalize $Paco_2$ to between 35 and 45 mm Hg is inappropriate for a patient like Mr. Johnson. Because of his long-standing pulmonary disease, his baseline $Paco_2$ is probably above 50 mm Hg.

During weaning, assess the patient's respiratory status to determine his ability to continue. Monitor the quality of his respirations (depth, use of accessory muscles, pattern) and have him rate his shortness of breath with the 0-to-10 dyspnea scale. Assess breath sounds every 2 to 4 hours, or more frequently if necessary, and intervene as needed. Finally, closely monitor secretions for changes in amount, color, and consistency.

The following values may help you further evaluate progress. Remember, though, that these numbers are only guides to help monitor patient capability-not strict criteria for success or failure.

- *Vital capacity* (normal, 10 to 15 ml/kg) is the amount of air expired after a maximal inspiration. It helps you assess the patient's ability to take deep breaths.
- *Negative inspiratory force* (normal, -20 to -30 cm H_2O) helps you assess the patient's respiratory muscle strength.
- *Tidal volume* (V_T) per breath is normally 7 to 9 ml/kg.
- *Minute ventilation* (normal, about 6 liters/minute) is equal to respiratory rate multiplied by tidal volume. This value should be less than 10 liters/minute in patients being considered for weaning.
- *Rapid/shallow breathing index* assesses breathing pattern. Calculate it by dividing respiratory rate by tidal volume. Patients with indexes below100 breaths/minute/liter are more successful at weaning.

Cardiovascular Status. Can the patient's cardiovascular system tolerate the demands placed on it by the weaning trials? The patient's heart rate and BP should be within normal ranges, with no frequent or uncontrolled arrhythmias.

If he has a history of heart failure, he should be on an angiotensin-converting enzyme inhibitor, a diuretic, and digoxin. Maximizing cardiac function and controlling circulating blood volume improve his chances for successful weaning.

Monitor daily intake and output and weights every other day to maintain adequate circulating volume and provide enough fluids to help liquefy secretions and aid in expectoration. If the patient is hypertensive, regulate his BP with medication as ordered; if he's hypotensive, give fluid resuscitation as needed. For best results, intervene as needed *before* weaning trials begin.

Monitor the patient's temperature for fever and assess for infection. To avoid nosocomial infections, take appropriate precautions with airway care, intravenous lines (particularly central catheters), urinary catheters, and other tubes and drains.

***Nutritional Status*.** Watch for signs of nutritional deficiencies, including decreased albumin and prealbumin. Regularly consult with the dietitian about meeting the caloric requirements necessary for weaning trials and keeping a positive nitrogen balance to avoid muscle loss. Maintaining muscle mass, particularly of the diaphragm, is vital to weaning.

Provide high-calorie supplements and have the family bring in your patient's favorite foods when he can take food orally. But discourage a diet that's high in carbohydrates because this can increase carbon dioxide production and tax the respiratory system.

A patient who can't eat needs enteral feedings and should be monitored closely for tolerance to these feedings.

Monitor all patients for risk of aspiration.

Neurologic Status. Closely monitor the patient for changes in level of consciousness, orientation, and strength. Avoid oversedation, but use anxiolytic agents and sleeping aids as necessary to keep him calm and allow him to rest.

Electrolyte Status. Additionally, monitor serum electrolytes and maintain serum potassium between 3.5 and 5 mEq/liter, calcium between 8.9 and 10.1 mg/dl, phosphorus between 2.5 and 4.5 mg/dl, and magnesium between 1.7 and 2.1 mg/dl.

Helping Mr. Johnson Through a Weaning Trial

Mr. Johnson's caregivers hold a conference the next morning. The physical therapist believes that he'll make more progress in strengthening, endurance, and gaining weight if his physical therapy is performed while he's on ventilatory support. The dietitian places Mr. Johnson on enteral feedings via the PEG tube until he can swallow without aspirating.

Mr. Johnson will start out on +10 cm H_2O PSV and +5 cm H_2O CPAP today, and the pressure support weaning protocol will be followed. To boost his morale, the team also plans to get him outdoors in a few days while his wife visits.

Mr. Johnson and his wife don't attend the meeting, but you update them and incorporate their preferences and suggestions into the plan.

Mr. Johnson makes slow but steady progress in weaning following the new protocol. After a week, he's down to +6 cm H_2O PSV and spending up to 14 hours off full ventilatory support. He's gained 3 pounds (1.36 kg) and hasn't experienced any electrolyte abnormalities since he began receiving enteral feedings. He occasionally gets anxious during his weaning trials but requires less-frequent p.r.n. doses of lorazepam.

By the third week in your unit, Mr. Johnson is completely weaned from mechanical ventilation. He's gained 9 pounds (4.1 kg) and tolerates oral feeding without risking aspiration, so his PEG tube can be removed. He can now walk a short distance down the hall with a walker.

The weaning team continues to have weekly multidisciplinary conferences. After discussion with Mr. Johnson and his wife, the team aims to transfer him to a rehabilitation facility closer to home once he can spend 72 continuous hours on a tracheostomy mask. He'll stay at the rehabilitation facility for about 2 weeks, where his tracheostomy can be discontinued.

Getting the Job Done

Collaboration, consistency, vigilant assessment, and prompt intervention all helped Mr. Johnson undergo weaning from long-term mechanical ventilation more efficiently. The caring attitude and team approach to his management provided the support necessary to meet the demands of weaning and facilitate his independence from the ventilator.

Team Approach to Weaning

Liberating Patients From Mechanical Ventilation: A Team Approach

Elizabeth A. Henneman

In the past decade, critical care clinicians have witnessed an increased interest in improving outcomes for patients being weaned from mechanical ventilation. The high cost of caring for patients who are receiving mechanical ventilation has spurred research in numerous areas, including the evaluation of both assessment strategies and methods of weaning. Research suggests that successful weaning outcomes are not related to the use of any single weaning method. Instead, improved outcomes often are due to the coordinated efforts of a skilled, multidisciplinary team. In this section, evidence is presented in support of a team, or collaborative, approach to weaning and discuss strategies for promoting such a team approach to weaning in the clinical setting.

