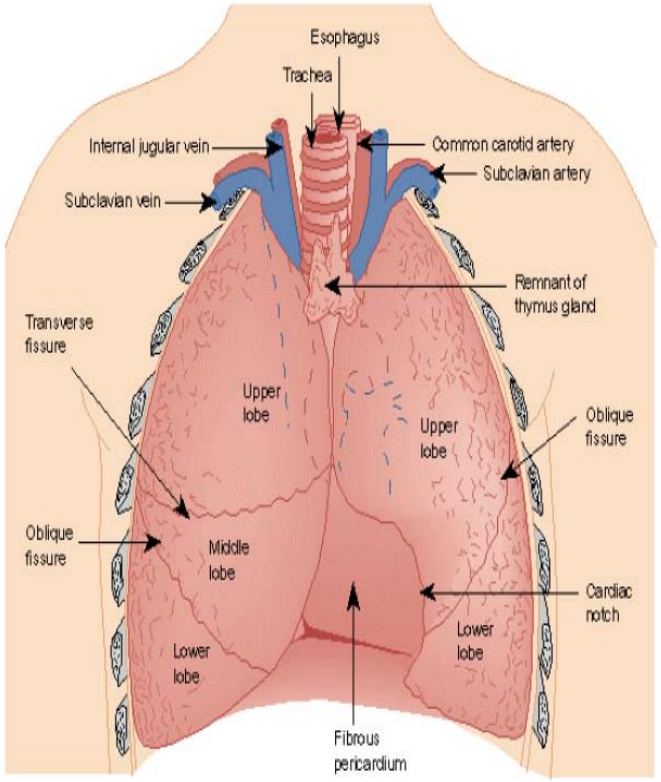
STRUCTURE AND FUNCTION OF RESPIRATORY TRACT IN RELATION TO ANAESTHESIA

CONTENTS

- Lung functions
- Respiratory control system
- Receptors in respiratory tract
- Respiratory tract reflexes
- Factors affecting respiration
- Static characteristics of the lungs
- Ventilation and perfusion
- Lung volumes and capacities
- Respiratory function during anaesthesia



LUNG FUNCTIONS

- Provides large surface area for gas exchange
- Moves air to and from the gas-exchange surfaces of lungs
- Produces sounds permitting speech
- Provides olfactory sensations to the CNS for sense of smell
- Reservoir of blood available for circulatory compensation
- Filter for circulation:
  - Thrombi, Microaggregates, etc.

LUNG FUNCTIONS (Contd.)

- Regulation of blood pH
- Protects:
  - Respiratory surfaces from dehydration and temp changes
  - Provides nonspecific defenses against invading pathogens
    - Secretory immunoglobulin's (IgA)
    - Collectins (including Surfactant A and D)
    - Defensins
    - Peptides and Proteases
    - Reactive oxygen species
    - Activated epithelium release PGE 2 that protects epithelium
    - Alveolar Macrophages - Chemotactic

      Antigen Processing

      Formation Of Granulocytes + Monocytes

LUNG FUNCTIONS (Contd.)

Metabolic and endocrine functions of the lungs

- ***Biologically active Substances handled In Pulmonary Vascular Bed***

Unaffected by lungs:
- Epinephrine
- Prostaglandin A
- Angiotensin II
- Vasopressin

