CLINICAL MANIFESTATIONS OF RESPIRATORY DISEASE

Lungs can be affected by diseases or trauma, causing anatomic alterations that can be classified in two categories: *obstructive lung disorders* and *restrictive lung disorders*. This classification system is important because it helps us apply and develop ventilatory strategies based on a group of diseases or trauma.

Table 1 Manifestations of Respiratory Disorders

Respiratory Disorder	*Obstructive*	*Restrictive*	*Combined*
Chronic bronchitis	v		
Emphysema	v		
Asthma	v		
Bronchiectasis			v
Cystic Fibrosis			v
Pneumoconiosis			v
Pneumonia		3	
Pulmonary edema		3	
Near-drowning		3	
Adult respiratory distress syndrome		3	
Chronic interstitial lung disease		3	
Flail chest		3	
Pneumothorax		3	
Pleural diseases		3	
Kyphoscoliosis		3	
Tuberculosis		3	
Fungal diseases		3	
Infant respiratory distress syndrome		3	

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OBSTRUCTIVE LUNG DISORDERS

Disorders of the respiratory system that manifest as obstructive disorders have common findings when looking at lung volumes and capacities.

Obstructive lung disorders tend to have the following characteristics:

Table 2 Characteristics of Obstructive Lung Disorders

Increased tidal volume (V_T)
Increased residual volume (RV)
Increased functional residual capacity (FRC)
Increased residual volume/total lung capacity ratio (RV/TLC)
Increased/normal total lung capacity (TLC)
Decreased/normal vital capacity (VC)
Decreased inspiratory capacity (IC)
Decreased expiratory reserve volume (ERV)

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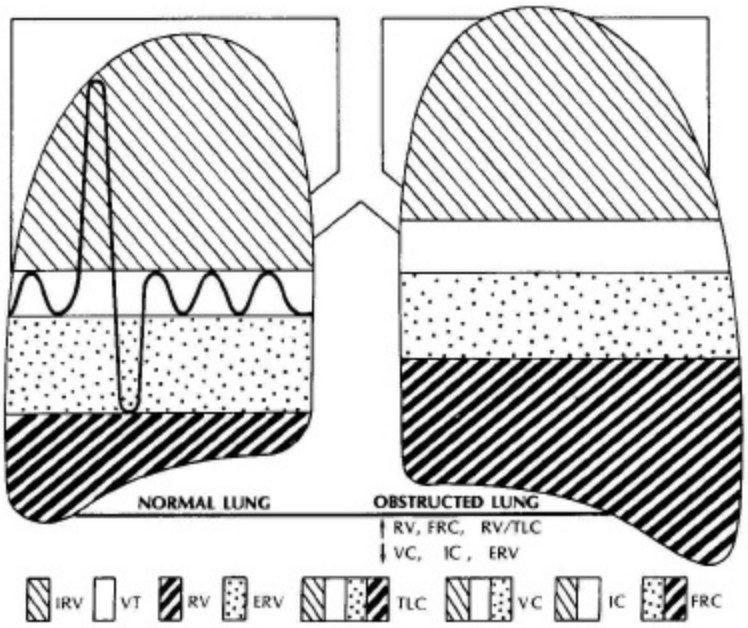

Figure 1 lung volumes and capacities typical of obstructive lung disorders

These changes in volumes and capacities occur in response to pathological changes in the lungs (see fig. 1). Many of these changes are common findings in several obstructive lung disorders:

- inflammation and swelling of the peripheral airways
- excessive mucus production and accumulation
- bronchial airway obstruction (e.g. from mucus or from a tumor projecting into a bronchus)
- destruction and weakening of the distal airways
- smooth muscle constriction of the airways (bronchospasm)

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This section will provide a brief overview of the common obstructive lung disorders and their manifestations.

VENTILATORY STRATEGIES

Ventilatory strategies for obstructive lung disorders are aimed at trying to minimize hyperinflation and air trapping, decreasing the amount of bronchoconstriction, and decreasing the resistance of the patient and the ventilator circuit. Strategy should also include a pattern of ventilation that allows as much time as possible for the patient to exhale. Some ventilatory changes are outlined below.

The less time that is spent in inspiratory time, the longer can be spent in expiratory time. To decrease inspiratory time in volume ventilation, the flow rate can be increased. This will deliver the tidal volume faster and then allow more time for

expiration. The inspiratory flow pattern should be set on a square flow waveform, as this will result in a shorter inspiration when compared with a decelerating flow waveform.

