Lung Volumes and Mechanical Ventilation

This is a rather comprehensive outline on the subject. So be selective and feel free to skip through the tedium that is not applicable to your interest.

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Principles of Surgery
2002-2003

Basics of Ventilation Aerodynamics

The airways consist of a series of branching tubes that become narrower, shorter, and more numerous as they penetrate deeper into the lung. This process continues down to the terminal bronchioles, which are the smallest airways without alveoli. All these bronchi make up the conducting airways. Their function is to lead inspired gas to the gas-exchanging regions of the lung. Because the conducting airways contain no alveoli and therefore take no part in gas exchange, they constitute the anatomic dead space.

Each terminal bronchiole serves a respiratory unit, or acinus. The terminal bronchioles divide into respiratory bronchioles that have occasional alveoli budding from their walls.

Finally, we come to the alveolar ducts, structures that are completely lined with alveoli. This alveolated region of the lung where gas exchange occurs is known as the respiratory zone.

The region distal to the terminal bronchioles is sometimes referred to as the transitional and respiratory zone because the nonalveolated regions of the respiratory bronchioles do not strictly have a respiratory function. The distance from the terminal bronchiole to the most distal alveolus is only about 5 mm, but the respiratory zone makes up most of the lung (its volume being some 2 to 3 L).

From a functional point of view, the morphology of the human airways was greatly clarified by the studies of Weibel. He measured the number, length, width, and branching angles of the airways. He proposed models that although they are idealized, make pressure-flow and other analyses much more tractable.
The most commonly used Weibel model is the so-called model A shown in Figure 1.

![Figure 1 Idealization of the human airways according to Weibel’s model A. Z = airway generation; BR = bronchus; BL = bronchiole; TBL = terminal bronchiole; RBL = respiratory bronchiole; AD = alveolar duct; AS = alveolar sac. Note that the RBL, AD, and AS make up the transitional and respiratory zone. (From Weibel ER: Morphometry of the Human Lung. Berlin and New York: Springer-Verlag, 1963.)](image)

Note that the first 16 generations (Z) make up the conducting airways ending in the terminal bronchioles. The next three generations constitute the respiratory bronchioles, in which the degree of alveolation steadily increases. This is the transitional zone. Finally, there are three generations of alveolar ducts and one generation of alveolar sacs. These last four generations constitute the true respiratory zone.

Aside: This idealized, dichotomously branching airway system is clearly an oversimplification. For example, in some regions of the human lung, there are far fewer than 23 generations from the trachea to the alveolar sacs, whereas other regions contain more generations. Some of the inadequacies of the model have been pointed out by Horsfield and colleagues,[2] who have proposed other models, particularly of the most distal regions of the airways. In some respects, it makes more sense to begin counting at the terminal alveoli and work backwards toward the origin. Such a system has been used to classify the tributaries of rivers.

However, the Weibel model has been of great value to respiratory physiology. When observing the total cross-sectional area of the airways of each generation is calculated, there is relatively little change in area until we approach generation 16, that is, the terminal bronchioles. However, near this level, the cross-sectional area increases very rapidly. This has led some physiologists to suggest that the shape of the combined airways is similar to a trumpet or even a thumbtack!
The result of this rapid change in area is that the mode of gas flow changes in the region of the terminal bronchioles. Proximal to this point, flow is convective, or "bulk," that is, the sort of flow that occurs when beer is poured out of a pitcher. However, when the gas reaches the region approximating the level of the terminal bronchioles, its forward velocity decreases dramatically because of the very rapid increase in cross-sectional area. As a consequence, molecular diffusion begins to take over as the dominant mode of gas transport. Indeed, diffusion within the gas phase is essentially the only mechanism of gas flow in the alveoli. Who cares? Well…

Many aerosol particles penetrate to the region of the terminal bronchioles by convective flow, but they do not penetrate further because of their large mass and resulting low diffusion rate. Thus, sedimentation of these particles is heavy in the region of the terminal respiratory bronchioles. This is one reason why this region of the lung is particularly vulnerable to the effects of air pollutants.