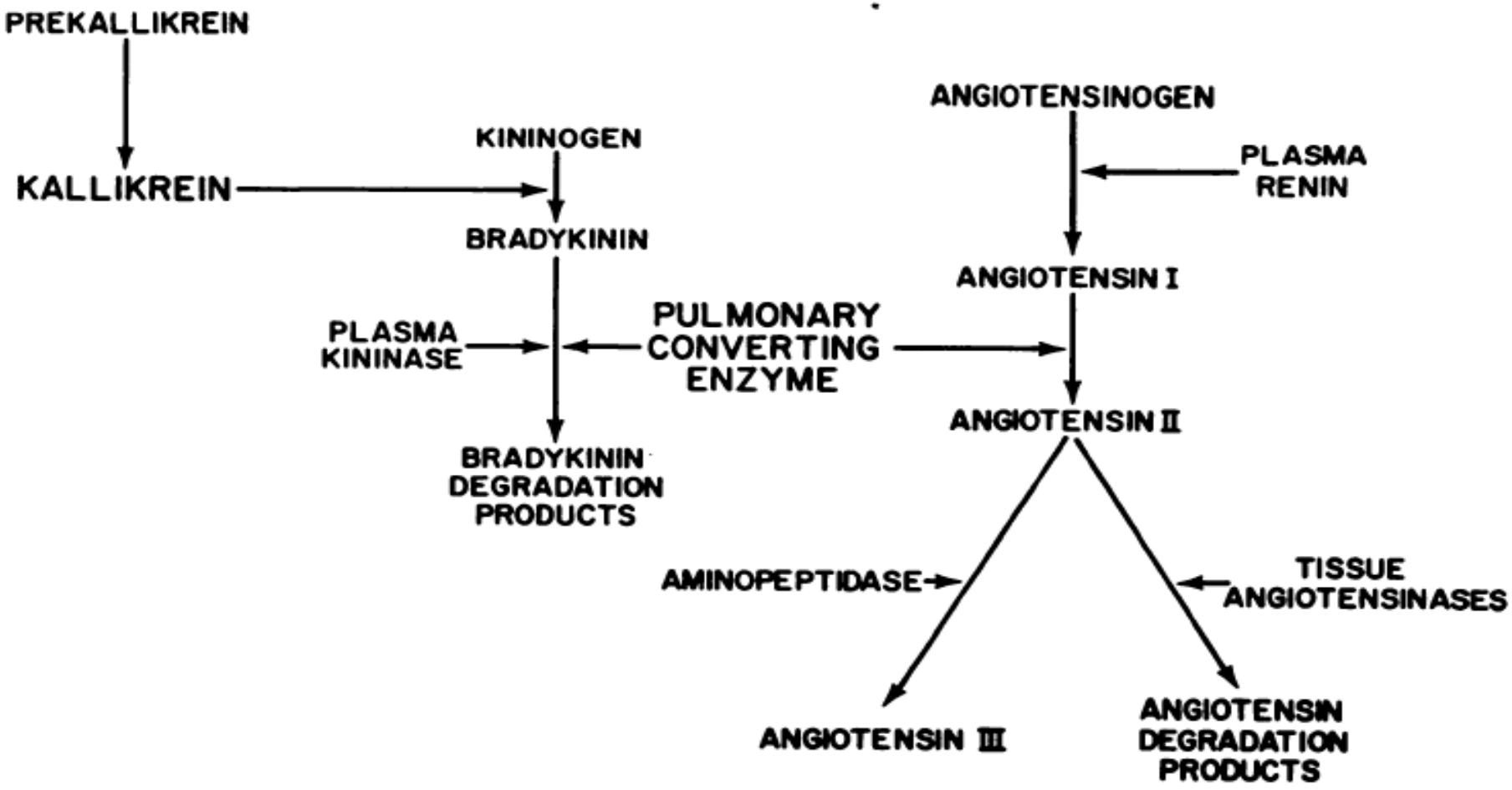
Cleared by lungs:
- Bradykinin
- Adenine nucleotides
- Norepinephrine
- Serotonin
- Prostaglandins E and F

Activated by lungs:
- Angiotensin I
- Cyclic endoperoxides

Released by lungs:
- Prostaglandins
- Histamine
- Slow-reacting substance of anaphylaxis
- Kallikreins

LUNG FUNCTIONS (Contd.)

Diagram of the two protease pathways that share angiotensin converting enzyme.

