Know this!

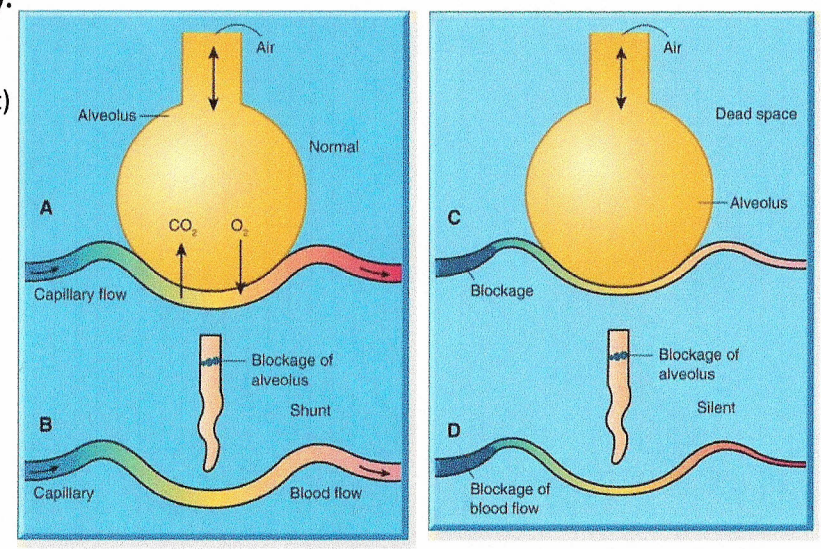
Table 21-8 Pulmonary Function Tests			
Term Used	Symbol	Description	Remarks
Forced vital capacity	FVC	Vital capacity performed with a maximally forced expiratory effort	Forced vital capacity is often reduced in COPD because of air trapping.
Forced expiratory volume (qualified by subscript indicating the time interval in seconds)	FEV_t (usually FEV_1)	Volume of air exhaled in the specified time during the performance of forced vital capacity; FEV_1 is volume exhaled in 1 second	A valuable clue to the severity of the expiratory airway obstruction
Ratio of timed forced expiratory volume to forced vital capacity	FEV_t/FVC%, usually FEV_1/FVC%	FEV_t expressed as a percentage of the forced vital capacity	Another way of expressing the presence or absence of airway obstruction
Forced expiratory flow	$FEF_{2001200}$	Mean forced expiratory flow between 200 and 1200 mL of the FVC	An indicator of large airway obstruction
Forced midexpiratory flow	$FEF_{2575\%}$	Mean forced expiratory flow during the middle half of the FVC	Slowed in small airway obstruction
Forced end expiratory flow	$FEF_{7585\%}$	Mean forced expiratory flow during the terminal portion of the FVC	Slowed in obstruction of smallest airways
Maximal voluntary ventilation	MVV	Volume of air expired in a specified period (12seconds) during repetitive maximal effort	An important factor in exercise tolerance

Pulmonary Diffusion and Perfusion

Diffusion is the process by which O2 and CO2 are exchanged at the air–blood (alveolar–capillary) interface.
In normal healthy adult, O2 and CO2 cross the thin alveolar–capillary membranes easily due to the differences in gas concentrations.
Pulmonary perfusion is the actual blood flow through the pulmonary circulation.
Blood is pumped into the lungs by the right ventricle through the pulmonary artery. The pulmonary artery divides into the right and left branches to supply both lungs. These two branches divide further to supply all parts of each lung.
Normally about 2% of the blood pumped by the right ventricle does not perfuse the alveolar capillaries.
This shunted blood drains into the left side of the heart without participating in alveolar gas exchange.
Pulmonary artery pressure, gravity, and alveolar pressure determine the patterns of perfusion.
Pulmonary circulation is considered a low-pressure system because the systolic pressure in the pulmonary artery is 20-30mm/Hg and the diastolic BP is 5-15 mm/Hg.
Due to these low pressures, the pulmonary vasculature normally can vary its capacity to accommodate the blood flow it receives.
But, **gravity affects perfusion** to the lungs:
When a pt is upright, pulmonary artery pressure isn't great enough to supply blood to the apex of the lung against gravity, the lung is considered to be divided into three sections: an upper part with poor blood supply, a lower part with max blood supply, and a middle section between the two with an intermediate supply of blood.
When a pt who is supine turns to one side, more blood passes to the dependent lung.
Perfusion is also **affected by alveolar pressure**.
Pulmonary capillaries are sandwiched between adjacent alveoli.
If alveolar pressure is sufficiently high, the capillaries are squeezed. With higher pressure some capillaries completely collapse, whereas others narrow.
In lung disease, these factors vary, and the perfusion of the lung may become very abnormal.

