# Lung Compliance

- The ease with which lungs can be expanded
- Specifically, the measure of the change in lung volume that occurs with a given change in transpulmonary pressure
- Determined by two main factors
  - Distensibility of the lung tissue and surrounding thoracic cage
  - Surface tension of the alveoli

### Compliance

#### Lung compliance

= change in lung volume change in TP pressure

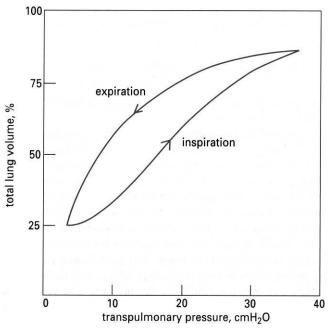


 Figure illustrates the change in lung volume that incurs as pleural pressure changes under normal resting breathing conditions

# Compliance

- A. Compliance of the lungs and the thoraic wall Visco-elastic structures
- 0.13 litre/cm H<sub>2</sub>0
- B. Compliance of lungs only
- 0.22 litre/cm H<sub>2</sub>0
- 1. Presence of large number of elastic fibers
- 2.Peculair arrangement of these elastic fibers Knitting arrangement of its fibers.

# Regional Lung Volume

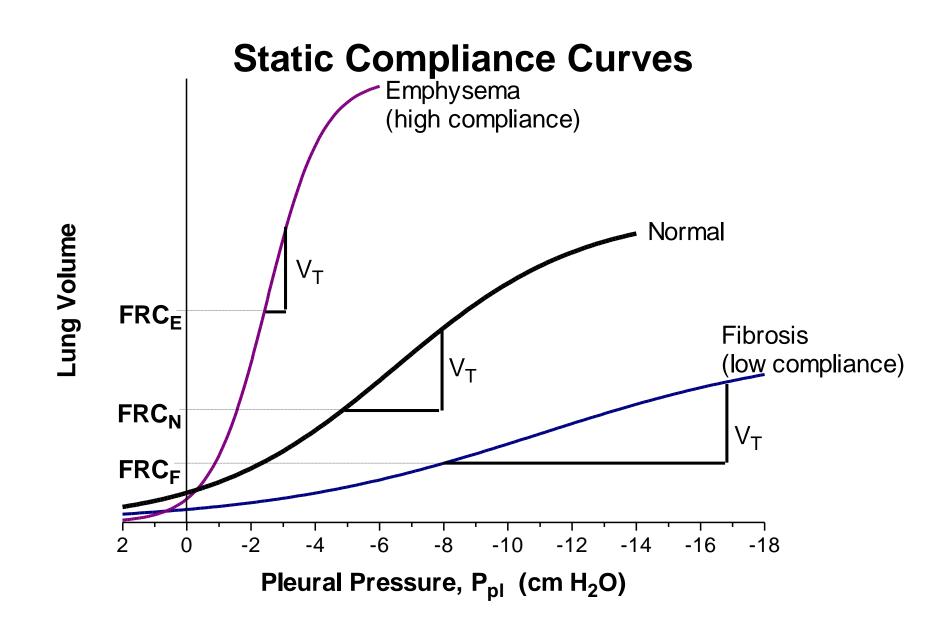
- In the upright posture:
  - Relative lung volume is greater at the apex
  - Lung is less compliant (stiffer) at the <u>apex</u>

# Factors That Diminish Lung Compliance

- Scar tissue or fibrosis that reduces the natural resilience of the lungs
- Blockage of the smaller respiratory passages with mucus or fluid
- Reduced production of surfactant
- Decreased flexibility of the thoracic cage or its decreased ability to expand

# Factors That Diminish Lung Compliance

- Examples include:
  - Deformities of thorax
  - Ossification of the costal cartilage
  - Paralysis of intercostal muscles



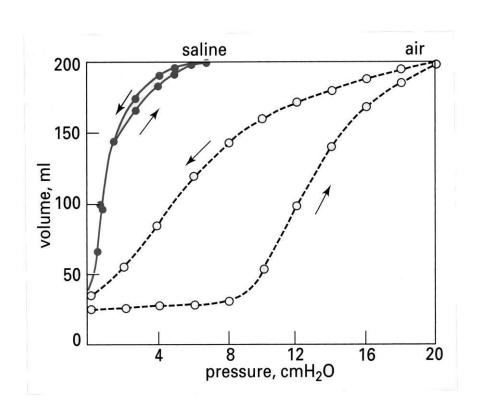
#### Pressure –Volume Relationship

During the beginning of inspiration, there is a small change in volume for a large pressure change. At this time the slope (V/P) is small and thus compliance small.

