Yanal A. Shafagoj MD. PhD

Textbook of medical physiology, by A.C. Guyton and John E, Hall,

.

- In general the **11** lectures will cover the following Respiratory Physiology Topics: This semester we will have only 11 lectures
- 1. Overview: causes of hypoxia...One lecture
- 2. Mechanics of Breathing (Lung Ventilation)...one lecture.
- 3. Airway Resistance...COPD...2 lectures.
- 4. Lung Compliance...1 lectures...Lung fibrosis, IRDS and ARDS
- 5. Pulmonary circulation Ventilation-Perfusion Ratio... 1 lecture.
- 6. Gas Exchange and Transport...2 lectures
- 7. Regulation of Lung Ventilation, high altitude, exercise
- etc...2 lectures.
- 7. Pulmonary Function Test and Pathophysiology (lung Diseases) and Clinical Applications...one lecture.

- What are the Potential Causes of Hypoxia
 - -inadequate oxygenation of lungs
 - Atmosphere..high altitude
 - decrease muscle activity ..paralysis
 - -pulmonary disease
 - -inadequate transport..

anemia

- anemia, and heart failure
- -inadequate usage
 - Cyanide poisoning

Introduction

- Respiration is the process by which the body takes in and utilizes oxygen and gets rid of CO₂.
- Exchange of gases
- Directionality depends on gradients "Pressure difference"!
 - From atmosphere to blood And from blood to tissues
- Three determinants of respiration
- Respiration depends on three things: the lungs, the blood, and the tissues.

- The lungs:
- The lungs must be adequately ventilated and be capable of adequate gas exchange.
- **Ventilation**: is determined by the activity of the control system (respiratory center), the adequacy of the feedback control systems (neural and hormonal), and the efficiency of the effector system (muscles of respiration).
- **Gas exchange**: depends on the patency of the airways, the pressure gradient across the alveolar-capillary membrane, the diffusability of individual gases and the area and thickness of the exchange membrane.

The Blood:

- The blood must pick up, carry and deliver O_2 and CO_2 in amounts that are appropriate to the body's need. It depends in the presence of adequate amount of the correct type of Hb, the cardiac output, and local perfusion.
- Normal Arterial Blood carries 20 ml O₂/dl blood.....at rest, the tissue extract only 5 ml (25% extraction ratio)...leaving 15 ml/dl in the venous blood
- The lungs, the cardiovascular system, the bone marrow ...etc they all work together to guarantee the availability of this 20 ml of $\rm O_2$ per 100 ml arterial blood.
- To do so we need 15 gm/dl Hb and 100% O₂ Sat. What we need? Two things
- 1. [Hb] 15 gm/dl
- 2. O_2 Sat 100%. To have the Hb to be 100% saturated with O_2 (100% O2 sat)...we need arterial PO_2 =100 mmHg
- Each 1 gm of Hb can carry reversibly 1.34 ml of oxygen....How much 15 gms can carry?

• The Tissues:

- Individual cells must be capable of taking up and utilizing O_2 properly.
- Hypoxia can therefore result from a fault at any point along this lungs-blood-tissue chain.

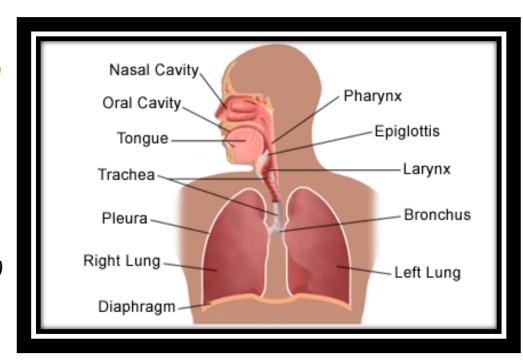
Functions of the respiratory system

• The primary function of the respiratory system is to deliver sufficient amount of O_2 from the external environment to the tissues and to remove CO_2 that is produced by cellular metabolism to the surrounding atmosphere....Therefore, it is homeostasis of O_2 , CO_2 , pH

One more time: To achieve these goals:

respiration can be divided into four major functions:

- (1) Pulmonary ventilation
- (2) Diffusion
- (3) Transport of O₂ & CO₂. (perfusion)
- (4) Regulation of ventilation.



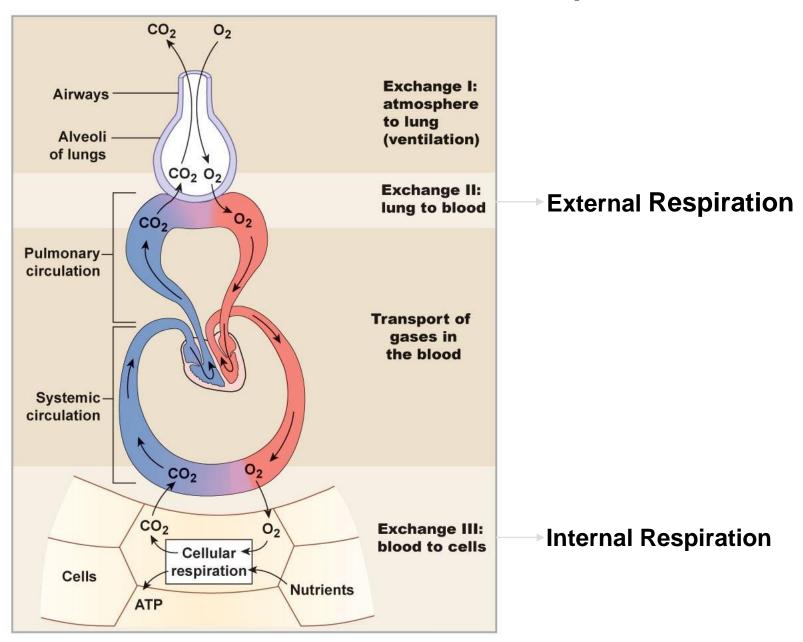
The lungs have several metabolic and endocrine function" the so called "the non respiratory functions"

- Excretion of some volatile waste products e.g. acetone and alcohol.
- Helps blood and lymph flow (venous return)
- Regulation of body temperature by evaporation of water from the respiratory passages to help heat loss from the body
- Regulation of pH...Acid-base balance which dependents on rate of CO2 release
- BP regulation by converting AI to AII
- Protection.....Vocalization etc
- Plus other things you learn them from your lecture outline

Respiration

- What is respiration?
 - Respiration = the series of exchanges that leads to the uptake of oxygen by the cells, and the release of carbon dioxide to the lungs
 - Step 1 = ventilation
 - Which includes: Inspiration & expiration
 - Step 2 = exchange between alveoli (lungs) and pulmonary capillaries (blood)
 - Referred to as External Respiration
 - Step 3 = transport of gases in blood
 - Step 4 = exchange between blood and cells
 - Referred to as *Internal Respiration*
 - Cellular respiration = use of oxygen and ATP synthesis

Schematic View of Respiration



Partial Pressures of Gases in Inspired Air and Alveolar Air

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	Insert fig.	16.20
	Inspired air	Alveolar air
H ₂ O	Variable	47 mmHg
CO ₂	000.3 mmHg	40 mmHg
O ₂	159 mmHg	105 mmHg
N ₂	601 mmHg	568 mmHg
Total pressure	760 mmHg	760 mmHg

Functional Anatomy

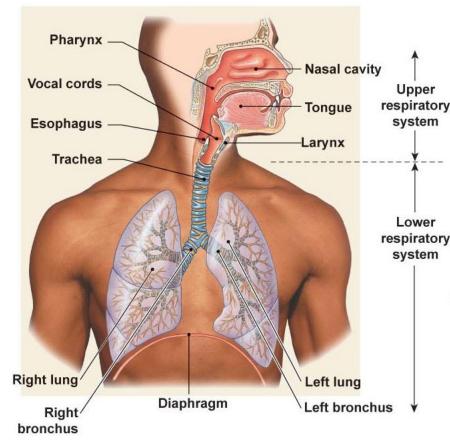
What structural aspects must be considered in the process

of respiration?