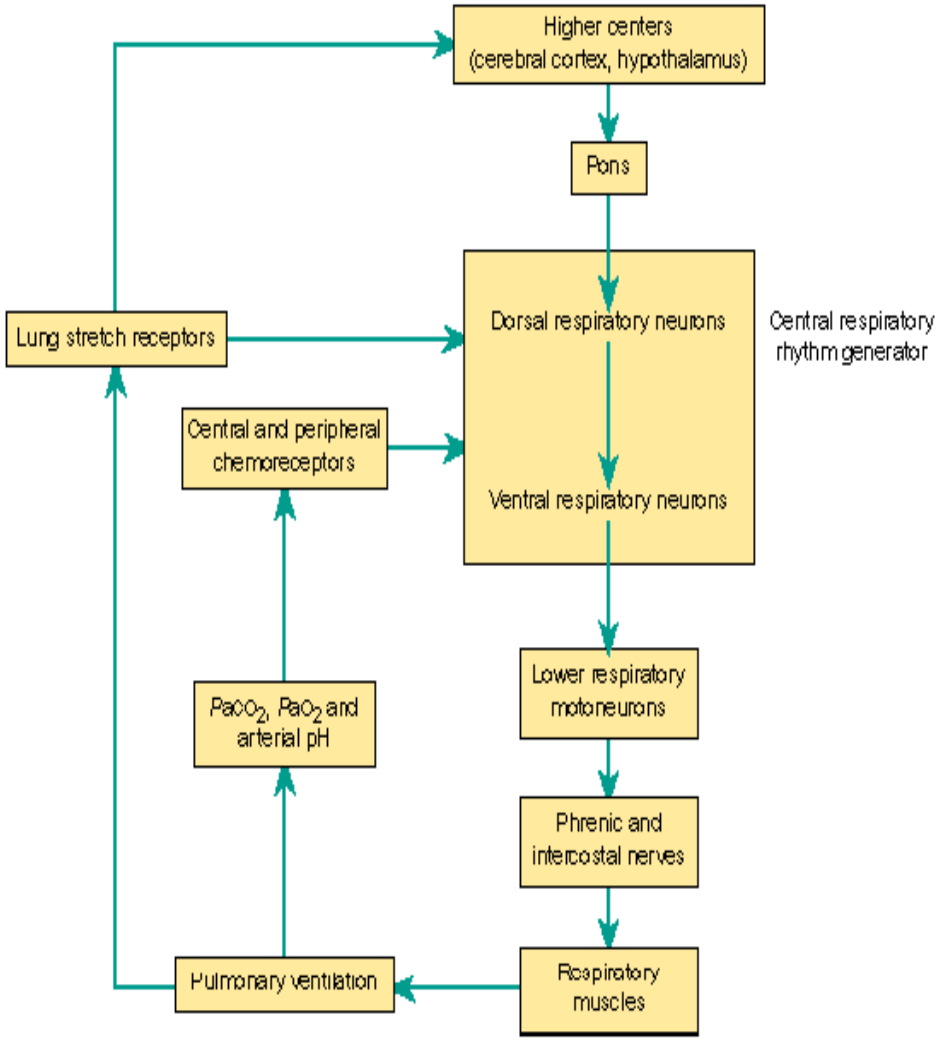


LUNG FUNCTIONS (Contd.)

- Synthesis of Phospholipids(Surfactant)
- Protein Synthesis
- Elaboration of Mucopolysaccharides of Bronchial mucus

CONTROL OF RESPIRATION

- Basic elements of the respiratory control system are
  - Central controller
  - Strategically placed sensors
  - Respiratory muscles