As inspiration continues the slope is steeper indicating an increase in compliance.

The V/P relation during expiration is different than during inspiration. This is known as hysteresis. Most of the hysteresis and also most of the compliance of the lung appears due to the surface tension on the inner wall of the alveoli

#### How surface tension affects the alveoli



- Inflation of the lung with saline eliminates air-liquid interface and surface tension.
- It is much easier to inflate the lung with saline as the elastic recoil of the lung is diminished due to lack of surface tension.
- Also not much difference between inflation and deflation –no hysteresis

## Work done during breathing

- To move the air into the lungs ,the muscles of respiration have to do work overcome all the different forms of resistance.
- A. In stretching the elastic tissue of the lungs and the chest wall, called Elastic resistance
- B. In moving viscous material of lung tissues
- C. Airway resistance

### Airway Resistance

- Friction is the major nonelastic source of resistance to airflow
- The relationship between flow (F), pressure (P), and resistance (R) is:

$$F = \frac{\Delta P}{R}$$

## Airway Resistance

- The amount of gas flowing into and out of the alveoli is directly proportional to ∆P, the pressure gradient between the atmosphere and the alveoli
- Gas flow is inversely proportional to resistance with the greatest resistance being in the medium-sized bronchi

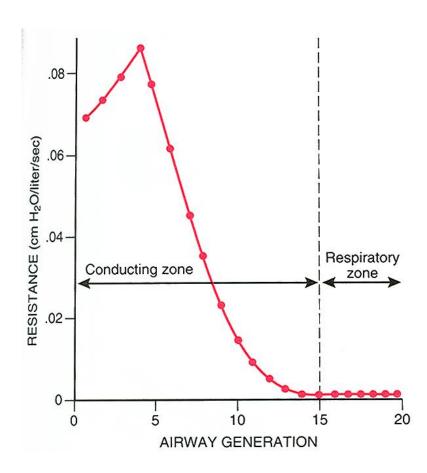
Resistance is defined by Poiseuille's law (laminar flow)

$$R = \frac{8}{\pi} \bullet \frac{\eta l}{r^4}$$

$$\dot{V} = \frac{\Delta P}{R}$$

#### Airway R as a function of airway generation

- The intermediate sized bronchi contribute most of the resistance.
- •In a healthy individual, relatively little resistance is located in the small airways.



## Airway Resistance

- As airway resistance rises, breathing movements become more strenuous
- Severely constricted or obstructed bronchioles:
  - Can prevent life-sustaining ventilation
  - Can occur during acute asthma attacks which stops ventilation

<b>TABLE 17-3</b>	Factors That Affe	ect Airway Resistance
FACTOR	AFFECTED BY	MEDIATED BY
Length of the system	Constant; not a factor	
Viscosity of air	Usually constant; humidity and altitude may alter slightly	
Diameter of airways		
Upper airways	Physical obstruction	Mucus and other factors
Bronchioles	Bronchocon- striction	Parasympathetic neurons (muscarinic receptors), histamine, leukotrienes
	Bronchodilation	Carbon dioxide, epinephrine (β <sub>2</sub> -receptors)

#### Other factors regulating airway resistance

#### 1. Autonomic nervous system

- a. Parasympathetic vagus nerve releases acetylcholine which acts on muscarinic receptors in the bronchial SMCs to cause constriction thus increasing resistance
- b. sympathetic control- norepinephrine dilates bronchi and bronchioles, decreasing airway resistance

#### 2. Humoral factors

- a. epinephrine- released from adrenal medulla, potent bronchodilator
- b. histamine- constricts alveolar ducts and increases airway resistance

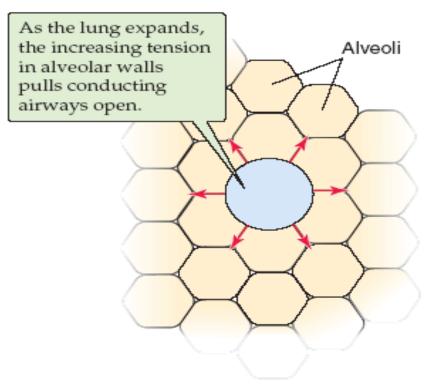
#### Other factors regulating airway resistance

Mechanical tethering- principle of interdependence

airways tend to hold their neighbors open

during inspiration the inflation of alveoli (higher compliance) will pull open conducting airways (less compliant) by radial traction