**Lung Volumes**

![Ventilatory tracing showing normal resting ventilation followed by a forced inhalation to total lung capacity (TLC) and then a forced exhalation to residual volume (RV). Functional residual capacity (FRC) is the resting lung volume at the end of a normal exhalation. Expiratory reserve volume (ERV) is the volume of air that can be exhaled from FRC down to RV.](image)

**Definitions.**

- Total lung capacity is the volume of gas contained in the lungs at maximal inspiration.
- The vital capacity is the volume of gas that can be exhaled by a maximal expiration from total lung capacity.
- The residual volume is the volume remaining in the lung after maximal expiration.
- Tidal volume refers to the normal respiratory volume excursion.
The functional residual capacity is the lung volume at the end of a normal expiration.

The diagram also indicates the inspiratory reserve volume and the expiratory reserve volume.

![Figure 3](image)

**Figure 3** A portion of a normal spirogram showing the forced exhalation from total lung capacity. FEV<sub>1</sub> is the volume exhaled in 1 second, and forced vital capacity (FVC) is the total volume that can be forcibly exhaled from total lung capacity.

**Spirometric Measurements of Expiratory Flow**

With common computerized equipment, more than 20 spirometric variables are often reported. The use of large numbers of variables can lead to false-positive findings, and it is recommended that only a few basic variables from the lung spirogram be used.

Among the most useful variables are FEV<sub>1</sub>, FVC, and the ratio of FEV<sub>1</sub> to FVC (Fig. 2). FVC is the maximal volume of air that can be exhaled during a forced exhalation beginning at TLC. FEV<sub>1</sub> is the volume of gas that can be exhaled during the first 1 second of a forced exhalation maneuver beginning at TLC. Spirometric measurements of lung function are most useful when the patient has physical findings, symptoms, or risk factors suggesting pulmonary disease. Lung functional studies can be used to define the basic class of a lung disorder, evaluate the severity of the abnormality being quantitated, or follow the progression of the disease process. It has been shown that physicians cannot consistently and reliably identify obstructive and restrictive ventilatory defects from history taking or physical examination. Age-related declines in lung function must be considered in evaluating test results. Non-smokers lose FEV<sub>1</sub> at a rate of 20 to 30 mL/year. In some smokers, this rate of decline can increase by two- to three-fold. In smokers younger than 35 years, quitting smoking can result in an increase in lung function. In smokers older than 35 years who quit smoking, the rate of decline of lung function generally slows to the normal rate associated with aging.

The magnitude of functional impairment in obstructive lung disease can be assessed using pulmonary function testing. When the predicted FEV<sub>1</sub> is close to 4 L, the subject should not have
a history of a significant exercise impairment until the FEV\textsubscript{1} falls below 3 L/second. In that individual, an FEV\textsubscript{1} between 2 to 3 L/second would be consistent with a history of mild exercise limitation. Mild exercise limitation means that the subject is able to walk significant distances but cannot do so at high rates of speed. An FEV\textsubscript{1} of between 1 to 2 L/second is consistent with a moderate degree of exercise impairment, meaning that intermittent rest periods are required to walk significant distances or to climb stairs. An FEV\textsubscript{1} less than 1 L/second predicts a severe exercise impairment, limiting the person to very short walking distances, perhaps restricting him or her to home. These guidelines for assessing severity of an exercise impairment in obstructive lung disease must be adjusted for age and body size in the same manner that predicted FEV\textsubscript{1} varies. It is important to correlate predicted functional capacity by pulmonary function testing with the history of exercise limitation described by the patient. A significant difference in the functional capacity predicted by pulmonary function testing with that described by the patient can be an important indicator of the presence of nonpulmonary disease processes.