• The conducting zone

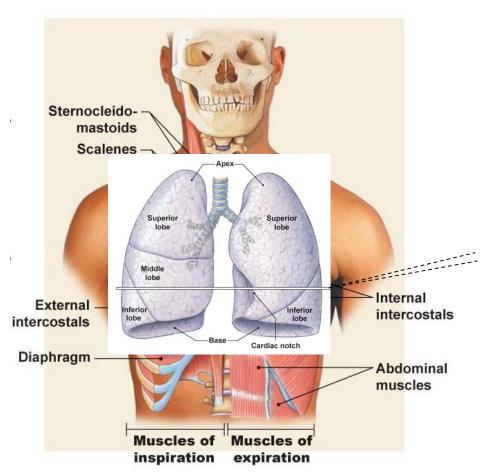
• The respiratory zone

- The structures involved with ventilation
 - Skeletal & musculature
 - Pleural membranes
 - Neural pathways
- All divided into
 - Upper respiratory tract
 - Entrance to larynx
 - Lower respiratory tract
 - Larynx to alveoli (trachea to lungs)



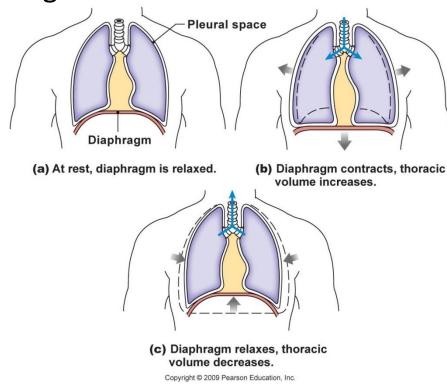
Functional Anatomy

Bones, Muscles & Membranes



Functional Anatomy

- Function of these Bones, Muscles & Membranes
 - Create and transmit a pressure gradient
 - Relying on
 - the attachments of the muscles to the ribs (and overlying tissues)
 - The attachment of the diaphragm to the base of the lungs and associated pleural membranes
 - The cohesion of the parietal pleural membrane to the visceral pleural membrane
 - Expansion & recoil of the lung and therefore alveoli with the movement of the overlying structures



Functional Anatomy

- Pleural Membrane Detail
 - Cohesion between parietal and visceral layers is due to serous fluid in the pleural cavity
 - Fluid (30 ml of fluid) creates an attraction between the two sheets of membrane
 - As the parietal membrane expands due to expansion of the thoracic cavity it "pulls" the visceral membrane with it
 - And then pulls the underlying structures which expand as well
 - Disruption of the integrity of the pleural membrane will result in a rapid equalization of pressure and loss of ventilation function= pneumothorax and collapsed lung

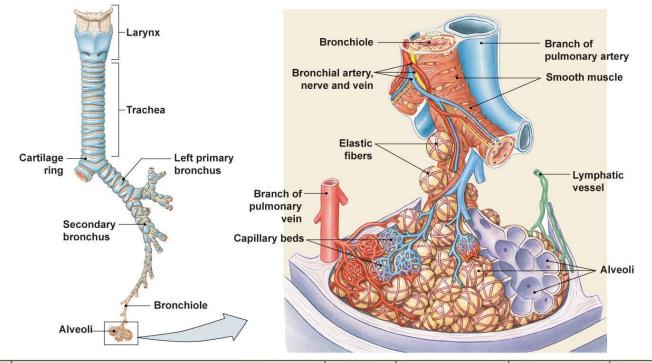
Functional Anatomy

- The Respiratory Tree
 - connecting the external environment to the exchange portion of the lungs...Trachea being generation zero (we may also call it "branch" or "division")...we have 23 generations/branches/divisions
 - similar to the vascular component
 - larger airway = high velocity
 - small cross-sectional area
 - smaller airway = low velocity
 - large cross-sectional area

Functional Anatomy

- The Respiratory Tree
 - Upper respiratory tract is for all intensive purposes a single large conductive tube
 - The lower respiratory tract starts after the larynx and divides again and again...and again to eventually get to the smallest regions which form the exchange membranes
 - Trachea
 - Primary bronchi
 - Secondary bronchi
 - Tertiary bronchi
 - Bronchioles
 - Terminal bronchioles
 - Respiratory bronchioles with start of alveoli outpouches
 - Alveolar ducts with outpouchings of alveoli

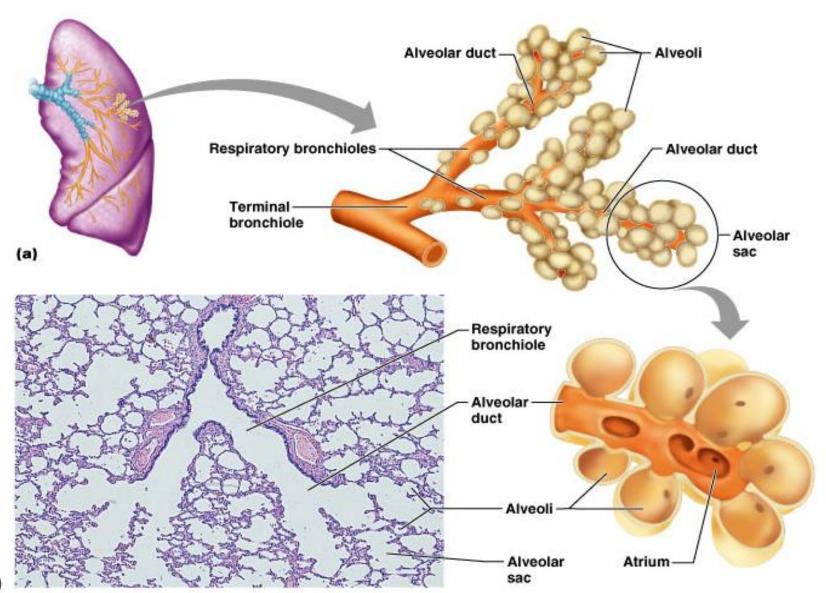
conductive portion...first 16 branches



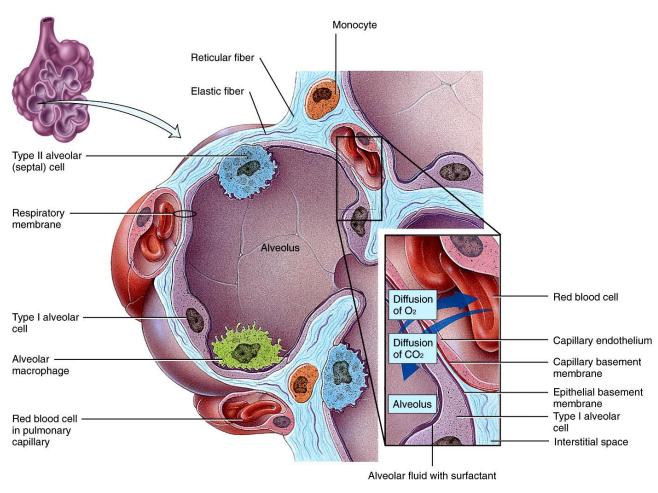
	Name	Division	Diameter (mm)	How many?	Cross-sectional area (cm ²)
Conducting system	Trachea	0	15-22	1	2.5
	Primary bronchi	1	10-15	2	
	Smaller bronchi	2	1-10	4	
	Di Di Cili	3			
		4			
		5			
		6-11		1 x 10 ⁴	↓
Exchange surface	Bronchioles	12-23	0.5-1	2 x 10 ⁴	100 ↓ 5 x 10 ³
	Alveoli	24	0.3	3-6 x 10 ⁸	>1 x 10 ⁶