The Weaning Process

Weaning from mechanical ventilation is the process of gradually liberating patients from artificial ventilatory support and allowing the resumption of spontaneous breathing. The

American Association of Critical-Care Nurses' (AACN's) Third National Study Group developed a conceptual model of weaning that presents an organized approach for evaluating the weaning process. The framework the group proposed for weaning included 3 phases: the pre-weaning phase, the weaning process, and the outcome phase. The AACN model is used here to examine structures and processes applicable to weaning teams that could improve weaning outcomes.

Phase 1: Pre-weaning

The first phase of weaning from mechanical ventilation is the pre-weaning or assessment phase. During this phase, clinicians must determine if the patient is capable of attempting spontaneous breathing. Assessment of readiness to wean includes an evaluation of both respiratory and non-respiratory factors. Respiratory factors include oxygenation, ventilation, and lung mechanics. Non-respiratory factors include neurological status, hemodynamics, hemoglobin levels, fluid and electrolyte levels, and nutrition. Other non-respiratory factors that may play a role in the patient's ability to be weaned include psychosocial factors, sleep, and medications.

The assessment of these numerous, often-interrelated variables falls under the purview of many disciplines, including nursing, medicine, respiratory therapy, pharmacy services, social work, and dietary services. The more complicated a patient's situation, the more crucial it is to have the expertise of the various team members. For example, a patient with an impaired nutritional status would benefit from the involvement of a dietitian who is knowledgeable about the implications of nutritional deficits in the weaning process.

Several important studies indicate the usefulness of a team approach to the assessment of readiness for weaning. One institution used a team of respiratory therapists, nurses, and physicians to determine which patients were capable of spontaneous breathing. Patients in that study were randomly assigned to either an experimental group or a control group. Patients in the experimental group received a multidisciplinary assessment of their weaning ability and a 2-hour trial of spontaneous breathing. Patients in the control group received "usual care," which consisted of physician-directed weaning. This multidisciplinary protocol resulted in a significant decrease in duration of mechanical ventilation (4.5 days vs. 6 days, $P = .004$) and cost of intensive care ($15 740 vs. $20 890, $P = .03$).

One research study focused on weaning readiness. They compared a multidisciplinary protocol-directed weaning process with a more traditional physician-directed plan. The protocol included both assessment criteria and a plan for implementing the weaning process. Patients assigned to the multidisciplinary protocol-directed group received fewer days of mechanical ventilation than did patients in the control group (35 hours vs. 44 hours, $P = .02$).

The aforementioned studies used protocols, combined with clinical expertise, to assess patients' readiness for weaning. The findings lend support to the benefits of having a multidisciplinary approach to weaning assessment that takes advantage of the unique skills and expertise of bedside clinicians.

Phase 2: Weaning Process

The weaning process begins once a patient has been deemed ready to wean and strategies to

reduce or discontinue support are made. The weaning process ends when the decision to stop weaning is made. The weaning process is perhaps the most complex of the 3 phases of weaning. During the weaning process, a number of complex decisions are made, including the method of weaning to be used, criteria for stopping a weaning trial, and therapies to facilitate weaning.

Most of the research on weaning has focused on weaning methods. Until quite recently, most of the biases for one method over another were based on individual clinicians' experience and preference, as opposed to data from well-conducted studies. In the mid-1990s, 2 groups of researchers attempted to determine the optimal weaning method. These researchers compared the 3 major weaning methods: use of a T-piece, intermittent mandatory ventilation, and pressure-support ventilation. One group reported that weaning was fastest with the T-piece method; the other group found that pressure-support weaning was more efficient.

The lack of evidence of the superiority of any one method of weaning over another has left experts to question whether aspects of the weaning process other than the method may be most important in influencing the outcome of weaning. This line of questioning led to several research studies and outcome management programs to evaluate the effect of a multidisciplinary team approach on weaning outcomes.

Another study examined the effect of a formal interdisciplinary team on patients receiving mechanical ventilation in the intensive care unit (ICU). These researchers used historical controls to compare outcomes before and after establishment of a ventilator management team. Their team included a physician, a charge nurse, and a respiratory therapist. The group managed and supervised patients via daily rounds and other communications with the staff. Goals of the team included (1) communicating the weaning plan to all involved staff members, (2) beginning weaning trials by 8 am, (3) placing more reliance on clinical findings than on arterial blood gas measurements, (4) weaning/extubating on weekends, and (5) encouraging unit-wide coordination.

The ventilator management team was effective in decreasing duration of mechanical ventilation by 3.9 days (8.4 days vs. 12.3 days, $P = .004$). They also estimated a cost savings of $1303 per episode of mechanical ventilation.

Special ventilator teams consist of a multidisciplinary team of experts specifically created to support bedside clinicians. Another approach to a weaning team would be to use existing personnel and give them new structures to guide the weaning process by using new tools or techniques such as protocols and communication tools (e.g., communication boards/weaning flow sheets). The latter approach of using existing personnel may be more feasible than the former, given the cost-conscious healthcare era we live in.

Phase 3: Weaning Outcomes

The outcome phase is the period when weaning stops, either because the patient has been completely liberated from mechanical ventilation or because no further progress is being made. The outcomes of weaning described by the AACN study group include complete weaning from

the ventilator, partial weaning, and terminal weaning.

The determination that a patient is completely weaned from the ventilator is generally straightforward and is based on the patient's ability to meet certain criteria, such as maintaining a normal respiratory rate or tidal volume while breathing spontaneously. Less straightforward is the circumstance when it is determined that a patient can be only partially weaned or when a patient, the patient's family, and the team have decided that terminal weaning (discontinuation of mechanical ventilation even though death will result) is appropriate. The need for the expertise of a multidisciplinary team in planning care for a patient who will require long-term ventilator support or will undergo terminal weaning cannot be overstated.

One example of an effective interdisciplinary team is the “comprehensive supportive care team.” This group consists of an advanced practice nurse, an attending physician, and other resource persons such as social workers and providers of pastoral care. The supportive care team provides a number of services to patients at the end of life, including terminal weaning when appropriate. Use of this team approach can decrease length of stay in the ICU and hospital and increase satisfaction for patients, patients' families, and staff members.