CONTROL OF RESPIRATION

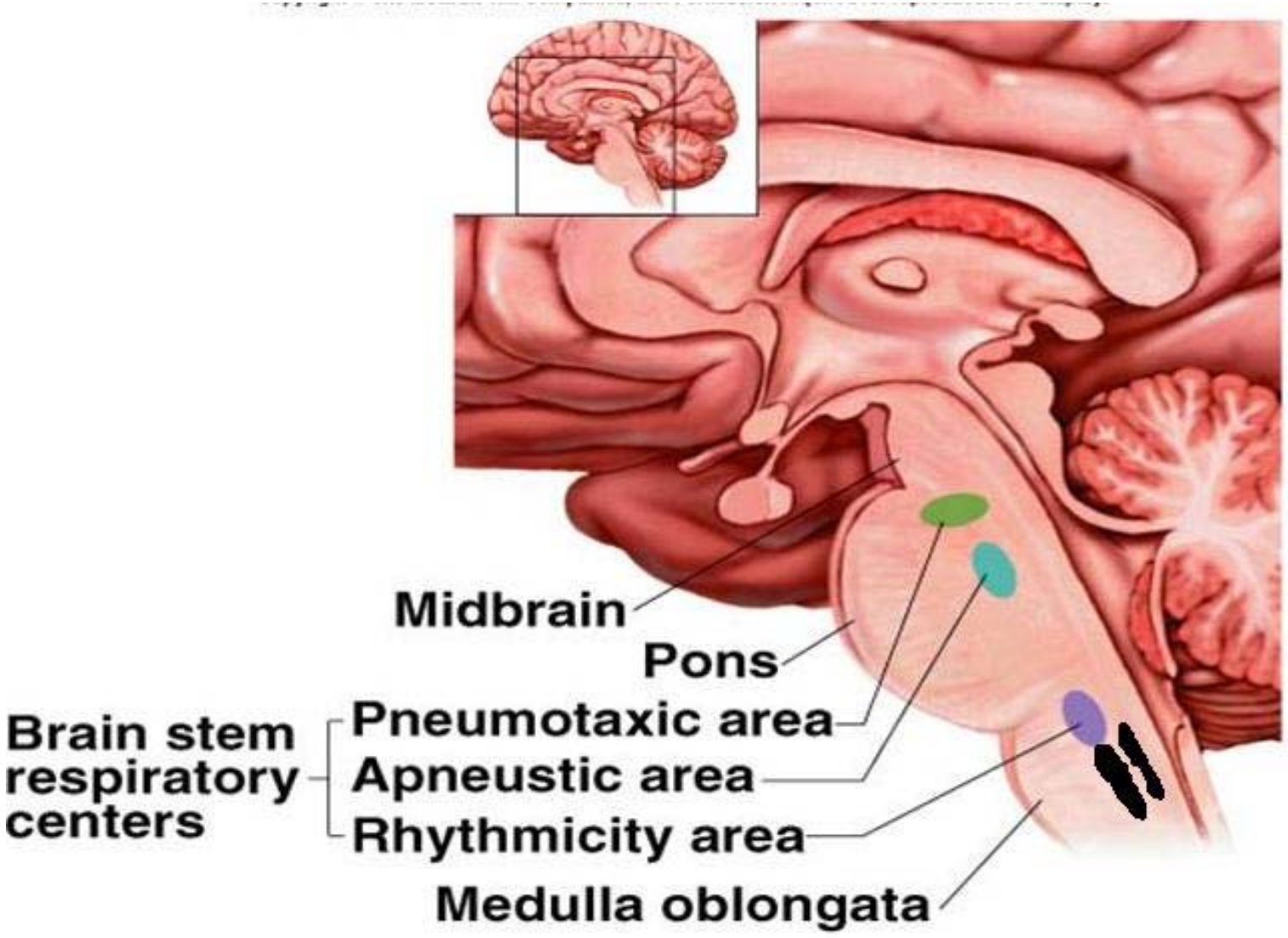
- CENTRAL CONTROLLER: Controlled Mainly at the level of brainstem
- Medullary respiratory centre:
  - Dorsal medullary respiratory neurones :
    - Associated with Inspiration
    - Neurons responsible for the basic rhythm of breathing
    - Activates Reticulospinal tract in the spinal cord, phrenic and intercostal nerves and finally stimulate the respiratory muscles
  - Ventral medullary respiratory neurons
    - Are associated with expiration.
    - These neurons are silent during quite breathing
    - Activated during forced expiration when the rate and the depth of the respiration is increased

CONTROL OF RESPIRATION

- APNEUSTIC CENTRE :
  - Located in the lower pons
  - Exact role of this centre in the normal breathing is not known
  - Without constant influence of this centre respiration becomes shallow and irregular

- PNEUMOTAXIC CENTRE:
  - Located in the upper pons.
  - Have an inhibitory effect on the both inspiratory and apneustic centres.
  - Responsible for the termination of inspiration by inhibiting the activity of the dorsal medullar neurones.
  - Regulates the volume and secondarily the rate of the respiration.