The I:E ratio can be decreased to allow a shorter time in inspiration and a longer time in expiration.

The respiratory rate can be decreased to prolong the expiratory time and provide more time for exhalation (this is the single most important ventilator change that can have the biggest effect on expiratory time).

Bronchodilators can be given to decrease the amount of airway resistance, therefore decreasing the time needed to exhale.

The size of the endotracheal tube can be changed. According to Poiseille's Law, the radius of the tube that flow is passing through has the greatest impact on the resistance of that tube (it increases resistance by a factor of 4). This same law applies to the travel of flow through the airways. Changing the endotracheal tube to one with a greater diameter even just 0.5 cm larger can have a great impact on decreasing autopeep and improving ventilation.

AUTOPEEP

Patients with obstructive lung disorders have a greater resistance in their airways, causing a decrease in their inspiratory and expiratory flows. On exhalation, small and medium sized airways can close off or collapse, causing air trapping and hyperinflation. When these patients are mechanically ventilated, they may not be given enough time to exhale completely between positive pressure breaths. This causes an increase in their FRC, resulting in hyperinflation, barotrauma, hemodynamic instability, and pneumothoraces. The trapping of air seen with mechanical ventilation is called "autopeep".

There are two forms of auto-peep.

1. Auto-peep can occur in patients with normal lungs who do not have any restrictive lung disorder. If the respiratory rate and tidal volumes are set too high, and the patient does not have time to exhale completely before the next breath begins, air can become trapped in the lungs. This can progressively increase the FRC to the point where pneumothorax or other forms of barotrauma can become likely. Autopeep can also occur in patients with normal lungs when inspiratory times are set too high on the ventilator, not giving the patient enough expiratory time to completely exhale. Autopeep can be caused by anything that increases resistance to air flow such as a small diameter endotracheal tube or a kink in the expiratory circuit.
2. Autopeep can occur in patients with obstructive lung disease when their smaller airways collapse on expiration, trapping air inside. Because they have a high resistance to gas flows in their airways due to increased secretions, bronchial smooth muscle constriction, and bronchial inflammation, their exhaled flows are slow. This further impedes their exhalation and can result in high autopeep.

Autopeep should be suspected in any patient on a mechanical ventilator with a history of an obstructive disorder. Most mechanical ventilators (PB 7200, PB 840, Bear 1000, Siemens 900C, Siemens 300) have the capabilities to measure autopeep by placing an expiratory hold immediately before the beginning of the next inspiration. As the

patient continues to exhale against the closed expiratory valve, the pressure in the circuit will increase. The expiratory hold should be at least 2-3 seconds to measure the full expiration, and even longer in the case of severe airway obstruction. When the pressure in the circuit no longer rises and has reached a plateau, that level is considered to be *total peep*. To calculate autopeep, take the total peep minus the set peep. The difference will be the autopeep.

total peep – set peep = autopeep

Autopeep can also be manually measured on ventilators that do not have this capability. A pressure transducer can be hooked up to a monitor and placed in line in the ventilator circuit. Inspiration and expiration are carefully timed. Immediately prior to the beginning of inspiration, the expiratory line is clamped. The total peep is then printed out and measured from the monitor (mm Hg may need to be converted to cm H_20). In order to detect autopeep, the patient must be either paralyzed or sedated enough that they are not trying to trigger a breath or actively exhale, otherwise the autopeep measurement will not be accurate.

The presence of autopeep can be detected just by looking at flow versus time waveforms on a mechanical ventilator (see fig. 2). If the flow does not return to baseline before the next breath begins, autopeep is present.