**Diffusing Capacity**

The diffusing capacity of the lung is defined as the lung's ability to take up an inhaled nonreactive test gas, such as CO, which binds to hemoglobin. CO binds to hemoglobin with a high affinity so that virtually all the CO that reaches an alveolar space, crosses the alveolar air-blood barrier, and reaches a red cell will bind to hemoglobin and thus be removed from the exhaled gas. Measurement of lung-diffusing capacity is critically influenced by three parameters:

1. the ability of the test gas to reach the alveolar gas-exchanging surfaces,
2. the ability of the test gas to cross the alveolar septa, and
3. the mass of red cells in the pulmonary capillary bed available to bind the test gas.

A defect in any one of the above three components influences measured lung-diffusing capacity. Airways obstruction and ventilation-perfusion mismatching prevent the test gas from reaching the alveolar gas-exchange surfaces and lower the diffusing capacity in proportion to the maldistribution of ventilation. Thickening of the air-blood barrier can increase the resistance of gas movement across the tissue barrier, increasing diffusing capacity. Alveolar filling, as in pulmonary edema, reduces the alveolar surface area available for test gas exchange. Finally, the volume of red cells in the alveolar capillary bed is crucial in determining how much CO is retained in the capillary bed. Of the parameters that influence CO uptake in the lung during a diffusing capacity measurement, the uniformity of ventilation and the volume of red cells in the pulmonary capillary bed dominate the overall reaction kinetics. Diffusing capacity measurements are less sensitive to changes in thickness of the air-blood barrier because CO has a high capacity for diffusion across pulmonary tissues.

The normal values for CO diffusing capacity vary widely between laboratories, and both the absolute values and their reproducibility are strongly influenced by the measurement techniques. Diffusing-capacity measurements are most commonly useful in following changes in a patient's lung function when measured by consistent techniques applied by the same laboratory.
Abnormalities of Co-Diffusing Capacity

Based on the above discussion, it should be apparent that DL<sub>CO</sub> can be altered in patients with a variety of cardiopulmonary disorders. Disorders of distribution of ventilation such as chronic obstructive pulmonary disease (COPD) and asthma are perhaps the most common causes of disordered diffusing capacity.

Because of the high sensitivity of DL<sub>CO</sub> to changes in pulmonary capillary blood volume, any disorder that alters pulmonary capillary blood volume significantly changes DL<sub>CO</sub>. Thus, mitral stenosis or heart failure, by increasing pulmonary vascular volumes, can increase DL<sub>CO</sub>. When heart failure is sufficiently severe to produce pulmonary edema, the alveolar filling decreases the surface area available for interaction with the test gas and may decrease measured DL<sub>CO</sub>. Anemia or polycythemia can alter measured DL<sub>CO</sub>, although the reported value is commonly corrected for changes in blood red cell content by measuring hemoglobin in venous blood near the time the test is carried out. Pulmonary vascular disorders such as pulmonary emboli and vasculitis can decrease the volume of blood in the pulmonary capillary bed and thereby decrease DL<sub>CO</sub>. It is also decreased in patients with a loss of lung tissue, either by surgical resection or by destruction of the lung by a disease process such as emphysema. Finally, DL<sub>CO</sub> may be decreased by interstitial lung diseases, which are characterized by interstitial fibrosis and thickening of the air-blood barrier. These processes were originally thought to be alveolar capillary block syndromes in which the block was thought to be an enhanced tissue thickness that the CO molecules had to cross to reach the pulmonary vascular bed. It is now recognized that these diseases also destroy the pulmonary capillary bed and that the primary cause for a low DL<sub>CO</sub> in these conditions is a decrease in capillary blood volume rather than an alveolar capillary block associated with thickening of the blood-gas barrier.