Cartilage and protection

- The first 10 generations have cartilage and thus have support and therefore are somehow not collapsible structures
- 12th to 16th are called bronchioles (diameter < 1 mm) lack cartilage....and thus are collapsible
- From 0-16 is the conductive zone...ADS (2 ml/kg BW)
- From 17-23 is the respiratory zone...
- Some times 17th -19th are called Transitional zone
- 20th to 22nd are called alveolar ducts (0.5 mm in diameter) and are completely lined with alveoli
- Alveoli can intercommunicate through the pores of Kohn



Components of Alveolus

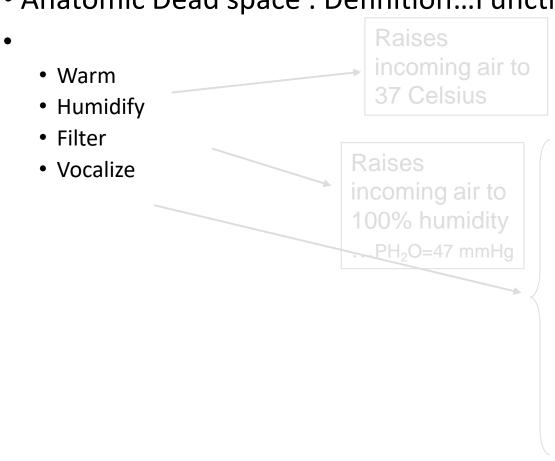


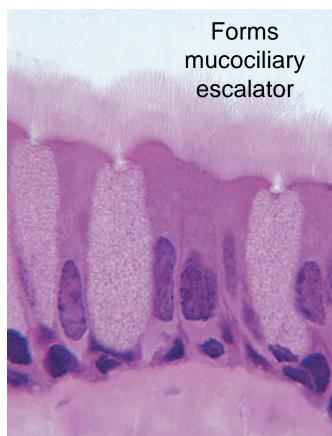
(a) Section through an alveolus showing its cellular components

Figure 23.11ab Tortora - PAP 12/e Copyright © John Wiley and Sons, Inc. All rights reserved. (b) Details of respiratory membrane

Functional Anatomy

• Anatomic Dead space : Definition...Function





Functional Anatomy

- What is the function of the respiratory zone?
 - Exchange of gases Due to
 - Alveoli have a volume of 5-6 liters. A sphere of this volume has a surface area of 0.16 m². However the alveolar surface area is 50-100 m². (150-times more)
 - Type I alveolar cells (simple squamous
 - Epithelium...flat cells).
 - The surface area of the alveoli available for diffusion is about the size of a tennis court
 - Associated network of pulmonary capillaries
 - 80-90% of the space between alveoli is filled with blood in pulmonary capillary networks
 - Exchange distance is less than 1 μm from alveoli to blood!

Protection

- Free alveolar macrophages (dust cells) <u>Alveolar macrophage</u> is the garbage man of the alveoli and thus clean the alveoli.
- Surfactant produced by type II alveolar cells (septal cells)

Respiratory Physiology Gas Laws

- Basic Atmospheric conditions
 - Pressure is typically measured in mm Hg
 - At sea level, atmospheric pressure is 760 mm Hg
 - Atmospheric components
 - Nitrogen = 78% of our atmosphere PN₂≈ 600 mmHg
 - Oxygen = 21% of our atmosphere $PO_2 \approx 160 \text{ mmHg}$
 - Carbon Dioxide = .033% of our atmosphere, and for practical purposes we will consider PCO₂≈ zero mmHg
 - Water vapor, krypton, argon, Make up the rest...but bcs we consider dry atmospheric air we are going to ignore them
- A few laws to remember
 - Dalton's law...the partial pressure law
 - Fick's Laws of Diffusion...Ohm's law..the most important law in physiology
 - Boyle's Law: volume versus pressure
 - Ideal Gas Law...conversion between units

• Consider PO₂ and PCO₂ in different compartments.

•	Atmospheric		ADS	Α	a	V	<u>E</u>
• PO ₂	160	150	102	102	40	120	
• PCO ₂			40	40	46	27	
• PH ₂ O		47	47	47	47	47	
• <u>PN</u> 2	600	563-	→ 571	571	571	<u>566</u>	
• Total	P 760	760	760	760	<u>704</u>	760	

How to calculate P_AO_{2 A=stands for alveolar}

$$P_AO_2 = P_IO_2 - (PCO_2/R)$$
 _{I=stands for inspired} $PO_2 = 149 - (40/0.8) = 99$

R is respiratory exchange ratio ~0.8

Remember in a normal person alveolar $P_AO_2\approx a~P_aO_2$...the difference is less than 5 mmHg for

reasons to be discussed later (V/Q ratio)

Also
$$P_ACO_2 = P_aCO_2$$
.

Gas Laws

Dalton's Law

- Law of Partial Pressures
 - "In a mixture of gases, each gas will exert a pressure independent of other gases present"
 - In a mixture of gases each gas behaves as if it is the only gas available in that mixture

Or

- The total pressure of a mixture of gases is equal to the sum of the individual gas pressures.
- What does this mean in practical application?
 - If we know the total atmospheric pressure (760 mm Hg) and the relative abundances of gases (% of gases)
 - We can calculate individual gas effects!
 - P_{atm} x % of gas in atmosphere = Partial pressure of any atmospheric gas
 - $PO_2 = 760$ mmHg x 21% (.21) = **160** mm Hg
 - Now that we know the partial pressures we know the gradients that will drive diffusion!

Again: Dalton's Law

In a gas mixture the pressure exerted by each individual gas in a space is independent of the pressure exerted by other gases.

P_{atm}=PH₂O+PO₂+PN₂ Pgas=% total gases * Ptotal

- Fick's Laws of Diffusion
 - Things that affect rates of diffusion of gases
 - Distance to diffuse...thickness of the respiratory membrane
 - ΔP for that gas
 - Diffusing molecule sizes ...least important
 - Temperature...usually it is stable 37C
 - In healthy individuals, most of the above variables are constant with the exception ΔP
 - So it all comes down to partial pressure gradients of gases... determined by Dalton's Law!

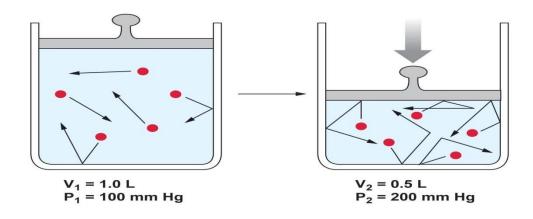
Fick's Law

• Fick's Law defines diffusion of gas

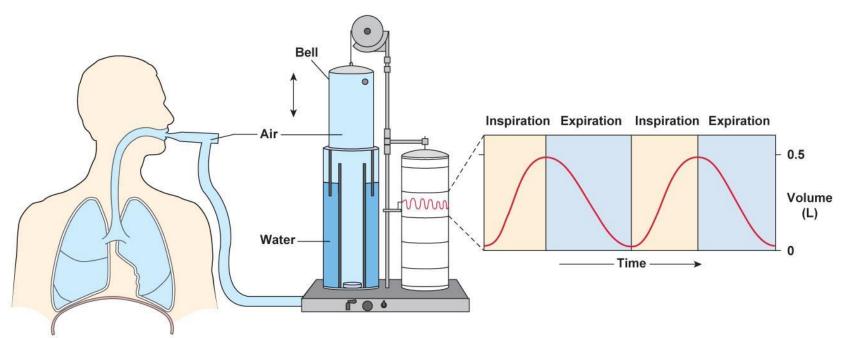
• GAS Diffusion=Area* \(\Delta \) Pressure * Diffusion Coefficient / Distance

- Diffusion Coefficient = Solubility/(Molecular weight)^{1/2}
- MW has small effect bcs it is the square root of MW

- Boyle's Law
 - Describes the relationship between pressure and volume...this law helps you to understand how we breath in and out.
 - "the pressure and volume of a gas in a system are inversely related if the temperature is kept constant
 - $P_1V_1 = P_2V_2$
 - The pressure of a gas inside container is *inversely* proportional to the volume of container