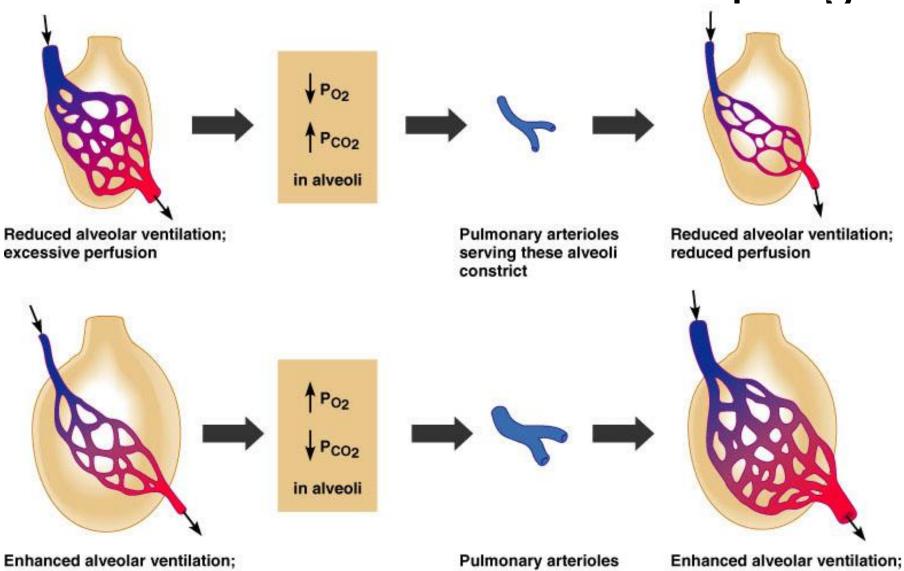
#### **B** MECHANICAL TETHERING



# Ventilation-Perfusion Coupling

- Ventilation the amount of gas reaching the alveoli
- Perfusion the blood flow reaching the alveoli
- Ventilation and perfusion must be tightly regulated for efficient gas exchange
- Changes in P<sub>CO2</sub> in the alveoli cause changes in the diameters of the bronchioles
  - Passageways servicing areas where alveolar carbon dioxide is high dilate
  - Those serving areas where alveolar carbon dioxide is low constrict

### Ventilation-Perfusion Coupling



inadequate perfusion

serving these alveoli dilate

enhanced perfusion

#### Ventilation

Total pulmonary ventilation and alveolar ventilation

Total pulmonary ventilation = ventilation rate × tidal volume

#### **Dead Space**

Anatomical dead space – volume of the conducting respiratory passages (150 ml)
Alveolar dead space – alveoli that cease to act in gas exchange due to collapse or obstruction
Total dead space – sum of alveolar and anatomical dead spaces

#### **Alveolar Ventilation**

 Alveolar ventilation rate (AVR) – measures the flow of fresh gases into and out of the alveoli during a particular time

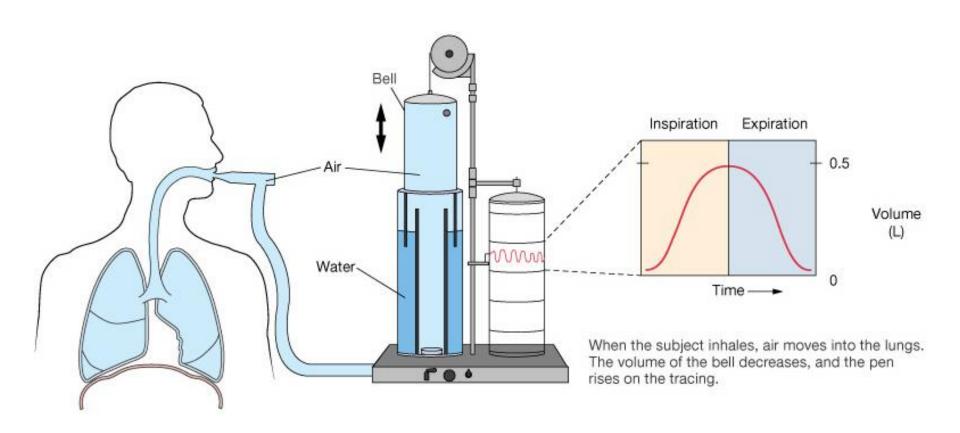
AVR	=	frequency	Х	(TV – dead space)
(ml/min)		(breaths/min)		(ml/breath)