CONTROL OF RESPIRATION



CONTROL OF RESPIRATION

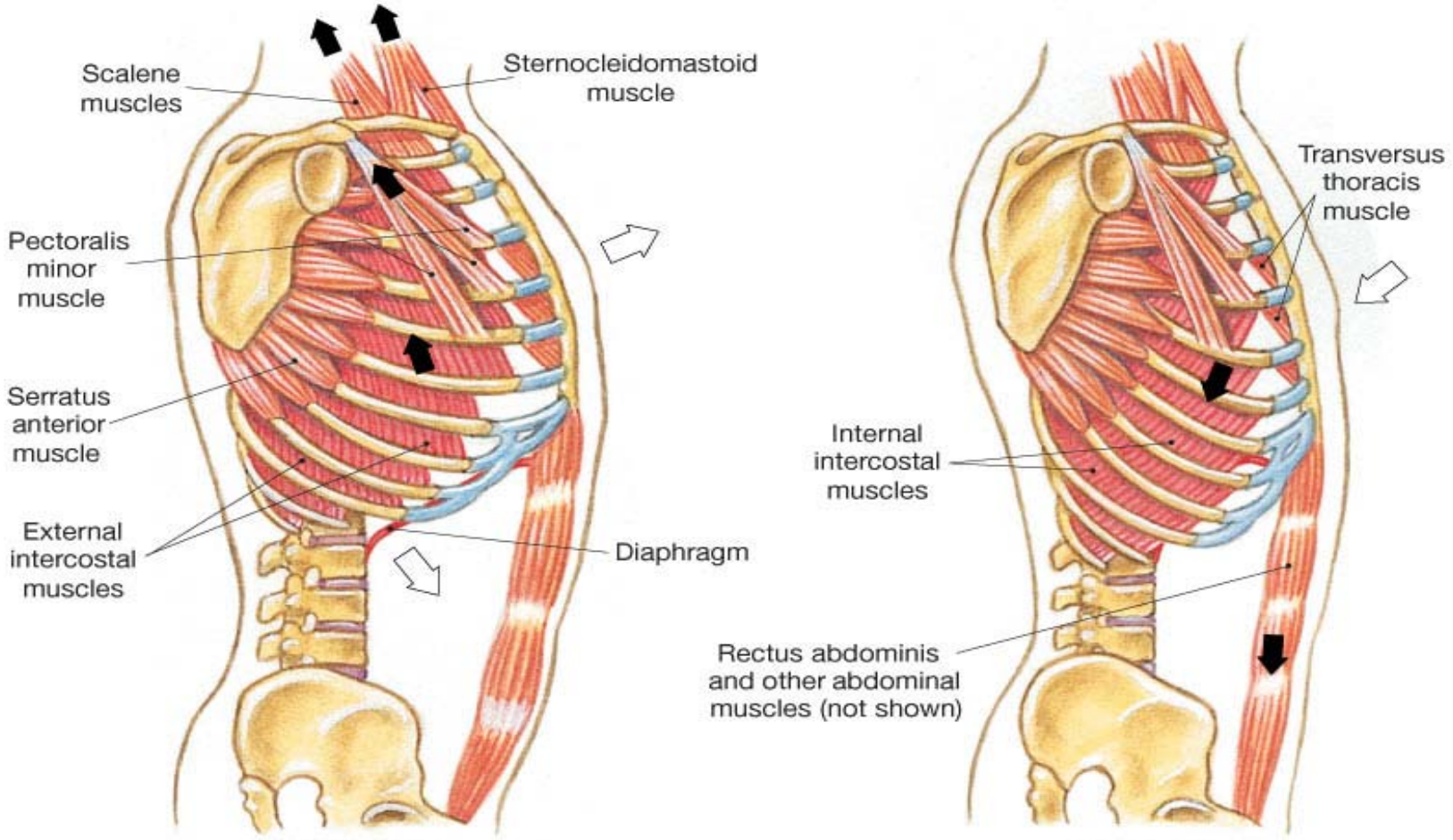
- RESPIRATORY MUSCLES:
  - Diaphragm
  - External intercostals
  - Accessory ms.
    - Scalene
    - Sternomastoids
    - Alae nasi

  Inspiratory

  - Abdominal ms.
  - Internal intercostal ms.