- How does Boyle's Law work in us?
 - As the thoracic cavity (container) expands the volume increases and pressure goes down
 - If it goes below 760 mm Hg what happens?
 - As the thoracic cavity shrinks the volume must go down and pressure goes up
 - If it goes above 760 mm Hg what happens



- Ideal Gas law
 - The pressure and volume of a container of gas is directly related to the temperature of the gas and the number of molecules in the container
 - PV = nRT
 - n = moles of gas
 - T = absolute temp
 - R = universal gas constant @ 8.3145 J/K·mol
- Do we care? It helps you to convert PCO2 (mmHg) to [CO2] in mMole/I later when you consider acid-base disturbance in renal physiology

Respiratory Physiology Gas Laws

Henry and his law

At a constant temperature, the amount of a given gas dissolved in a given type and volume of liquid is directly proportional to the partial pressure of that gas multiplied by the solubility of a gas in a

* Solubility has a constant which is different for each gas

Using this law you can predict how much O_2 and CO_2 are available in dissolved form

Partial Pressures of Gases in Blood

- When a liquid or gas (blood and alveolar air) are at equilibrium:
 - The amount of gas dissolved in fluid reaches a maximum value (Henry's Law).
- Depends upon:
 - Solubility of gas in the fluid.
 - Temperature of the fluid.
 - Partial pressure of the gas.

• .

- Cause of Inspiration?
 - Biological answer
 - Contraction of the inspiratory muscles causes an increase in the thoracic cavity size, thus allowing air to enter the respiratory tract
 - Physics answer
 - As the volume in the thoracic cavity increases (due to inspiratory muscle action) the pressure within the respiratory tract drops below atmospheric pressure, creating a pressure gradient which causes molecular movement to favor moving into the respiratory tract
 - Cause of Expiration? What you think?

- Inspiration is active process. In order to remove any elastic structure from its resting state you need to apply force...!The resting state of the lung is FRC. However, to bring the lung back to its resting state is free...passive....you don't need force! Therefore, expiration is passive process.
 - Air moves from an area of high pressure to an area of low pressure
 - During inspiration diaphragm pulls down and lungs expand
 - When lungs expand, it INCREASES the lung VOLUME, which DECREASES the intrapulmonic PRESSURE
 - Lung pressure is lower than outside pressure, so air moves in...It is the inflated lung which pulls air in, it is not the air which inflates the lung.
- Expiration (passive process) breathing out
 - Diaphragm and muscles relax
 - Volume in lungs and chest cavity decreases, so now pressure inside increases
 - Air moves out because pressure inside is HIGHER than OUTSIDE atmosphere

- During Inspiration intra-alveolar pressure is subatmospheric and during expiration it is above atmospheric. At the end of inspiration or expiration, intra-alveolar pressure equals atmospheric.
- Q <u>During</u> Inspiration in normal individual
- A. Alveolar pressure equals zero mmHg
 - B. Alveolar pressure is more than zero mmHg
 - C. Alveolar pressure is less than zero mmHg
 - D. Intrapleuarl pressure becomes positive
 - E. Intrapleural pressure becomes less negative

Mechanics of Breathing

Airflow is governed by the basic flow equation, which relates flow to driving force (pressure) & airways resistance.

Always remember Ohm's law: Flow is directly proportional to the driving force and inversly proportional to the resistance

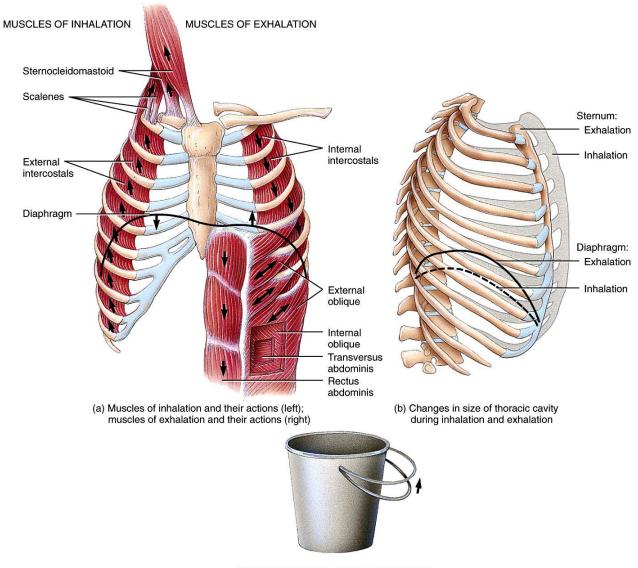
Flow = pressure difference (driving force) / resistance = $\Delta P/R$

- 1. By positive Pressure Breathing: **resuscitator**: P at the nose or mouth is made higher than the alveolar pressure (P_{alv}) . This is artificial type of breathing
- 2. <u>By negative Pressure Breathing:</u> P_{alv} is made less than P_{atm}. **This is normal** pattern of breathing
- It is the pressure difference between the two opposite ends of the airways: $(P_{alv} P_{atm})$
- If R is large then ΔP must be large too to keep flow constant, we recognize the magnitude of airway resistance from the ΔP needed...indirectly.

• ,

Inhalation

- Inhalation is active bcs it involves contraction of:
 - Diaphragm most important muscle of inhalation
 - Flattens, lowering dome when contracted
 - Responsible for 75% of air entering lungs during normal quiet breathing
 - External intercostal muscles (not internal intercostam muscles!)
 - Contraction elevates ribs
 - Responsible 25% of air entering lungs during normal quiet breathing
 - Accessory muscles for deep, forceful inhalation
- When thorax expands...lungs expand too and intra-pulmonic (intra-alveolar) pressure drops... which create a driving force for air flow. Parietal and visceral pleurae adhere tightly



(c) During inhalation, the ribs move upward and outward like the handle on a bucket

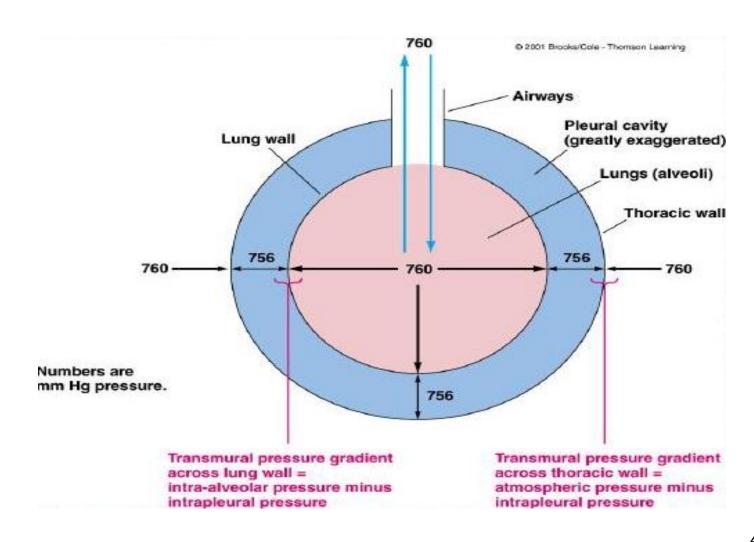
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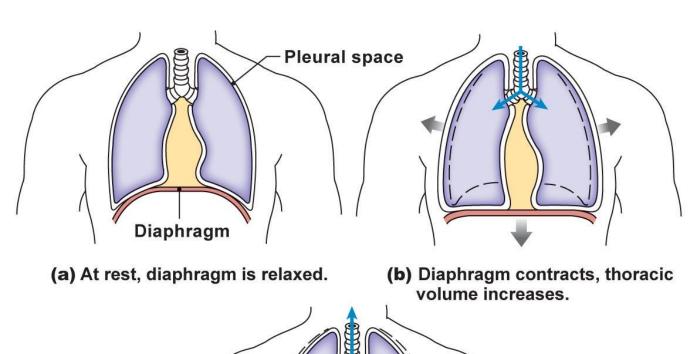
Inspiration