Ventilation and Perfusion Balance and Imbalance

Adequate gas exchange depends on an **adequate ventilation–perfusion (V/Q) ratio**.
In different areas of the lung, the ratio varies.
Perfusion alterations may occur with change in pulmonary artery pressure, alveolar pressure, or gravity.
Ventilation alterations may occur with airway blockages, local changes in compliance, and gravity.
V/Q imbalance occurs as a result of inadequate ventilation, inadequate perfusion, or both.
There are four possible V/Q states in the lung:
normal ratio, low (shunt), high ratio (dead space), and absence of ventilation &perfusion (silent unit)
V/Q imbalance causes shunting of blood, resulting in hypoxia (low level of cellular oxygen).
Shunting appears to be the main cause of hypoxia after thoracic or abdominal surgery



Normal Ratio (A) amt of blood passes an alveolus and is matched with equal amt of gas, ratio is 1:1 (ventilation matches perfusion). Low Ventilation–Perfusion Ratio: perfusion exceeds ventilation, a shunt exists (B). Blood bypasses the alveoli without gas exchange occurring. This is seen with obstruction of the distal airways: pneumonia, atelectasis, tumor, or a mucus plug. High Ventilation–Perfusion Ratio: Dead Space , ventilation exceeds perfusion, dead space results (C). alveoli do not have adequate blood supply for gas exchange to occur: pulmonary emboli, pulmonary infarction, and cardiogenic shock. Silent Unit (D) absence of both ventilation and perfusion or with limited ventilation and perfusion: pneumothorax and severe acute respiratory distress syndrome.