Promoting a Team Approach

The concept that a team or collaborative approach to care is superior to other approaches and should be incorporated into the operations of an ICU has long been espoused by critical care and other healthcare organizations. Unfortunately, despite much rhetoric, collaboration still remains the exception rather than the rule in many critical care units. Promoting a team approach to weaning takes more than an organizational philosophy or a mission statement that incorporates the verbiage associated with teams. Rather, it takes a thoughtful, purposeful implementation of structures that force a change in practice from a traditional, hierarchical approach to a multidisciplinary, collaborative endeavor.

Organizational theorist Peter Senge proposed the concept that teamwork does not just happen but is the product of organizational structures. Senge suggested that in order to change outcomes, the behaviors of persons in an organization must be altered. Senge proposed that the way to change these behaviors is to implement structures that alter the way people work. This model can be easily applied to the weaning process and provides a strategy for creating a successful team approach to weaning.

Structures to Facilitate A Team Approach to Weaning

A number of different structures can be implemented in an ICU setting to facilitate a team approach to weaning. The type and number of strategies used in any given unit ultimately depends on a number of factors, including the population of patients and the amount of available resources.

Weaning Protocols

The efficacy of successful weaning protocols is clearly multifaceted but stems in part from allowing the members of various disciplines to use their expertise to make decisions about patients' care. Multidisciplinary protocols and standards may foster teamwork by providing

guidelines for expected processes and outcomes of care.

Weaning protocols generally fall into 2 categories: those for assessing readiness to wean and those that guide the weaning process itself. The key to a successful protocol is that a multidisciplinary team participates in its development. This point is critical. Even if only 2 or 3 members of a team carry out the protocol, all parties with an expertise or stake in the patients' outcomes should be involved in developing the protocol. For example, a protocol for assessing readiness for weaning in a straightforward postoperative patient is typically carried out by a respiratory therapist and the bedside nurse. Nonetheless, a variety of other persons have a stake in this process and must be included in the development of this protocol, including pulmonologists, anesthesiologists, intensivists, and surgeons.

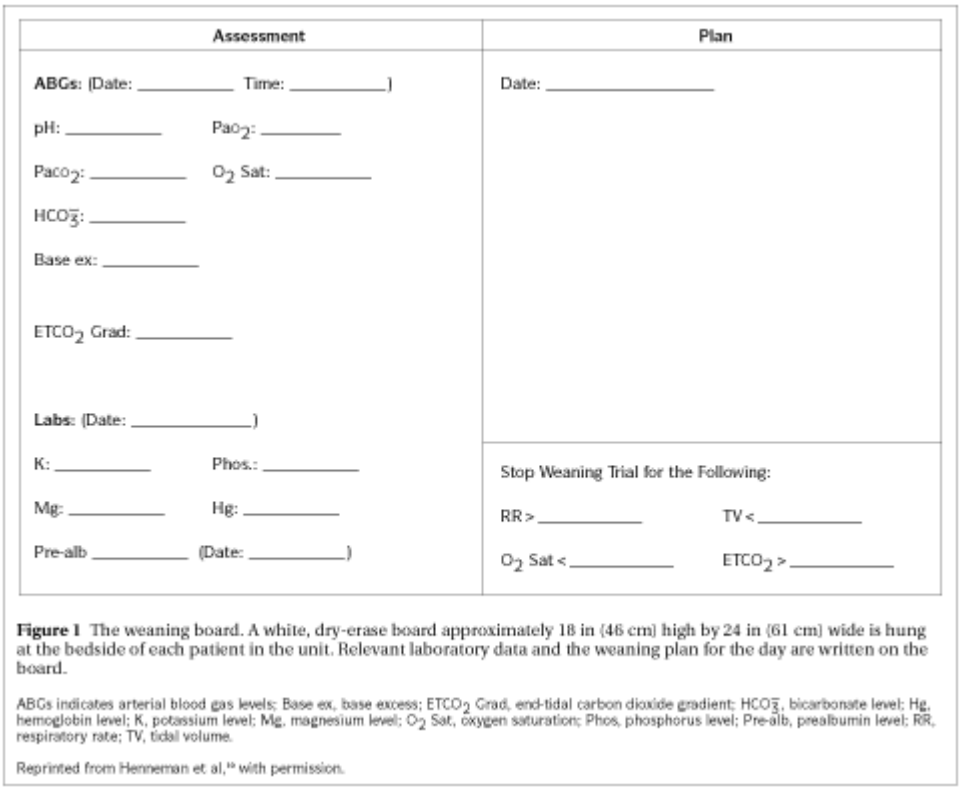
The implementation of a weaning protocol, regardless of how straightforward, can be fraught with obstacles. A wise approach is to start with a fairly basic protocol, such as one to assess readiness for weaning uncomplicated postoperative patients, and then move to a more involved protocol as the team experiences success. As they work together and experience positive outcomes, members of the team will gain increasing respect and trust for one another, a situation that in turn will foster continued multidisciplinary endeavors.

Weaning Flow Sheets/Communication Boards

Units that care for patients receiving mechanical ventilation for longer than 3 days (i.e., long-term)[1] need access to structures that facilitate communication and documentation of the entire weaning event. These structures include methods of documenting readiness to wean, the weaning plan, and weaning progress. Communication about the weaning process is not the straightforward task it may appear to be. In fact, evaluations[7,10] of innovative weaning strategies typically include strategies to improve communication about weaning.

It is difficult to track a process such as weaning that takes place over several days or weeks on a 24-hour flow sheet. Plans of care involving weaning are often complex and may change frequently, depending on how the patient progresses. This type of fluidity in a plan is also challenging because of the documentation systems typically used in ICUs. The reality is that documentation in the ICU is classically short-term and discipline specific. Both of these factors hinder a process that occurs over an extended period and demands a multidisciplinary approach.

Structures such as communication boards and weaning flow sheets allow multiple disciplines to have "at-a-glance" access to information about weaning. The more visible the information, the easier it is for staff members, patients, and patients' families to remain up to date on the weaning progress. Weaning boards (Figure 1) are simple, white, dry-erase boards that are kept at each patient's bedside. The information written on the board can be adapted to a particular unit's standards and typically includes information about readiness for weaning (arterial blood gas levels, lung mechanics, other relevant laboratory values) and the weaning plan. Weaning flow sheets (Figure 2) are like typical ICU flow sheets, except that they focus on variables related to weaning and allow for documentation over several days. The weaning flow sheet should be accessible to all members of the team, including the patient and the patient's family.