Pulse Oximetry

Blood oxygenation can be noninvasively and continuously measured using pulse oximetry across an ear lobe or fingertip. This technique uses transmission spectroscopy at two wavelengths (red and near-infrared) to measure oxyhemoglobin and total hemoglobin and thereby estimate blood arterial oxygen saturation (Sa<sub>O2</sub>). Sa<sub>O2</sub> of 80% indicates a Pa<sub>O2</sub> near 50 mm Hg, and an Sa<sub>O2</sub> of 90% indicates a Pa<sub>O2</sub> near 60 mm Hg. The technique has become a standard of practice for critically ill patients and for patients undergoing anesthesia or procedures in which precipitous drops in oxygen saturation can occur. Pulse oxygenation probes are most accurate in high saturation ranges and become less reliable when oxygen saturation is below 75%. Probes for pulse oximeters are sensitive to placement, are prone to movement artefact, and require adequate pulse pressure; interference occurs with carboxyhemoglobin and methemoglobin, which can potentially give falsely high estimates of Sa<sub>O2</sub> in smokers. The readings are limited to oxygen saturation, giving no data on oxygen content of the blood, which cannot be determined without measurement of hemoglobin content. Rapid changes in hemoglobin content or in distribution of perfusion may not be reflected in Sa<sub>O2</sub>. Thus, although the pulse oximeter is a valuable monitoring tool and is reasonably accurate under most conditions, its reliability will diminish as the clinical situation deteriorates; at this time, other methods of assessing the adequacy of tissue oxygen delivery should be used.
Disorders of Ventilation
Diseases are commonly divided into two broad categories--obstructive lung disease and restrictive lung disease--based on fundamental differences in the pulmonary function assessment.

Obstructive Ventilatory Disorders
This term is used for the constellation of diseases characterized by limitation of expiratory air flow. The primary criterion for air flow obstruction is a reduced FEV₁ /FVC%. In the presence of a normal or elevated TLC, the absolute value of the FEV₁ can be used to estimate severity of the obstructive lung disease. The obstruction may be caused by a variety of airway diseases including chronic bronchitis, bronchiectasis, and mucous gland hyperplasia, leading to physical obstruction or plugging of airways. In emphysema, extensive destruction of alveolar and/or airway walls occurs, leading to loss of elasticity and collapse of airway walls during exhalation, thus trapping gas in the distal lung. Emphysema is also associated with abnormalities of diffusing capacity due to extensive destruction of the alveolar capillary bed. Reversible forms of airway obstruction, such as asthma, are classified as obstructive lung diseases in which the abnormality is primarily due to restriction of size of the airway walls by inflammation, enhanced muscular tone, and/or enhanced mucus secretion. If underlying tissue destruction does not occur, this form of obstructive lung disease can be reversible.

Restrictive Ventilatory Disorders
This class of lung dysfunction is characterized by fibrotic reactions of the alveolar septa and commonly includes the walls of small airways. The increased fibrotic tissue increases the elastic recoil in parenchymal lung tissue, and because these walls are interconnected to airway walls, the airways can be held open. The hallmarks of restrictive lung diseases are a low TLC and a fall in FVC. When caused by a fibrotic process, it is associated with smaller alveoli having thickened walls, increased amounts of elastic and connective tissue, and destruction of portions of the pulmonary capillary bed. Airways are generally held open by enhanced elastic recoil. The enhanced elastic recoil increases the resistance against inspiration, making it difficult for the patient to inhale and lowering the FVC. The enhanced elastic recoil facilitates rapid exhalation; thus FEV₁/FVC% is generally increased in patients with restrictive lung diseases. Other forms of restrictive ventilatory impairment can include

diseases of the chest wall with altered chest wall compliance (obesity, kyphoscoliosis);
neuromuscular diseases in which the patient has difficulty carrying out ventilatory maneuvers (Guillain-Barre syndrome, myasthenia gravis);
diseases of the pleura, which may entrap the lung (extensive pleural thickening);
space occupying lesions in the lung (tumors, cardiac enlargement, pleural effusions); and
removal of portions of the lungs via surgical resection.

Table 1 gives the common changes in lung volumes in various classes of restrictive lung diseases. Note that fibrotic lung diseases cause significant decreases in all lung volumes as the fibrosis creates a smaller, less compliant lung. In contrast, massive obesity tends to preserve the RV
because the underlying lung is normal and the primary problem is inspiring against the excess weight of the chest wall.