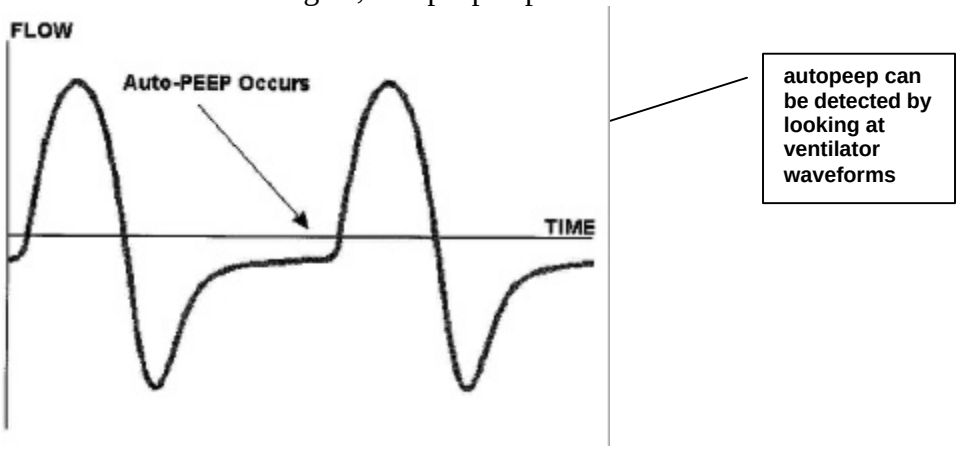

Figure 2 autopeep

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Autopeep will affect the performance of the ventilator in several ways. The measurement of compliance will be altered, because the calculation for compliance is the tidal volume divided by the plateau pressure minus the set peep. In the presence of autopeep, this must be factored into the equation, so that it becomes tidal volume divided by the plateau pressure minus the total peep.

Autopeep will also affect the ability of the patient to trigger the ventilator. For example, if the set peep is 5 cm H_20 and the sensitivity is set at –2 cm H_20, the patient must draw back –2 cm H_20 through the ventilator circuit and humidifier in order to trigger the ventilator. If this patient had an additional 4 cm H_20 of autopeep, they would have to draw back –6 cm H_20 to trigger the ventilator. This problem can usually be resolved by decreasing the sensitivity and increasing the peep to match the total peep.

PERMISSIVE HYPERCAPNIA

In some cases, the above ventilatory manipulations, designed to prevent air trapping and lung damage, can cause the PCO_2 to rise above normal ranges. Permissive hypercapnia is a deliberate decrease in mechanical support (e.g. decreasing the respiratory rate or tidal volume) to protect the lung and allowing the $PaCO_2$ to climb to as high as 100 mm Hg.

Elevation of $PaCO_2$ can have detrimental effects on body function (see table 3).

Table 3 Effects of Hypercapnia

Shift in the oxyhemoglobin dissociation curve to the right
Decreased alveolar PO_2
Both stimulation and depression of the cardiovascular system
Central nervous system
Stimulation of ventilation
Dilation of vascular bed
Increased intracranial pressure
Anesthesia ($PaCO_2$ >200 mm Hg)
Decreased renal blood flow ($PaCO_2$ >150 mm Hg)
Leakage of intracellular potassium ($PaCO_2$ > 150 mm Hg)
Alteration of the action of pharmacologic agents (a result of intracellular acidosis)

Kacmarek RM, Hickling KC. Permissive Hypercapnia. Respiratory Care 1993; 38:373-387.

Most of the physiologic problems associated with permissive hypercapnia occur when the $PaCO_2$ increases greater than 150 mm Hg. However, in patients with head injuries at high risk of increased intracranial pressure, permissive hypercapnia should be avoided, as even a small increase in $PaCO_2$ can result in an increase in intracranial pressure and cerebral swelling or edema.

An increase in $PaCO_2$ causes a strong stimulation to increase ventilation, but most of the patients who need this ventilatory technique are already paralyzed or sedated, therefore this is not usually an issue.

One of the important complications that can occur due to a rising level of $PaCO_2$ is an increase in cardiac output. This will concomitantly increase the heart rate as well as the level of cardiac work. This can cause myocardial ischemia. Therefore, permissive hypercapnia may not be the best ventilation strategy in a patient with a history of cardiac dysfunction.

Permissive hypercapnia can cause a decrease in oxygenation. When the oxyhemoglobin dissociation curve shifts to the right, the affinity for oxygen is reduced. As well, an increase in alveolar PCO_2 will cause a decrease in alveolar PO_2, therefore the patient in whom permissive hypercapnia is used as a ventilatory strategy must have adequate oxygenation.

The level that $PaCO_2$ is allowed to rise is limited by the pH. In the absence of cardiovascular disease or renal failure, patients can usually tolerate a pH of 7.20 – 7.25.

This needs to be evaluated on an individual basis. The renal system will gradually compensate for increases in carbon dioxide by increasing the level of plasma bicarbonate. This will help to decrease the level of acidosis.