Assessment	Plan
ABGs: (Date: _____ Time: _____) pH: _____ Pao_2: _____ $Paco_2$: _____ O_2 Sat: _____ HCO_3^-: _____ Base ex: _____ $ETCO_2$ Grad: _____ **Labs:** (Date: _____) K: _____ Phos.: _____ Mg: _____ Hg: _____ Pre-alb _____ (Date: _____)	Date: _____ Stop Weaning Trial for the Following: RR > _____ TV < _____ O_2 Sat < _____ $ETCO_2$ > _____

Figure 1 The weaning board. A white, dry-erase board approximately 18 in (46 cm) high by 24 in (61 cm) wide is hung at the bedside of each patient in the unit. Relevant laboratory data and the weaning plan for the day are written on the board.

ABGs indicates arterial blood gas levels; Base ex, base excess; $ETCO_2$ Grad, end-tidal carbon dioxide gradient; HCO_3^-, bicarbonate level; Hg, hemoglobin level; K, potassium level; Mg, magnesium level; O_2 Sat, oxygen saturation; Phos, phosphorus level; Pre-alb, prealbumin level; RR, respiratory rate; TV, tidal volume.

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Patient name: ______________________________

	Date	Date	Date	Date	Date	Date	Date	Date	
Trial START time									**Trial START time**
Trial STOP time									**Trial STOP time**
TOTAL trial duration									**TOTAL trial duration**
REASON Trial Stopped	Tachypnea Tachycardia Low T.V. Agitation Short of breath Planned stop Other:	Tachypnea Tachycardia Low T.V. Agitation Short of breath Planned stop Other:	Tachypnea Tachycardia Low T.V. Agitation Short of breath Planned stop Other:	Tachypnea Tachycardia Low T.V. Agitation Short of breath Planned stop Other:	Tachypnea Tachycardia Low T.V. Agitation Short of breath Planned stop Other:	Tachypnea Tachycardia Low T.V. Agitation Short of breath Planned stop Other:	Tachypnea Tachycardia Low T.V. Agitation Short of breath Planned stop Other:	Tachypnea Tachycardia Low T.V. Agitation Short of breath Planned stop Other:	**REASON Trial Stopped**
Type wean (PS,IMV,T-piece)									**Type Wean (PS, IMV, T-piece)**
PS level									**PS level**
IMV rate									**IMV rate**
Set TV									**Set TV**
Pt. Response	Start / End	Start / End	Start / End	Start / End	Start / End	Start / End	Start / End	Start / End	**Pt. Response**
HR 150 125 100 75 50 0									**150** **HR** **125** **100** **75** **50** **0**
RR 50 40 30 20 10 0									**50** **RR** **40** **30** **20** **10** **0**
SPONT. TV 500 400 300 200 100 0									**500** **SPONT. TV** **400** **300** **200** **100** **0**
Comments (Pt. Position, etc.)									**Comments (Pt. Position, etc.)**

Figure 2 The weaning flow sheet, a multidisciplinary form used to document a patient's progress with weaning. It is approximately 20 x 30 in (51 x 76 cm) and is hung on the wall at the patient's bedside.

HR indicates heart rate; IMV, intermittent mandatory ventilation; Pt., patient; PS, pressure support; RR, respiratory rate; Spont. TV, spontaneous tidal volume.

Reprinted from Henneman et al,[10] with permission.

Multidisciplinary Rounds

The discussion of a patient and plan of care typically occurs during patient care rounds. These rounds may be held at the patient's bedside or in another location such as a conference room. Depending on the unit, multidisciplinary rounds may be held daily or even more often, or they may be held only once a week. In any case, these forums offer the opportunity for a multidisciplinary approach to weaning. The key to successful rounds are not necessarily where or how often they are held, but rather that representatives from different disciplines are involved and that the plans decided upon at these meetings are communicated effectively to the rest of the team.

Ventilator Management and Quality Improvement Teams

Research[7] suggests that a designated ventilator management team is highly effective in improving outcomes for patients being weaned from mechanical ventilation. The key to a successful ventilator management team includes having the right mix of skilled individuals who can effectively communicate a plan to the clinicians at the bedside. The most economically feasible approach to a ventilator team is to use existing ICU personnel already in leadership positions, such as intensivists, respiratory specialists, and advanced practice nurses or charge nurses, as team members.

The use of an "outcomes management" approach by a multidisciplinary team also may be effective in improving weaning outcomes, especially for long-term ventilator patients. This model uses a team of highly motivated, committed individuals who specifically care for and track variables related to weaning. These team members may or may not provide care directly to patients being weaned but are involved in evaluating patients' care and promoting strategies for improvement.

Finding a Team Approach That Works

The suggestions made here are applicable to a wide variety of settings. Nonetheless, both the population of patients and the availability of resources should be examined when deciding what would work best in a particular setting. Regardless of the strategy, any new approach to care should be evaluated for its effectiveness. For example, the multidisciplinary team may want to compare the number of ventilator days per patient before and after the implementation of the new team approach. Or staff members, physicians, and patients could be surveyed to assess their satisfaction with the weaning process.

Some structures such as weaning protocols may be more appropriate for patients with a more predictable course, such as postoperative patients receiving mechanical ventilation for short periods. Structures such as weaning boards and flow sheets may not be as practical for patients treated with mechanical ventilation in the very short-term and might be reserved for more complicated weaning scenarios. The goal is to work together in a team mode, whether with protocols, standards, or more complex plans determined during patient-care rounds.

Conclusion

The process of weaning patients from mechanical ventilation can be complex. It seems obvious that the weaning process would benefit from an approach that uses the combined skills and expertise of a multidisciplinary approach. Unfortunately, many of the structures and processes used in ICUs today continue to promote hierarchical and discipline-specific decision-making.

An increasing body of research suggests that the key to successful weaning lies not in the use of a particular method but rather in the use of a coordinated approach by a skilled multidisciplinary team. Standards and protocols serve as important structures for facilitating a team approach. Other structures that have been successful include ventilator teams, collaborative care planning using weaning boards and flow sheets, and outcomes management teams.

MECHANICAL VENTILATION-PAST, PRESENT AND FUTURE

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'The lungs are the center of the universe and the seat of the soul'
-Unknown philosopher

Mechanical ventilation is the mainstay of resuscitation, intensive care medicine, and anesthesia. Mechanical ventilation as with many things in medicine was born out of necessity. Historical discovery often precedes clinical adaptation of a technique not by days but by decades. Sometimes because of human prejudice, lack of insight and unadaptability it takes centuries for the new technique to be put into practice. However history not only informs us where we have been but often predicts the place to which we will return.