 Slow, deep breathing increases AVR and rapid, shallow breathing decreases AVR

#### **TABLE 17-6** Normal Ventilation Values in Pulmonary Medicine

Total pulmonary ventilation	6 L/min
Total alveolar ventilation	4.2 L/min
Maximum voluntary ventilation	125–170 L/min
Respiration rate	12–20 breaths/min

# Lung Volumes: Spirometer Measurements



# Respiratory Volumes

- During normal quiet breathing, about 500mL moves in and out of the lungs w/ each breath.
  - This is the tidal volume
- The amount of air that can be inspired beyond the TV is the inspiratory reserve volume.
- Expiratory reserve volume is the amount of air that can be expired after a tidal expiration.
- After the most strenuous expiration, about 1200mL of air remains in the lungs – the residual volume (helps prevent atalectasis).

# **Respiratory Capacities**

 Inspiratory capacity is the total amt of air that can be inspired after a tidal expiration:

$$IC = TV + IRV$$

 Functional residual capacity is the amt of air in the lungs after a tidal expiration:

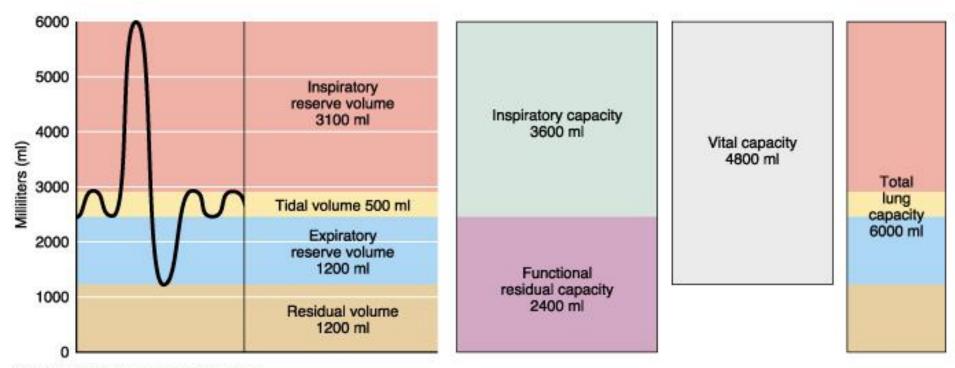
$$FRC = ERV + RV$$

Vital capacity is the total amt of exchangeable air:

$$VC = TV + IRV + ERV$$

 Total lung capacity is the sum of all lung volumes and is normally around 6L in males:

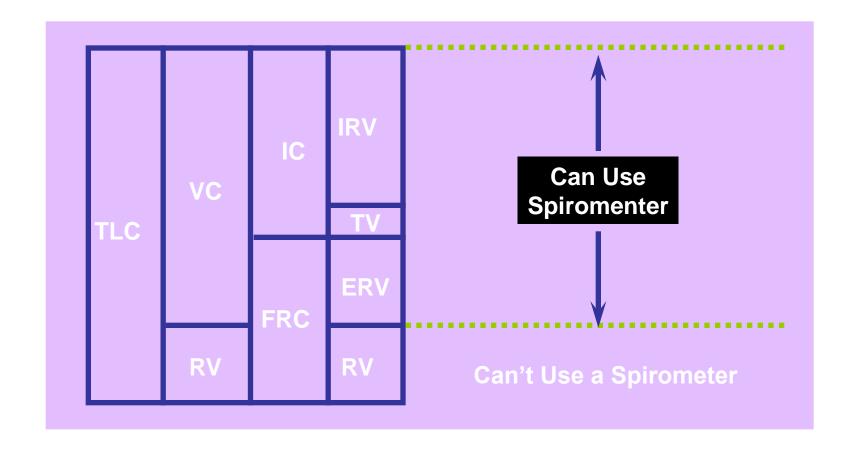
$$TLC = VC + RV$$



(a) Spirographic record for a male

# Measuring vital capacity and its subcomponents.

Use a spirometer.



### Measuring Residual Volume

- Can't use a Spirometer
- Use instead:
  - Nitrogen Washout
  - Helium Dilution Method

## **Pulmonary Function Tests**

- Forced Vital Capacity (FVC) measures the amt of gas expelled when one takes a deep breath and then forcefully exhales maximally and rapidly.
- Forced Expiratory Volume (FEV) determines the amt of air expelled during specific time intervals of the FVC test.
  - For example, the volume exhaled during the 1<sup>st</sup> second is the FEV<sub>1</sub>. People w/ healthy lungs can exhale about 80% of the FVC in the 1<sup>st</sup> second.