- Occurs as alveolar pressure drops below atmospheric pressure... becomes less than atmospheric and thus we call this pattern as: **negative pressure breathing**
 - For convenience atmospheric pressure = 0 mm Hg
 - A negative value (-) indicates pressure below atmospheric P
 - A positive (+) value indicates pressure above atmospheric P
 - At the start of inspiration (time = 0),
 - atmospheric pressure = alveolar pressure=zero mmH
 - No driving force (Ohm's)...No net movement of gases!
 - At time 0 to 2 seconds
 - Expansion of thoracic cage and corresponding pleural membranes and lung tissue causes alveolar pressure to drop to -1 mm Hg (negative)
 - Now...air enters the lungs down the partial pressure gradient

Respiratory pressures

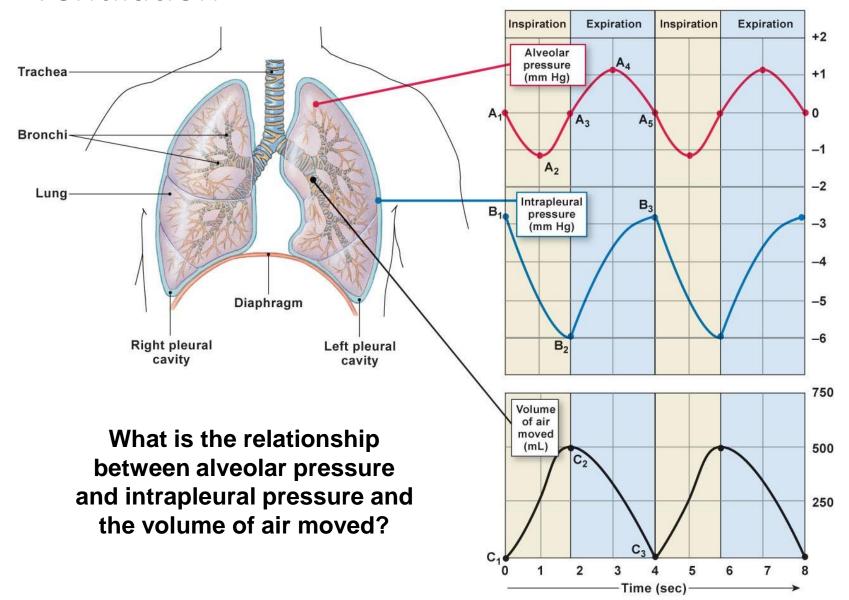


Besides the diaphragm (only creates about 60-75% of the volume change) what are the muscles of inspiration & expiration?



(c) Diaphragm relaxes, thoracic volume decreases.

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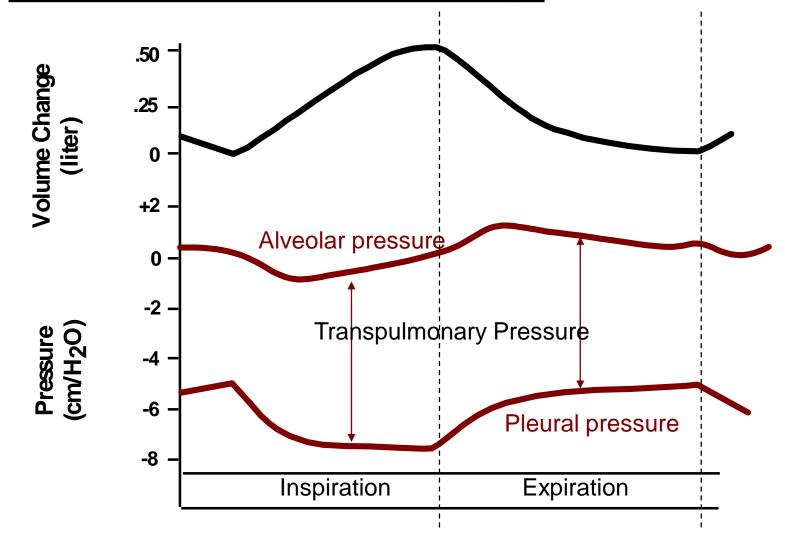


Expiration

- Occurs as alveolar pressure elevates above atmospheric pressure due to a shrinking thoracic cage
 - At time 2-5 seconds
 - Inspiratory muscles relax, elastic tissue of corresponding structures initiates a recoil back to resting state
 - This decreases volume and correspondingly increases alveolar pressure to 1 mm
 Hg
 - This is above atmospheric pressure, causing...?
 - At time 5 seconds
 - Atmospheric pressure once again equals alveolar pressure and there is no net movement

(Intra-alveolar is the same as Intrapulmonic) and (intrapleural is the same as intrathoracic.) We also have transthoracic which is equal to (intrapleural minus intra-alveolar).

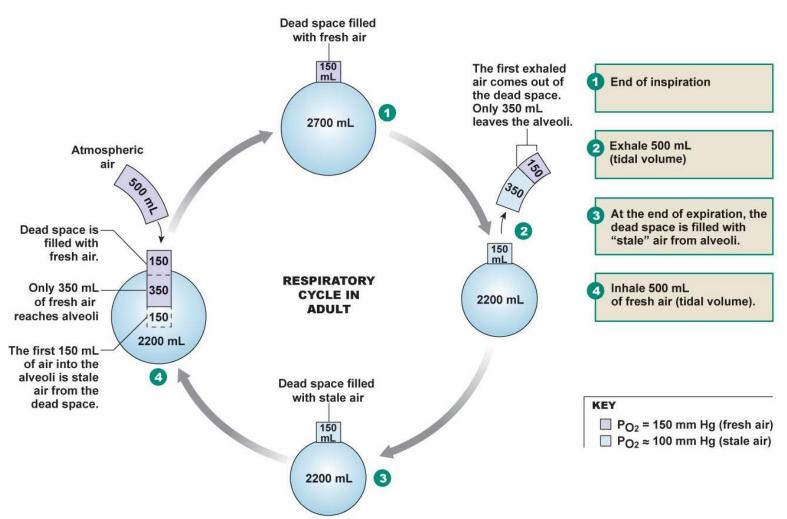
Transmural (intrapleuaral- minus intra-airways)



- What are the different respiratory patterns?
 - Quiet breathing (relaxed)
 - Forced inspirations & expirations
- Respiratory volumes follow these respiratory patterns...
- Definition of HYPERVENTILATION is when alveolar ventilation is more than CO₂ production → decrease PaCO₂
- HYPOVENTILATION is when alveolar ventilation is LESS than CO2 production → increase PaCO2