PAST:
Stories of brilliant insight, clinical application, public ridicule, and abandonment only to have the principle "rediscovered" and reapplied are frustratingly frequent in the history of mechanical ventilation.

Early History of Ancient times

Egyptian, Chinese and Greek writings dating back to BC described Theories of Respiration and Air. In Old testament (800BC) there is a mention of : Prophet Elisha induced pressure breathing from his mouth into the mouth of a child who was dying–Hippocrates (460-375BC) wrote in his book –'Treatise on Air' "One should introduce a cannula into the trachea along the jaw bone so that air can be drawn into the lungs". This is the first written instance of Endo-tracheal intubation.

Modern History

Paracelsus (1493-1541) used 'Fire Bellows' connected to a tube inserted into patient's mouth as a device for assisted ventilation. This was the first study (1550) which credited him of first form of Mechanical Ventilation. John Fathergill in 1744 reported a successful case of 'mouth to mouth' resuscitation. Humane society (Dutch Society for Rescue of Drowned persons) was established in 1767 which advocated the principles of treatment for victims of drowning which are even true today. John Hunter developed double bellows for resuscitation in the year 1775 - one for blowing air in and the other for drawing bad air out. Drager designed artificial breathing device"Drager Pulmoter" in 1911 that was used by fire and police units.

Negative Pressure Ventilators

From mid 1800 to 1900 incredible number of devices were invented that applied negative pressure, around the body or thoracic cavity – these devices became known as negative pressure ventilators or Iron Lungs. Two successful designs became popular; in one, body patient was enclosed in an iron box or cylinder and the patient's head protruded out on one end. Second design was a box or shell that fitted over the thoracic area only (Chest Cuirass). Patients with chronic paralytic disorders were successfully ventilated on this cuirass ventilators at home for 25-30 years.

Scandinavian Polio Epidemic - 1952

Between mid july to early december of 1952, in Copenhagen, 2722 patients with poliomyelitis were treated in the Community Disease Hospital of which 315 patients required ventilatory support. Many principles of IPPV were defined during that time – viz
use of cuffed tube, periodic sigh breaths, weaning by reduction of assisted breaths. Towards the end of epidemic a few positive pressure ventilators were invented viz. the Engstrom, Lundi and the Bang which became popularly known as mechanical students.

Era of Respiratory Intensive Care

After polio epidemics, the 1960's became an era of respiratory intensive care. Positive pressure ventilation with use of an artificial airway replaced the bulky and cumbersome negative pressure technology of respiratory support. Two types of ventilators and
two modes of mechanical ventilation evolved during this period. First type of ventilator was pressure cycled –PCV; two ventilators were commonly used for PCV in 1960's and 1970's viz. Bird Mark 7 and Bennet PR2. Second type of ventilator that got evolved from historical perspective is the volume cycled ventilator – VCV. The first fluidic ventilator utilizing moving streams of liquid or gas for sensing, logic, amplification and controls was designed for the US army in 1964 by Barila and the first commercial versatile fluidic ventilator "Hamilton standard PAD" appeared in 1970. The term 'weaning' was used to explain various techniques to test the quality of patient's spontaneous ventilation before extubation.

PRESENT

A mechanical change of substantial importance in the late 1960's and early 1970's that shaped the present era was the introduction of Positive End Expiratory Pressure (PEEP). Two modes of ventilation viz. Assisted Ventilation (AV) and Controlled

Mechanical Ventilation (CMV) came together in a single piece of equipment and modern era of multiple choice respiratory support was born. Introduction of IMV, permitted spontaneous respiration in the midst of substantial respiratory failure which paved the way for a superb means of weaning ie SIMV. PSV proved to be an addition to IMV that facilitated spontaneously breathing patients. Pressure support does for the respiratory muscles what vasodilators do to ventricular muscles unloading. Another rather important justification for newer modes of ventilation was the persistently high mortality rate of ARDS despite application of PEEP which lead to the redefining of PCV and description of IRV.

A quick glance through any modern ICU will reveal sophisticated electronic ventilators that have multiple options of various newer modes of ventilation. It is to be noted that newer modes of ventilation are integrated in many of the present day machines, well before any evidence convincingly supports the value of these new techniques. When any new mechanical ventilation technology and strategies are introduced, a question we need to ask ourselves always is - whether these are the solutions for problems or whether these are simply solutions in search of problems.

FUTURE OF MECHANICAL VENTILATION

Future mechanical ventilation strategies can certainly be predicted based on some of the following recently published literature.

1) **Is the injured lung to be rested or recruited?**
Indeed two RCTs on the use of PEEP suggested that its use is associated with increase in mortality. The policy that recruits as well as rests the lungs should be followed by adapting both the 'Open lung concept' as well as 'Protective lung ventilation' allowing 'Permissive Hypercapnia.'

2) **Non-invasive ventilation: Current status**
The use of NIV is increasing for patients with acute and chronic respiratory failure, because of its convenience, lower cost and minimal complications. NIV has been shown to decrease endotracheal intubation rates, complication rates and median hospital length of stay. But unfortunately NIV has not found a place in all intensive care units. It is expected that this modality will be used more frequently in the near future.

3) **High-frequency Oscillatory Ventilation:**
High-frequency oscillatory ventilation seems theoretically ideal for the treatment of patients with ARDS who are no longer responding to conventional mechanical ventilation., allowing adequate oxygenation and ventilation to be maintained without causing further damage to the already injured lung. Future research may define the role of HFOV as a more routine strategy for preventing VALI in these patients.

4) **Gas Exchange in the Ventilated Patient:**
Several studies to evaluate different treatment regimen to improve oxygenation and outcome in acute respiratory failure have been undertaken.

a. Changes in body position (mainly prone positioning) can significantly improve gas exchange in patients with ARDS and acute lung failure, with few complications related to the maneuver; however, no survival advantage has yet been detected.

b. New data support the hypothesis that maintenance of even small amount of spontaneous breathing during mechanical ventilation with airway pressure release ventilation -APRV or biphasic positive airway pressure-BiPAP can improve gas exchange, whereas other unconventional ventilatory modes have not yet proved advantageous.