<table>
<thead>
<tr>
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<th>Pulmonary Fibrosis</th>
<th>Obesity (Chest Wall Restriction)</th>
<th>Neuromuscular Disorders</th>
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<tbody>
<tr>
<td>TLC</td>
<td>↓</td>
<td>↓</td>
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<tr>
<td>FRC</td>
<td>↓</td>
<td>↓</td>
<td>Normal</td>
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<tr>
<td>ERV</td>
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<td>↓↓</td>
<td>↓</td>
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<tr>
<td>RV</td>
<td>↓</td>
<td>Normal</td>
<td>↑</td>
</tr>
<tr>
<td>Collapse Point</td>
<td>Zero</td>
<td>RV</td>
<td>FRC</td>
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The ERV is often markedly reduced out of proportion to the other lung volumes in obesity, and the FRC approaches RV as the heavy chest wall pushes the “zero energy point” downward. Neuromuscular diseases create a restrictive lung disorder in which all lung volumes move closer to FRC. These patients lack the respiratory strength to fully inhale or exhale from FRC and thus have a low TLC with an elevated RV. The simplified analysis shown in Figure 2 assumes that other processes such as atelectasis are not complicating the classic presentation of these forms of restrictive lung disease.

Exercise
Most pulmonary function measurements are taken while the subject is at rest. Defects in pulmonary function can be brought out by assessing pulmonary function under conditions of exercise. In addition, complaints of fatigue or exercise limitation can be more rigorously assessed by complete cardiopulmonary exercise testing in which cardiac and pulmonary function are simultaneously quantified under conditions of gradually increasing exercise. Measurements of heart rate, electrocardiogram, arterial blood gases, and exhaled gases can allow simultaneous assessment of cardiac and pulmonary function and can both separate disorders of heart and lung function and distinguish these from exercise limitation due to poor cooperation or deconditioning. These tests can also facilitate the diagnosis of pulmonary vascular and parenchymal infiltrative diseases.

Measurement of Functional Residual Capacity, Residual Volume, and Total Lung Capacity
These three volumes cannot be measured with a simple spirometer because there is no way of knowing the volume remaining in the lung after a maximal expiration (i.e., the residual volume). However, if the functional residual capacity is measured, the other two volumes can be derived by simple spirometry. The functional residual capacity can be measured conveniently by helium dilution in a closed circuit. The subject is connected to a spirometer of known volume that
contains a known concentration of helium (a very insoluble gas). He or she then rebreathes until the helium concentration in the spirometer and in the lungs is the same. The exhaled carbon dioxide is absorbed with soda lime, and oxygen is added to maintain a constant total volume. After equilibration, the total amount of helium is assumed to be unchanged because so little of it is removed by the blood because of its very low solubility. The functional residual capacity can then be derived from the following equation:

\[ C_1 \times V_1 = C_2 \times (V_1 + V_2) \]

where \( C_1 \) and \( C_2 \) are the helium concentrations before and after equilibration, \( V_1 \) is the volume of the spirometer, and \( V_2 \) is the volume of the lung. If the subject is switched into the equipment when at functional residual capacity, \( V_2 \) gives that volume.

Another popular way of measuring the functional residual capacity is with a body plethysmograph. This is a large airtight box in which the subject sits. At the end of a normal expiration, a shutter closes the mouthpiece, and the subject is asked to make respiratory efforts. As the subject tries to inhale, the gas in the lungs expands, lung volume increases slightly, and the pressure in the box rises slightly because its gas volume decreases. Boyle's law (pressure times volume is constant at constant temperature) can then be used to calculate the change of volume of the lung. If mouth pressure is also measured during the respiratory efforts, Boyle's law can also be applied to the lung and functional residual capacity can be derived.

In patients with lung disease, the functional residual capacity measured by helium dilution may be substantially less than that measured by body plethysmography. The reason is that the body plethysmograph measures the total volume of gas in the lung, including any that is trapped behind closed airways, and that therefore does not communicate with the mouth. By contrast, the helium dilution method measures only communicating gas or ventilated lung volume. In young normal subjects, these volumes are virtually identical, but they may be considerably different in patients with severe lung disease. Also in these patients, the volume that is obtained for functional residual capacity rises as the time for equilibration is increased because helium continues to penetrate to additional areas of poorly ventilated lung.