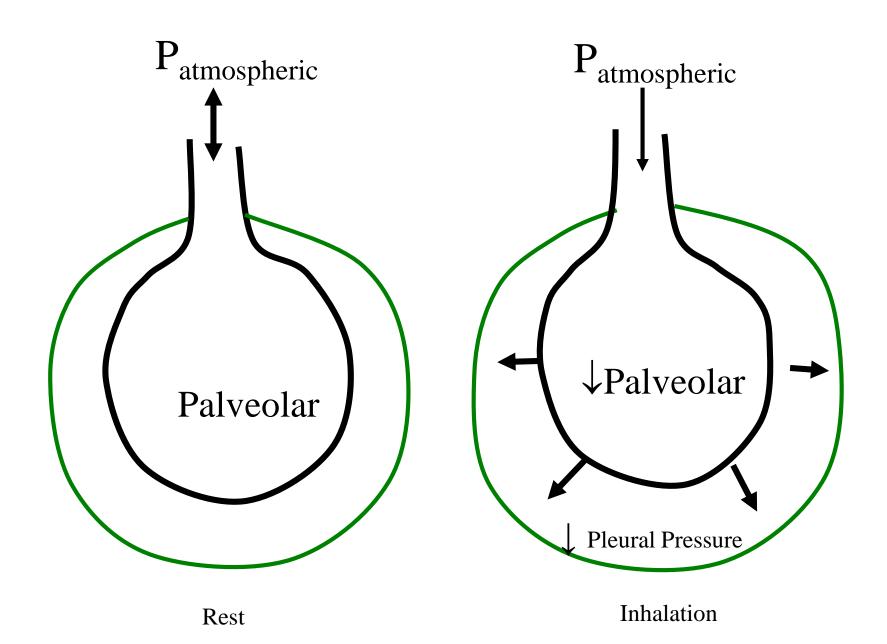
- Both inspiration and expiration can be modified
 - Forced or active inspiration
 - Forced or active expiration
 - The larger and quicker the expansion of the thoracic cavity, the larger the gradient and the faster air moves down its pressure gradient
- It is the PaCO2 we use to determine wither HYPERVENTILATION or HYPOVENTILATION. It is not the PaO2. In exercise when ventilations increased and CO2 production is increased in the same proportion and thus PaCO2 remain NORMAL we don't call this case hyperventilation. At sever exercise when ventilation is more than CO2 production PaCO2 is decreased, this is called hyperventilation.
- Hyperventilation occurs when the rate of alveolar ventilation reduces the arterial PCO₂ below 40 mmHg such as in pregnancy

The relationship between minute volume (total pulmonary ventilation) and alveolar ventilation & the subsequent "mixing" of air



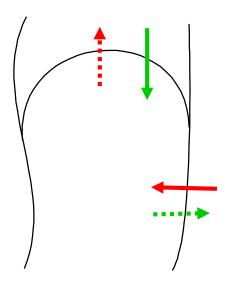
• Transpulmonary Pressure

- The difference between the alveolar pressure and the pleural pressure.
- It is a measure of the elastic forces (recoil tendency) of the lungs.
- called the *recoil pressure*.



Mechanics Of Respiration

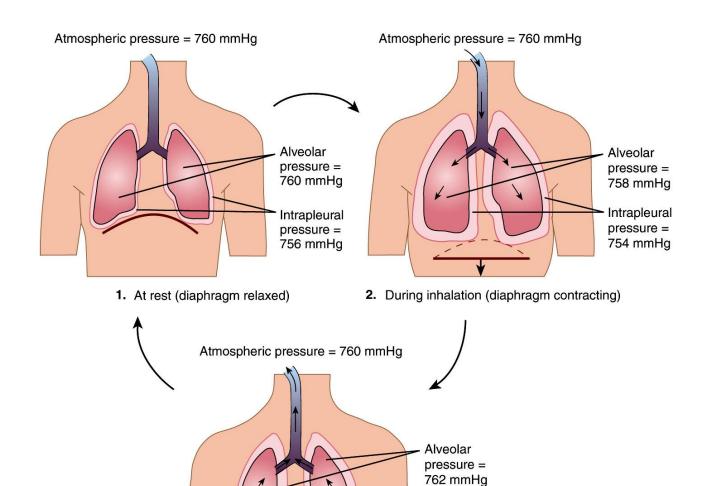
- Expiration
 - Active
 - Abdominals
 - decrease chest volumes



Active exhalation abdominal compressionActive inspiration abdominal relaxation

Exhalation/ expiration

- Pressure in lungs greater than atmospheric pressure
- Normally passive muscle relax instead of contract
 - Based on elastic recoil of chest wall and lungs from elastic fibers and surface tension of alveolar fluid
 - Diaphragm relaxes and become dome shaped
 - External intercostals relax and ribs drop down
- Exhalation only active during forceful breathing



3. During exhalation (diaphragm relaxing)

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Intrapleural pressure = 756 mmHg

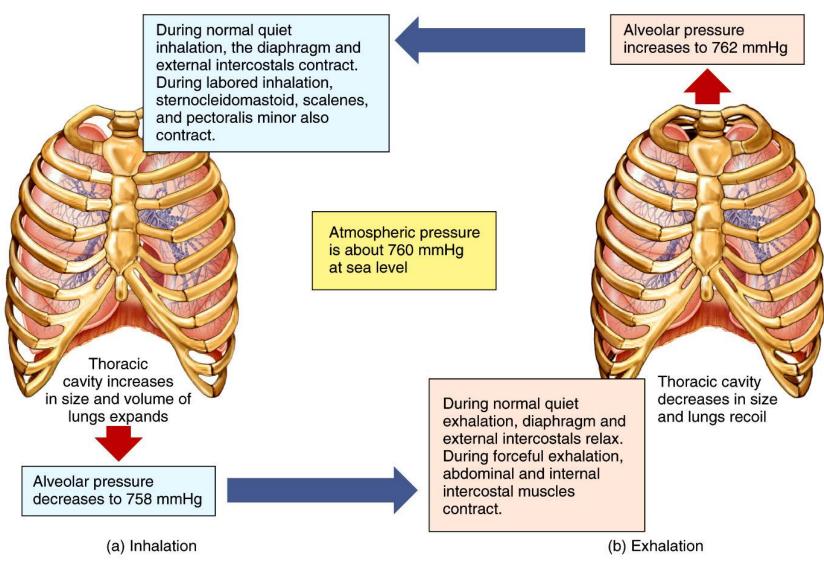


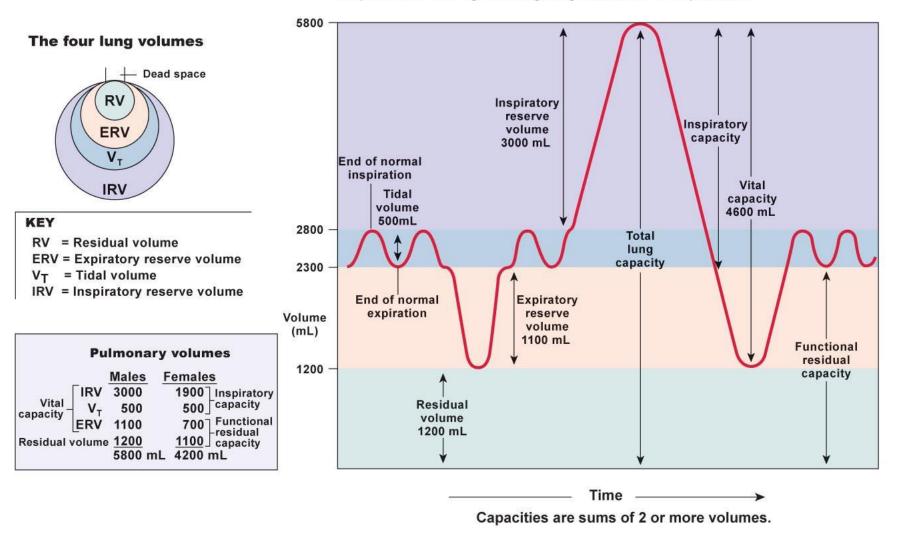
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- Respiratory Minute respiration RMV (respiratory rate times tidal volume 0.5 L * 12=6L/min). if you remember how the cardiac output is claculated then it is easy for you to understand the RMV: Q=HR * SV...it is the same principle
- RMV=RR * V_T
- Anatomical dead space ventilation and alveolar ventilation
- RMV= ADS ventilation + alveolar ventilation

PFT Pulmonary Function Tests

- Lung Volumes and Capacities
- In lecture 1+2 we will discuss lung volumes and capacities. Other tests will be discussed in lecture 3-4. Diffusing Capacity of the Lung for Carbon Monoxide will be discussed with Gas Exchange lecture.
- Maximum voluntary ventilation: = 3 X 50 = 150 1/min.
- It lasts only for just 15-30 second. It is not indicator for the upper limits of the respiratory system.