5) **Lung Recruitment Maneuvers:**
Recruitment maneuvers that used continuous positive airway pressure levels of 35-40 cm H2O for 40 secs, with PEEP set at 2 cm H2O above the Pflex (the lowest inflection point on the pressure-volume curve), and tidal volume <6 mLkg-1 were associated with a 28-day intensive care unit survival rate of 62% in contrast to the survival rate of only 29% with conventional ventilation.

High airway pressures can open collapsed ARDS lungs and partially open edematous ARDS lungs. High PEEP levels and low tidal volume
ventilation decrease bronchoalveolar and plasma inflammatory mediators and improve survival compared with low PEEP/high tidal volume ventilation.

6) **Ventilatory Adjuncts:**
Automatic tube compensation (ATC), the regulation of airway pressure by estimation of tracheal pressure appears to be an ideal approach to unloading the resistive effort imposed by the endotracheal tube. Recently performed RCTs with HFO, partial liquid
ventilation (PLV), and prone positioning have documented, no use of PLV and marginal benefit with both HFO and prone positioning.

7) **Innovative Practices of Ventilatory Support with Pediatric Patients:**
The recognition that alveolar over distension rather than peak inspiratory airway pressure is the primary determinant of lung injury has shifted the understanding of the pathogenesis of ventilator-induced side effects. A strategy combining recruitment maneuvers, viz. Low TV and higher PEEP, volume control ventilation with high PEEP levels, have been proposed as the preferable protective ventilatory modes. PRVC and HFOV have taken on an important role as protective lung strategies. Spontaneous breathing with PSV is widely used to maintain or reactivate spontaneous breathing and to avoid haemodynamic variations.

Volume support ventilation reduces the need for manual adaptation to maintain stable TV and MV and can be useful in weaning. Surfactant and NO have been proposed in specific lung pathologies to facilitate ventilation and gas exchange and to reduce inspired oxygen concentration. Investigation of lung ventilation using a liquid instead of gas has opened new vistas on several lung pathologies with high mortality rates. The
conviction emerges that the best ventilatory treatment may be obtained by applying a combination of types of ventilation and supportive treatments as outlined above. The methods proposed require confirmation through large RCTs to define the optimal method(s) to treat individual pathologies in the various pediatric age groups.

8) **Nasal IPPV versus nasal CPAP for apnea of prematurity.**
Apnea of prematurity is almost universal in infants born before 34 weeks gestation. Previous RCTs and systematic reviews have found methylxanthines to be effective in its prevention. However, recent concerns about their potential long term side effects on
the neurodevelopment of low birth weight infants have led to an increased interest in alternate methods of treatment. Nasal CPAP reduces the incidence of obstructive or mixed apnea. However, apneic infants managed with NCPAP, with or without methylxanthines, sometimes require endotracheal intubation with its attendant morbidity and cost. Nasal IPPV is a simple, effective mode of respiratory support for older children and adults. However case reports of gastrointestinal perforations on its application have limited its widespread use. Future trials with sufficient power should assess the efficacy (reduction in failure of therapy) and safety (GI complications) of NIPPV. The recent ability to synchronise NIPPV with an infant's spontaneous respirations is a promising development requiring further assessment.

9) **Computer control of mechanical ventilation.**
Computer control of mechanical ventilators include the operator-ventilator interface and the ventilator-patient interface. New ventilation modes represent the evolution of engineering control schemes. The various ventilation control strategies behind the modes
have an underlying organization, and understanding that organization improves the clinician's appreciation of the capabilities of various ventilation modes and gives an idea of what we can and should expect for the future.

FINAL WORD:
Mechanical ventilation with IPPV is in use for the past 40 years, initially to treat hypoventilation of normal lungs and subsequently to treat pulmonary complications after surgery, pneumonia, asthma, ARDS etc. During the past 25 years the main thrust
of research has been to develop techniques that minimize the harmful effects of mechanical ventilation. Despite description of many newer modes, there has been acute lack of valid evidence to state that a particular ventilatory mode improves patient survival, for the obvious reasons of ethical constraints to conduct a proper trial and also small number of patients available to conduct a study.

Prof. P. F. Kotur
Editor.

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American Thoracic Society, Patient Information (http://thoracic.org/sections/clinical-information/critical-care/patient-information/icu-devices-and-procedures/mechanical-ventilator.html)

Society of Critical Care Medicine, Patient and Family Website (http://www.icu-usa.com/tour/procedures/vent.htm)

CCM Tutorials.com (with technical details) (http://www.ccmtutorials.com/rs/mv/index.htm)

Glossary

Air Flow Air will flow from an area of higher pressure to one of lower pressure; during inspiration, the pressure in the alveoli must be less than the pressure at the mouth for air to flow in, and during expiration, the reverse is true. Air flow may be laminar, turbulent or transitional, depending on the velocity of flow and on the diameter and configuration of the tube.

Airway The anatomical structures through which air passes on its way to or from the alveoli; the nasopharynx and oropharynx, the larynx, the trachea, bronchi, and bronchioles.

Airway Resistance Driving pressure divided by flow (P/V); or, the opposition to motion caused by the forces of friction, which is a function of flow rate, airway caliber, nature of gas breathed, and type of flow (laminar vs. turbulent).

Alveolar Pressure The pressure within the alveoli, conventionally given in cm H20, with reference to an atmospheric pressure of zero. Thus, a negative alveolar pressure indicates that the pressure is lower than atmospheric; a positive alveolar pressure indicates that the pressure is above atmospheric.

Alveoli The air sacs that act as the primary gas exchange units of the lung.

Atmospheric Pressure Ambient air pressure, averages 760 mm Hg at sea level. In pulmonary calculations, atmospheric pressure is taken as the reference value, 0 cm H20. Pressures higher than atmospheric pressure then are positive; those lower than atmospheric pressure are negative.

Body Plethysmograph A large airtight box used for measuring lung volumes; the subject sits in the box where pressure and volume changes are measured by Boyle's Law during respiratory efforts.

Boyle's Law P1V1=P2V2; the principle that at constant temperature the volume of a gas varies inversely as the absolute pressure applied to the gas.

Compliance Volume change per unit of pressure change across an elastic structure.