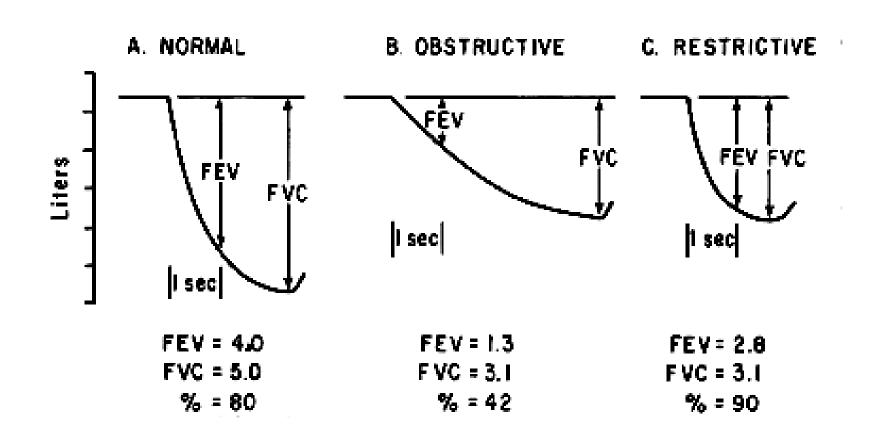
#### Restrictive Disease

- Difficult to get air in to the lungs
- "Restrict" inspiration
- Examples:
  - intersitial fibrosis
  - muscular diseases
  - chestwall deformities.

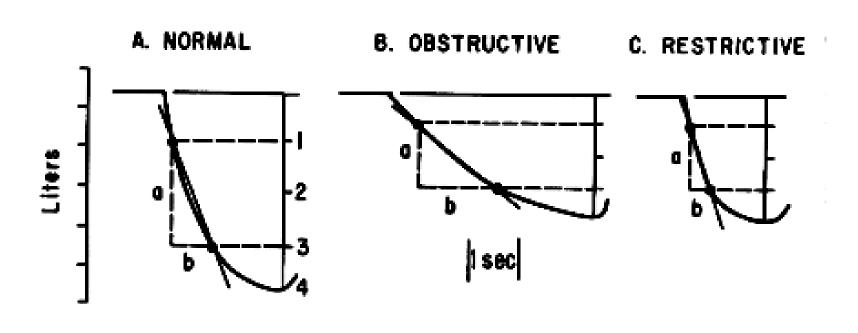
# Lung Capacity and Disease— Summary

- Obstructive Disease:
  - Decreased VC
  - Increased TLC, RV, FRC.
- Restrictive Disease:
  - Decreased VC
  - Decreased TLC, RV, FRC.

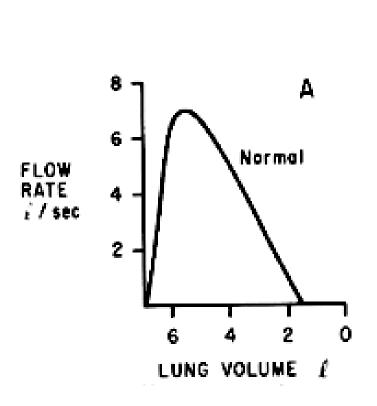
# Forced Vital Capacity FEV<sub>1.0</sub> / FVC Ratio

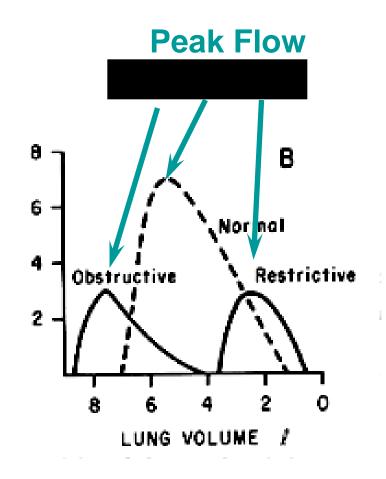


# Small Airways Disease FEF<sub>25-75</sub>



#### Flow -Volume Curves





# Pulmonary Function Summary

	Obstructive Disease	Restrictive Disease
FEV <sub>1.0</sub>	Decreased	Decreased
FVC	Decreased	Decreased
FEV <sub>1.0</sub> /FVC	Decreased	Unchanged or Increased
<b>Peak Flow</b>	Decreased	Decreased or Unchanged
RV/TLC	Increased	Unchanged