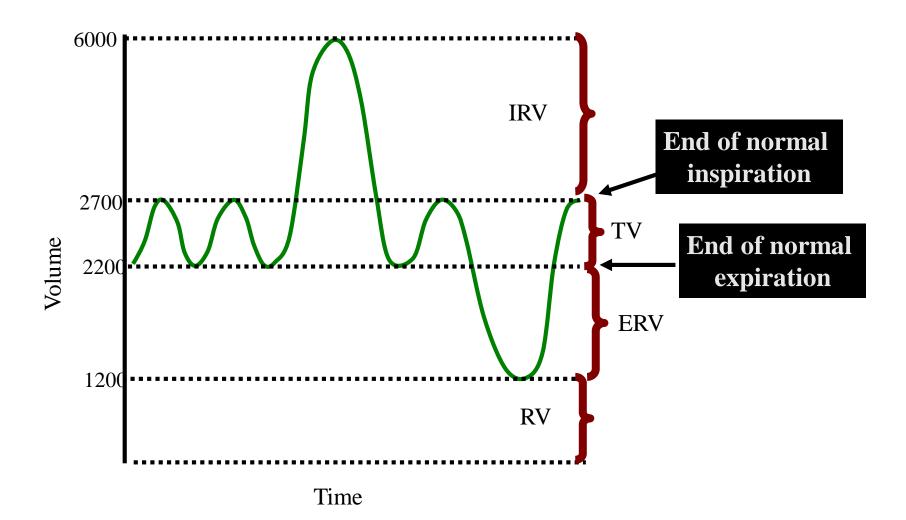
A spirometer tracing showing lung volumes and capacities.

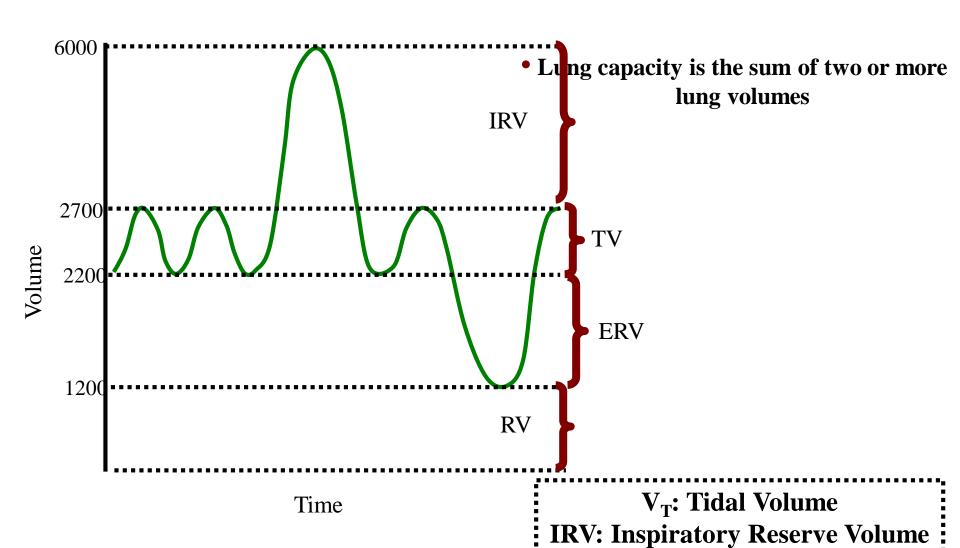


 Residual volume RV can be measured using a non-absorbable tracer gas (Helium) & measuring its dilution when it is mixed with unknown volume of gas initially in the lings.

•
$$C_1 V_1 = C_2 V_2 = C_2 (V_1 + FRC)$$

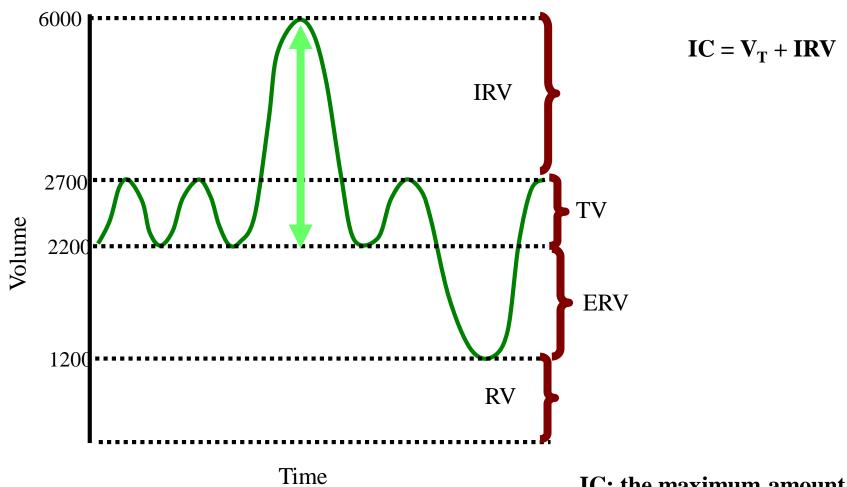
- FRC = $V_1 (C_1 C_2) / C_2$
- C1=Ci_{He} is the initial concentration of the He.
- With helium equilibration, the patient exhales to FRC and then is connected to a closed system containing known volumes of helium and O2. Helium concentration is measured until it is the same on inhalation and exhalation, indicating it has equilibrated with the volume of gas in the lung, which can then be estimated from the change in helium concentration that has occurred.
- This technique and nitrogen washout technique may underestimate FRC because they measure only the lung volume that communicates with the airways. In patients with severe airflow limitation, a considerable volume of trapped gas may communicate very poorly or not at all.