Compliance Curve The pressure-volume curve for the lung or relaxed chest wall; plotting volume as a function of pressure inside minus pressure outside. The slope of this curve is the compliance.

Dead Space The portion of each breath that does not participate in gas exchange. Anatomic dead space is the volume of the conducting airways; physiologic dead space also includes the contribution of alveoli that are well-ventilated but poorly perfused.

Distending Pressure The inside pressure minus the outside pressure of an elastic structure; for the lung, this is also referred to as the transpulmonary pressure or the recoil pressure of the lung.
FEV_1 Forced expiratory volume in one second; the volume that a subject can exhale in the first second during a forced expiration test.

FEV_1/FVC The volume that a subject can forcibly expel in one second (FEV1) divided by the total volume that can be expelled (FVC); this result can be especially useful in diagnosing obstructive and restrictive disorders.

Forced Expiration The recording of a maximal expiration from Total Lung Capacity (TLC). This permits the of forced vital capacity (FVC) and various of air flow.

FRC Functional Residual Capacity Also known as FRC, this is the lung volume at the end of a normal expiration, when the muscles of respiration are completely relaxed; at FRC and at FRC only, the tendency of the lungs to collapse is exactly balanced by the tendency of the chest wall to expand.

Functional Residual Capacity Also known as FRC, this is the lung volume at the end of a normal expiration, when the muscles of respiration are completely relaxed; at FRC and at FRC only, the tendency of the lungs to collapse is exactly balanced by the tendency of the chest wall to expand.

FVC Forced vital capacity; the total volume of air that can be exhaled from the lungs during a forced expiration following a maximal inspiration.

Residual Volume Also known as RV, this is the volume of the lungs after a maximal expiration—The lowest voluntary volume attainable.

Tidal Volume Also known as VT, this is the volume of an individual breath during quiet breathing. It averages about 500 ml.

TLC Total Lung Capacity. Also known as TLC, this is the volume of the lungs after a maximal voluntary inspiration.

VC Vital Capacity. Also known as VC, this is the difference between Total Lung Capacity (TLC) and Residual Volume (RV); i.e, it is the maximum volume of air which can be exhaled starting at full lung inhalation.

Mechanical Ventilation Test

Select the ***best*** answer to each of the following items. Mark your responses on the Answer form.

1. Respiratory failure is caused by failure to ventilate, characterized by _____ or failure to oxygenate, characterized by decreased arterial oxygen tension.

a. decreased arterial carbon dioxide tension
b. increased arterial carbon dioxide tension
c. increased pulmonary carbon monoxide levels
d. decreased pulmonary carbon monoxide levels

2. The treatment for failure to ventilate is to_____ , that is the rate and depth of breathing, either by reversing the cause or by using mechanical ventilation; invasively or non-invasively.

a. increase the patient's alveolar ventilation
b. decrease the patient's alveolar ventilation
c. increase the patient's surfactant levels
d. decrease the patient's surfactant levels

3. _____ is a term which describes a patient fighting the ventilator.

a. Resistance
b. Dysynchrony
c. Counter-balancing flow
d. None of the above

4. Modes of ventilation describe the primary method of _____ assistance.

a. inspiratory
b. flow volume
c. pressure
d. surfactant

5. The ideal level of PEEP is that which puts the majority of lung units on the favorable part of the pressure-volume curve (remember each lung unit has a different curve), maximizes gas exchange and minimizes _____.

a. flow
b. surfactant intake
c. over-distention
d. None of the above

6. Hysteresis is the tendency of the lungs, due to _____, to exist at higher volumes in expiration than in inspiration.

a. surfactant
b. nitric oxide
c. mucus
d. None of the above

7. A mechanical ventilator is a machine that generates a _____ into a patient's airways.

a. mist
b. spray
c. controlled flow of gas
d. medicinal mixture

8. Inhalation serves to replenish alveolar gas. Prolonging the duration of the higher volume cycle enhances oxygen uptake, while increasing _____ pressure and reducing time available for CO_2 removal.

a. lung
b. volume
c. intrathoracic
d. None of the above

9. Failure to oxygenate is caused by reduced _____ and ventilation perfusion mismatch.

a. breathing rate
b. diffusing capacity
c. pressure
d. None of the above

10. The magnitude of PEEP is determined by a _____on the expiratory valve.

a. knob
b. switch
c. spring loaded mechanism
d. None of the above

11. Ventilator “cycling” refers to the mechanism by which the _____ switches from inspiration to expiration.

a. timing mechanism
b. flow rate
c. phase of the breath
d. None of the above

12. Mechanical ventilation is used when natural (spontaneous) breathing is absent (apnea) or insufficient. This may be the case in cases of _____. intoxication, , neurological disease or, paralysis of the breathing muscles due to spinal cord injury, or.

a. circulatory arrest
b. the effect of anesthetic or muscle relaxant drugs
c. head trauma
d. All of the above

13. The main advantage of volume-controlled ventilators is guaranteed minute ventilation. This is particularly important in _____.

a. pediatric patients
b. elderly patients
c. the operating room
d. None of the above

14. There is _____ difference between mandatory and spontaneous breaths.

a. little
b. no
c. a considerable
d. None of the above

15. The inspiratory flow rate determines how quickly the breath is delivered. The time required to complete inspiration is determined by the _____ and the flow rate: Ti = VT/Flow Rate.

a. flow volume
b. potential volume
c. tidal volume delivered
d. None of the above

16. When SIMV is used, the patient receives different types of breath, including:

a. The controlled (Mandatory) breath.
b. Assisted (synchronized) breaths.
c. Spontaneous breaths, which can be pressure supported
d. All the above

17. Flow of gas is calculated in _____.

a. milliliters per second
b. liters per minute
c. milliliters per minute
d. tidal volume terms

18. The normal flow pattern of gas moving in and out of the lungs is _____.

a. irregular
b. sinusoidal
c. regular
d. rapid

19. Pressure Control refers to _____, not the mode of ventilation.

a. person controlling the machine
b. the knobs found at the top right of the machine
c. the type of breath delivered
d. none of the above

20. Pressure assist ventilation (PAV) is pressure control _____.

a. with the rate set high
b. with the rate set low
c. without a set rate
d. none of the above

21. In _____, a negative pressure is created in the pleural cavity by the muscles of respiration, and the resulting gradient between the atmospheric pressure and the pressure inside the thorax generates a flow of air.