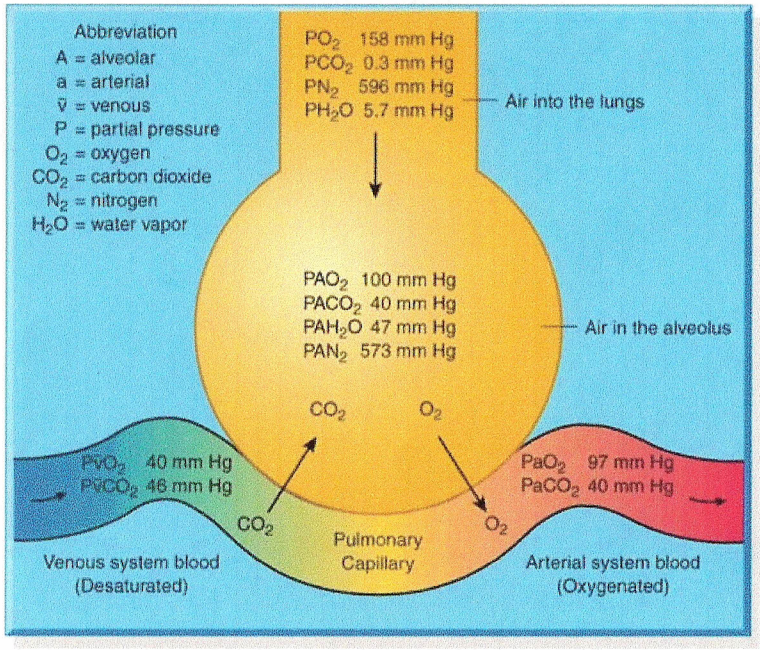
Gas Exchange

O2 and Co2 exist in three physiological areas: in atmosphere and alveoli as gas & in the blood in soln.
Inspired air: 79% nitrogen, 21% O2, and, 0.04% CO2, 0.5% water vapor
Alveoli air: 74.9% nitrogen, 13.6% O2, and, 5.3% CO2, 6.2% water vapor
Expired air: 74.5% nitrogen, 15.7% O2, and, 3.6% CO2, 6.2% water vapor
O2 &CO2 diffuse from alveolar space→capilaries→tissues→and back, based on their conc & pressures
Arterial blood transports O2 to tissues in 2 ways, 3% dissolved in plasma & 97% bound to hemoglobin
Low ph and higher temperatures enhances O2 release from hemoglobin
Blood transports CO2 to the lungs in several forms,
a sm amt is dissolved in plasma,
some loosely combines w/ hemoglobin,
And, as hemoglobin liberates O2 to supply tissue, most is converted in the RBCs (by the NZ carbonic andhydrase) to carbonic acid (H2CO3), then carbonic acid dissociates into HCO3- and H+.
Once HCO3- lvls in the RBC exceed plasma lvls it diffuses out to the plasma where it combines w/ sodium and the plasma transports it as sodium bicarbonate.
As HCO3- is diffusing out of the RBCs, Cl- diffuses in to replace it (this is called a Chloride shift).
In the lungs the reverse process occurs. HCO3- diffuses into the RBC, O2 is picked up, carbonic acid is reformed, and it then dissociates to H2O and CO2. CO2 is then released into the alveoli and expired.
These processes are rapid, simultaneous, and continuous.

Neurologic Control of Ventilation

Resting respiration is the result of cyclic excitation of the respiratory muscles by the phrenic nerve.
The rhythm of breathing is controlled by respiratory centers in the brain.

- The inspiratory and expiratory centers in the medulla oblongata and pons control the rate and depth.
- The apneustic center in the lower pons stimulates the inspiratory medullary center to promote deep, prolonged inspirations.
- The pneumotaxic center in the upper pons is thought to control the pattern of respirations.
- Several groups of receptor sites assist in the brain's control of respiratory function.
- Central chemoreceptors, located in the medulla, respond to chemical changes in the CSF, which result from an increase or decrease in the pH and convey a message to the lungs to change the depth and then the rate of ventilation to correct the imbalance.
- Peripheral chemoreceptors are located in the aortic arch and the carotid arteries and respond first to changes in PaO_2, then to partial pressure of carbon dioxide ($PaCO_2$) and pH.
- The Hering-Breuer reflex is activated by stretch receptors in the alveoli. When the lungs are distended, inspiration is inhibited; as a result, the lungs do not become overdistended.
- Proprioceptors in the muscles and joints respond to body movements, such as exercise, causing an increase in ventilation. Thus, range-of-motion exercises in an immobile patient stimulate breathing.
- Baroreceptors, also located in the aortic and carotid bodies, respond to an increase or decrease in arterial blood pressure and cause reflex hypoventilation or hyperventilation



Partial Pressure Abbreviations
P = pressure
PP = partial pressure
PO_2 = PP of oxygen
PCO_2 = PP of carbon dioxide
PAO_2 = PP of alveolar oxygen
$PACO_2$ = PP of alveolar carbon dioxide
PaO_2 = PP of arterial oxygen
$PaCO_2$ = PP of arterial carbon dioxide
$Pv–O_2$ = PP of venous oxygen
$Pv–CO_2$ = PP of venous carbon dioxide
P_{50} = PP of O2 when hemoglobin is 50% saturated

Oxyhemoglobin Dissociation Curve
Normal levels—PaO_2 above 70 mm Hg
Relatively safe levels—PaO_2 45 to 70 mm Hg
Dangerous levels—PaO_2 below 40 mm Hg
The normal (middle) curve (N) shows that 75% saturation occurs at a PaO_2 of 40 mm Hg. If the curve shifts to the right (R), the same saturation (75%) occurs at the higher PaO_2 of 57 mm Hg. If the curve shifts to the left (L), 75% saturation occurs at a PaO_2 of 25 mm Hg.