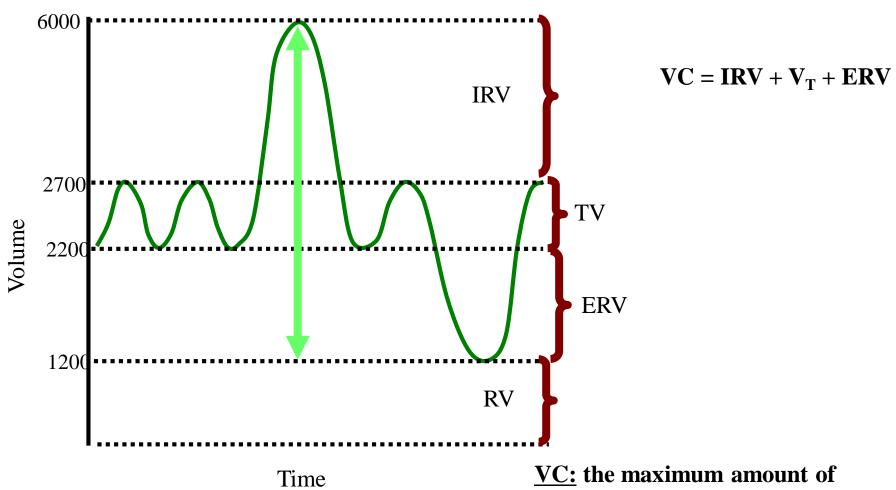


ERV: Expiratory Reserve Volume

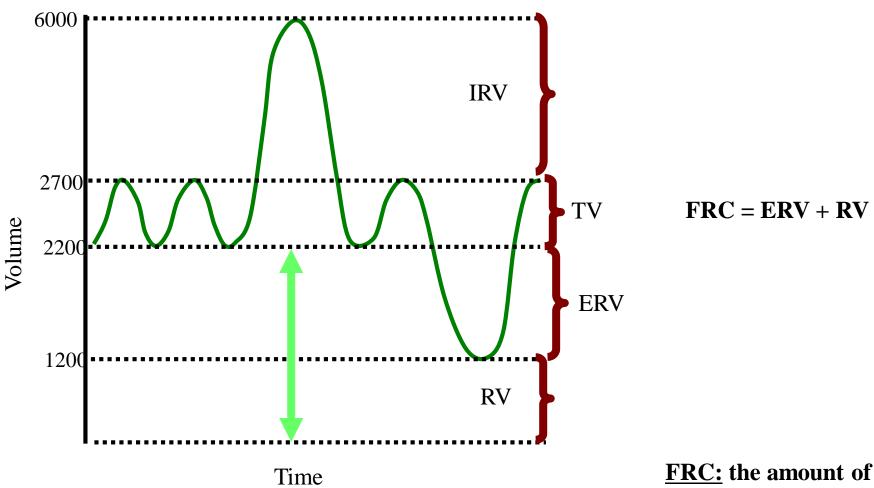
RV: Residual Volume



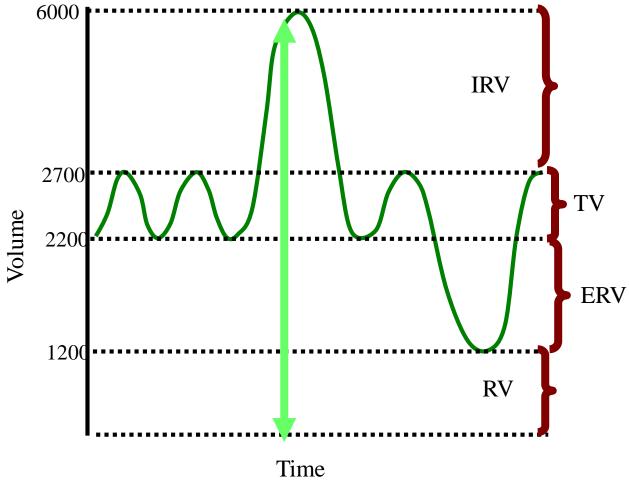
IC: the maximum amount of air that can be inspired following a normal expiration



VC: the maximum amount of air that can be expired following a maximal inspiration



FRC: the amount of air remaining in the lungs following a normal expiration.



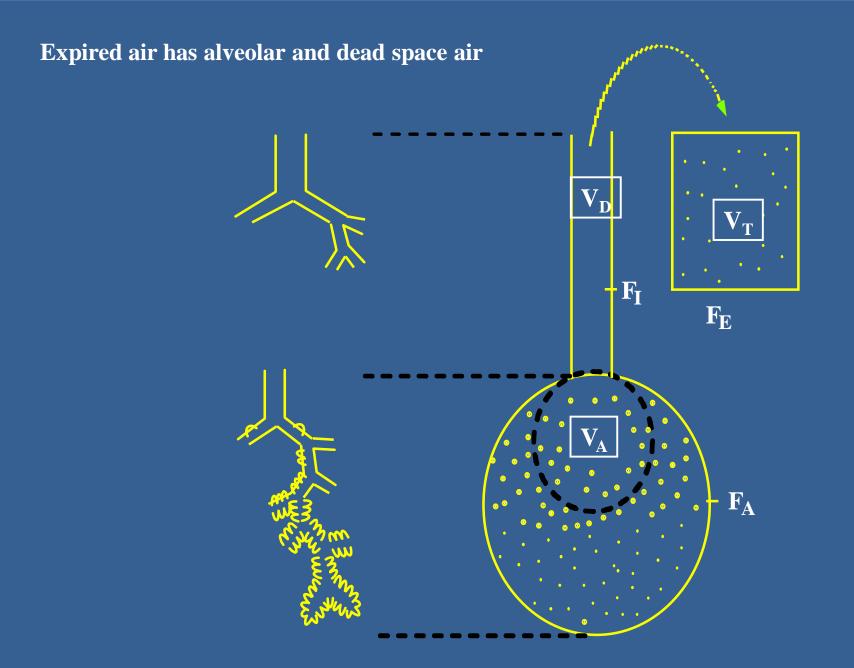
 $TLC = IRV + V_T + ERV + RV$

TLC: the amount of air in the lungs at the end of a maximal inspiration.

• FRC

- 1) Composes 40% of TLC at any chest size.
- 2) Minimum gas available for exchange at all time.
- 3) Keep alveoli open following expiration, thus ↓ work of breathing during inspiration.
- 4) Makes PO₂ fluctuations less at either expiration or inspiration.
- 5) Dilutes inhaled noxious gases.
- 6) \downarrow by any disease which increase the elastic recoil of the lung, such as in lung fibrosis.
- 7) Measured by dilution method or by body plethysmograph.
- C1V1=C2V2=C2(V1+FRC)...lecture 4
- P (FRC) = P' (FRC + V1).
- Body plethysmograph it is a closed box where P: is the pressure in the upper airways.
- This instrument measure any change in V or P.
- OR BY Helium Dilution method.
- In young adult the ERV is more than RV and in older age is the opposite. The reason behind that is because lung looses elastic recoil and the stiffening of the chest wall with age.

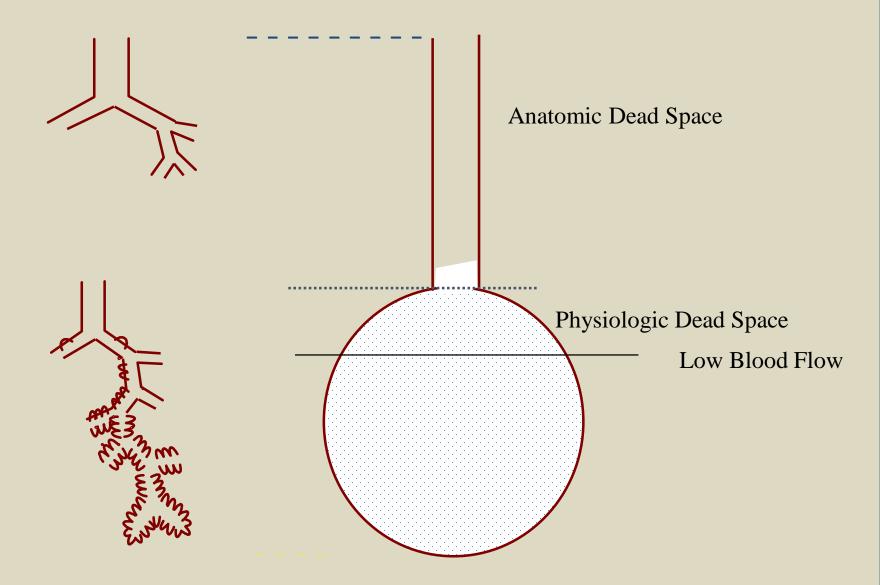
- Minute ventilation or RMV: Total amount of air moved into and out of respiratory system per minute
- Respiratory rate or frequency RR: Number of breaths taken per minute
- Anatomic dead space: Part of respiratory system where gas exchange does not take place ≈ 150 ml in an adult (2 ml/kg)
 - Physiological dead space=ADS + alveolar wasted volume
- Alveolar ventilation: How much air per minute enters the parts of the respiratory system in which gas exchange takes place
- With the lung volume and capacities you should also know and understand the **minimal volume**: which is the resting volume of the lung.



- ANATOMICAL: Anatomical designace is the "clume of air that does not participate in gas exchange
 - 150 ml (2 ml/Kg body weight)
- PHYSIOLOGICAL
 - Depends on ventilation-perfusion ratio
 - Physiologic Dead Space = Anatomic Dead Space + alveolar dead space
- Alveolar dead Space
- Sometimes, some alveoli are ventilated but not perfused (High V/Q ratio)...we will discuss V/Q ratio later
- air in these alveoli doesn't exchange with blood and is part of the alveolar wasted volume or alveolar dead space
- "Alveolar dead space is the volume of air in non-functioning alveoli"
- Physiologic dead space=Anatomical DS + non functioning alveolar dead space

This equation will be explained in the lecturure

$$V_D = VT \left[\frac{PaCO_2 - PECO_2}{PaCO_2} \right]$$



Next Time...

• Airway Resistance Lecture 3-4