a. assisted breathing
b. spontaneous breathing
c. weaned
d. None of the above

22. The lungs of ventilated patients have a tendency to _____, leading to impaired gas exchange.

a. fill up
b. spontaneously expand
c. collapse partially
d. None of the above

23. The gist of CPAP is that the technique is delivered to _____ (and cannot support life in an unconscious patient on it's own).

a. asthmatic patients
b. spontaneous breathing patients
c. neonatal patients
d. None of the above

24. Mechanical ventilation will be unsuccessful and dangerous unless the patient's airways are _____, meaning air can flow unimpeded back and forth into the lungs.

a. patent
b. diseased
c. overdistended
d. None of the above

25. In _____, an English clergyman and physiologist named Stephen Hales invented a device to aid people on ships and in mines. This was the first mechanical ventilator.

a. 1743
b. 1803
c. 1843
d. 1903

26. During the 1920s, it was discovered that the lungs worked on the principle of _____ to inflate the chest cavity. Attempts to duplicate this negative pressure brought about the likes of the iron lung.

a. expired fraction of carbon dioxide
b. positive thoracic pressure
c. negative thoracic pressure
d. None of the above

27. In 1978, Coon and his colleagues developed a ventilator that had a(n) _____ for monitoring the pH or pCO_2 levels of the subject.

a. intra-arterial sensor
b. electronic sensor
c. physiological sensor
d. None of the above

28. Ventilation is used as a therapeutic technique to keep appropriate arterial levels of_____, and, at the same time, to minimize risk, discomfort, and time on ventilation.

a. oxygen
b. pH
c. CO_2
d. All of the above

29. Low tidal volume and high respiratory frequency get rid of the risk for a buildup of high trans-thoracic pressures, while high tidal volume and low respiratory frequency help to _____ and to avoid problems due to airway resistance.

a. increase ventilation distribution
b. decrease ventilation distribution
c. reduce lung volumes
d. None of the above

30. When a patient suffers from impaired gas exchange, mechanical ventilation controlling parameters that are able to remove an adequate quantity of CO_2 may not oxygenate the patient's blood sufficiently since oxygen diffuses more slowly than CO_2. In such patients, hypoxia can be avoided through _____.

a. decreasing tidal volume
b. use of inspired gas
c. hyperventilation of the lungs
d. None of the above

31. Generally speaking, mechanical ventilators try to give the patient a ventilation aid which is as close as possible to normal respiration. For example, the inspired gas needs to be at a temperature between 32 C and 39 C with a _____% relative humidity to avoid the risk of airway drying.

a. 50
b. 75
c. 90
d. None of the above

32. There is no clinical evidence that a PaO_2 greater than normal is advantageous.

a. True
b. False

33. To achieve and maintain an increased FRC using PEEP in settings in which a reduction in FRC may be detrimental (*e.g.,* decreased PaO_2, increased lung injury) as in adult respiratory distress syndrome (ARDS) and postoperative pain.

a. True
b. False

34. Mechanical hyperventilation to arterial carbon dioxide levels of 25 to 30 mm Hg acutely lowers ICP. Controlled data on the impact of hyperventilation in patients with head trauma are not available. Decreases in ICP reflect increases in cerebral perfusion pressure.

a. True
b. False

35. In severe congestive heart failure, positive pressure ventilation (PPV) would be expected to _____ venous return.

a. increase
b. decrease
c. have no impact on
d. None of the above

36. The use of high inspiratory flow rates will maximize expiratory time and minimize VEE and intrinsic PEEP (auto-PEEP, dynamic hyperinflation). This is accomplished, however, at the expense of _____ in the central airways.

a. a lowering of peak airway pressure (PAP)
b. higher peak airway pressure (PAP)
c. lower end-inspiratory lung volume
d. None of the above

37. Patients with unilateral lung disease who require mechanical ventilation are _____.

a. common
b. infrequently encountered
c. usually encountered in the ER
d. None of the above

38. Blood returns to the thorax along a pressure gradient from peripheral vessels to the right atrium (RA).

a. True
b. False

39. One of the main objectives of ventilatory support is to ensure that tissues are provided with their O_2 requirements.

a. True
b. False

40. Dynamic hyperinflation develops in the setting of high expiratory resistance or expiratory flow limitation and is influenced by _____.

a. the compliance of the respiratory system
b. the volume from which exhalation begins
c. the expiratory time
d. All of the above

41. High fractions of inspired O_2 (FIO_2) are potentially injurious when applied over extended periods.

a. True
b. False

42. Tips on preventing the development of pneumonia in ventilated patients include _____.

a. Perform chest physiotherapy
b. Meticulously wash your hands
c. Manage your patient's pain so he doesn't guard his breathing
d. All of the above

43. _____, the collapse of alveoli, can occur as a result of inadequate tidal volume or secondary to pneumonia.

a. Pneumonia
b. Atelectasis
c. hypoxemia
d. None of the above

44. A patient who receives oxygen at concentrations greater than _____% for as little as 16 to 24 hours is susceptible to oxygen toxicity.

a. 30
b. 50
c. 70
d. None of the above

45. Problems that may interfere with your patient's ability to tolerate weaning off of mechanical ventilation include _____.

a. underlying respiratory disease
b. poor nutrition
c. neuromuscular dysfunction
d. All of the above

46. Tips to avoid problems when weaning patients off of mechanical ventilation include _____.

a. begin weaning soon after mechanical ventilation is initiated
b. provide adequate nutrition
c. minimize use of sedating medications
d. All of the above

47. Respiratory distress and hypoxemia signal weaning difficulty, so when you attempt to wean your patient, monitor for _____.

a. tachycardia
b. dyspnea
c. a respiratory rate greater than 30
d. All of the above

48. Pulse oximetry, which is now more common than ABGs for monitoring oxygen saturation, has the advantage of being continuous and noninvasive.

a. True
b. False

49. Weaning protocols generally fall into 2 categories: those for assessing readiness to wean and those that guide the weaning process itself.

a. True
b. False

50. The determination that a patient is completely weaned from the ventilator is generally straightforward and is based on the patient's ability to meet certain criteria, such as maintaining a normal respiratory rate or tidal volume while breathing spontaneously.

a. True
b. False

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