**Total and Alveolar Ventilation Definitions**

**Total ventilation** is the total volume of gas exhaled per minute. It is equal to the tidal volume times the respiratory frequency. The volume of air entering the lungs is slightly greater because more oxygen is inhaled than carbon dioxide is exhaled, but the difference is usually less than 1%.

Alveolar ventilation is the amount of fresh inspired air (non-dead space gas) that enters the alveoli per minute and is therefore available for gas exchange. Strictly, the alveolar ventilation is also measured on expiration, but the volume is almost the same.

**Measurement of Alveolar Ventilation**

Because the tidal volume, \( V_T \), is made up of the dead space volume, \( V_D \), and the volume of gas entering (or coming from) the alveoli, \( V_A \), the alveolar ventilation can be measured from the following equations:

\[ V_T = V_D + V_A \]
Multiplying by respiratory frequency gives

\[ V_E = V_D + V_A \]

where \( V_A \) is the alveolar ventilation, and \( V_E \) and \( V_D \) are the expired total ventilation and dead space ventilation, respectively. Alveolar ventilation can be then obtained through easy rearrangement of this equation.

A difficulty with this method is that the anatomic dead space is not easy to measure, although a value for it can be assumed with little error. One millilitre per pound of body weight is a common assumption.

Another way of measuring alveolar ventilation in normal subjects is to use a fancy alveolar ventilation equation that we needn’t bother with for the purposes of POS.

**Anatomic Dead Space**
The anatomic dead space is the volume of the conducting airways. The normal value is in the range of 130 to 180 mL and depends on the size and posture of the subject. The value increases slightly with large inspirations because the radial traction exerted on the bronchi by the surrounding lung parenchyma increases their size.

Anatomic dead space can be measured by Fowler's method, in which a single breath of oxygen is inhaled and the concentration of nitrogen in the subsequent expiration is analyzed.

**Physiologic Dead Space**
Unlike anatomic dead space, which is determined by the anatomy of the airways, physiologic dead space is a functional measurement based on the ability of the lungs to eliminate carbon dioxide.

Physiologic dead space is very nearly the same as anatomic dead space when the lung is normal. However, in the presence of ventilation-perfusion inequality, physiologic dead space is increased, chiefly because of the ventilation going to lung units with abnormally high ventilation-perfusion ratios. Indeed, the physiologic dead space is often reported as one of the indices of the degree of mismatching of ventilation and blood flow within the lung.

**Inequality of Ventilation**
Not all the alveoli are equally ventilated, even in the normal lung. There are several reasons for this, related both to gravitational (topographic) and to nongravitational influences on gas distribution.

**Topographic Inequality**
Regional differences of ventilation can be measured by having the patient inspire a radioactive gas such as xenon (133 Xe).

In one technique, the patient inhales a single breath of gas, and its radiation is detected by a radiation camera placed behind the chest. An additional measurement is made after the patient has rebreathed long enough to allow the xenon to equilibrate throughout the different regions of
the lungs, thus reflecting regional lung volumes. By comparing the first and the second measurements, the ventilation per unit alveolar volume can be obtained.

Measurements in upright normal subjects show that the ventilation per unit volume of the lung is greatest near the base of the lung and becomes progressively smaller toward the apex. When the subject lies supine, this difference becomes much less, but the ventilation of the lowermost (posterior) lung exceeds that of the uppermost (anterior). In the lateral decubitus position, the dependent lung is better ventilated.

These results refer to an inspiration from functional residual capacity. However, if a normal subject makes a small inspiration from residual volume, an interesting change in the distribution of ventilation is seen. The major share of the ventilation goes to the apex of the upright lung, whereas the base is very poorly ventilated. Experimental studies show that the intrapleural pressure is less negative at the bottom than the top of the lung. This pattern can be attributed to the weight of the lung, which requires a larger pressure below the lung than above it to balance the downward-acting weight forces.

So who cares? Post op patients who are not ambulating and taking shallow small infrequent breaths secondary to pain will be relying on their top portions of the lung for ventilation. This will predispose lower regions of the lung for atelectasis and pneumonia.

**Airway Closure**

The compressed region of the lung at the base does not have all its gas squeezed out because small airways, probably in the region of the respiratory bronchioles, close first and trap gas in the distal alveoli. This is known as airway closure. It occurs only at lung volumes below functional residual capacity in young normal subjects. However, this volume at which the basal airways close (closing volume) increases with age and may encroach on the functional residual capacity in older, apparently normal people. The reason for this increase is that the aging lung loses some of its elastic recoil and the intrapleural pressures therefore become less negative. Under these conditions, basal regions of the lung may be only intermittently ventilated, with resulting defective gas exchange. A similar situation frequently develops in patients with chronic obstructive pulmonary disease in whom lung elastic recoil may be reduced.

**Nontopographic Inequality**

In addition to the topographic inequality of ventilation caused by gravitational factors, nongravitational mechanisms also exist. This is proved by the fact that astronauts in space show uneven ventilation by both the single breath and the multibreath nitrogen washout methods. A variety of obscure, academically fascinating by POS-irrelevant stuff can be elaborated upon but we shall be spared from that at this point in time.

**Basic Approaches to Mechanical Ventilation**

Three basic concepts must be considered in the initial discussion of intensive ventilatory support:

1. negative-pressure ventilation (NPV) versus PPV,
2. invasive versus noninvasive ventilatory management, and
3. **volume-cycled versus pressure-cycled ventilators.**

NPV is no longer used in acute care and has no role in the ED management of respiratory emergencies. PPV occurs when a preset gas mixture at supraatmospheric pressure is cyclically introduced into the upper airway. The pressure gradient created between the upper airway and the lungs entrains gases into the lungs.

PPV can be delivered invasively through an endotracheal (ET) tube or noninvasively via a face mask or nose mask. Forcing gases into the lungs with PPV involves assuming the risk of several potential adverse effects (see table). Each of these potential complications must be anticipated when any PPV technique, invasive or noninvasive, is used in the ED.

<table>
<thead>
<tr>
<th>Box 1 Potential Adverse Effects of Positive-Pressure Ventilation</th>
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<tbody>
<tr>
<td>Increased mean intrathoracic pressure</td>
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<tr>
<td>Decreased venous return to the heart and</td>
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<tr>
<td>decreased cardiac output</td>
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<tr>
<td>Increased ventilation/perfusion ratio</td>
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<tr>
<td>Decreased renal blood flow and glomerular filtration rate with fluid retention</td>
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<tr>
<td>Air trapping and intrinsic positive end-expiratory pressure (iPEEP, auto-PEEP)</td>
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<tr>
<td>Barotrauma</td>
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<tr>
<td>Nosocomial infections of the lungs and sinuses</td>
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<td>Respiratory alkalosis</td>
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<td>Agitation and increased respiratory distress</td>
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<td>Increased work of breathing</td>
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Ventilators that deliver PPV may be pressure or volume cycled. With pressure-cycled ventilation, a peak inspiratory pressure (PIP) is established for inspiration regardless of tidal volume (VT), inspiratory time, or inspiratory flow rate; when this preset pressure is reached, inspiration is terminated and exhalation occurs passively. With volume-cycled ventilation, inhalation is terminated once a preset VT is delivered, even though peak airway pressures, inspiratory time, and inspiratory flow rates may vary. As the peak pressures in the patient's lungs increase, however, a greater proportion of the preset VT is left behind in the ventilator's circuit. No consensus in the literature exists regarding which approach to PPV is more efficacious. Pressure-cycled ventilators are less useful than volume-cycled respirators in critically ill patients with rapidly changing pulmonary mechanics because, if compliance decreases or airway resistance increases, hypoventilation might result. Pressure-cycled machines are generally much less expensive than their volume-cycled counterparts, however, and are in widespread clinical use.