  Expiratory

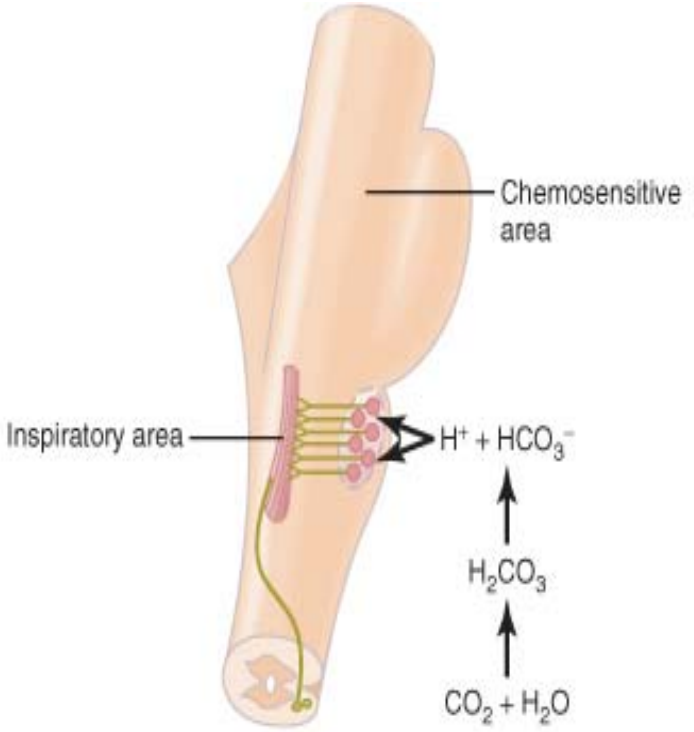
CONTROL OF RESPIRATION



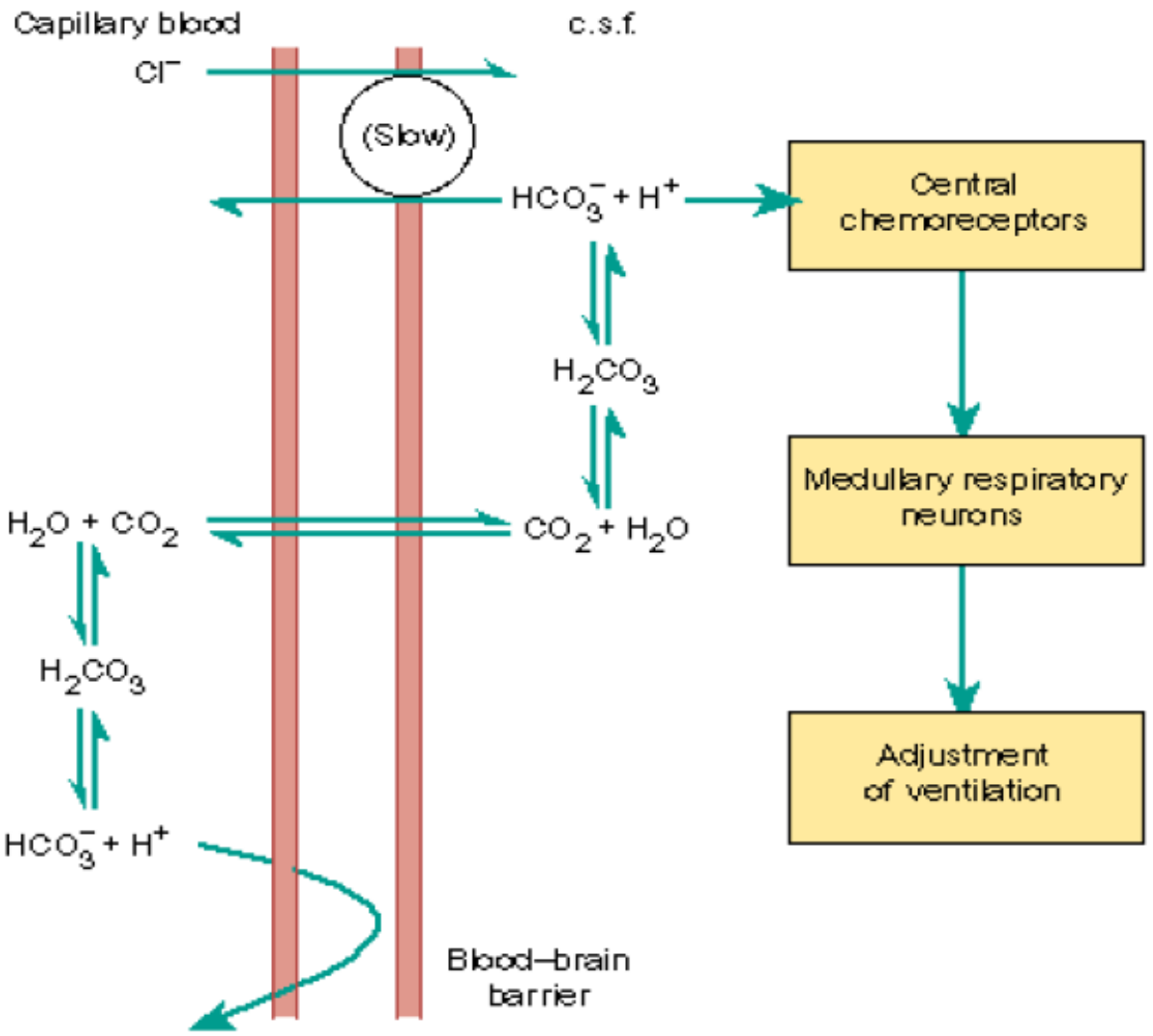
CONTROL OF RESPIRATION

SENSORS

- CENTRAL CHEMORECEPTORS
  - Located near the ventral surface of medulla
  - Bathed in brain ECF
  - Actually respond to changes in H+ concentration in these compartments
  - Increase in H+ stimulates chemoreceptors resulting in hyperventilation



CONTROL OF RESPIRATION

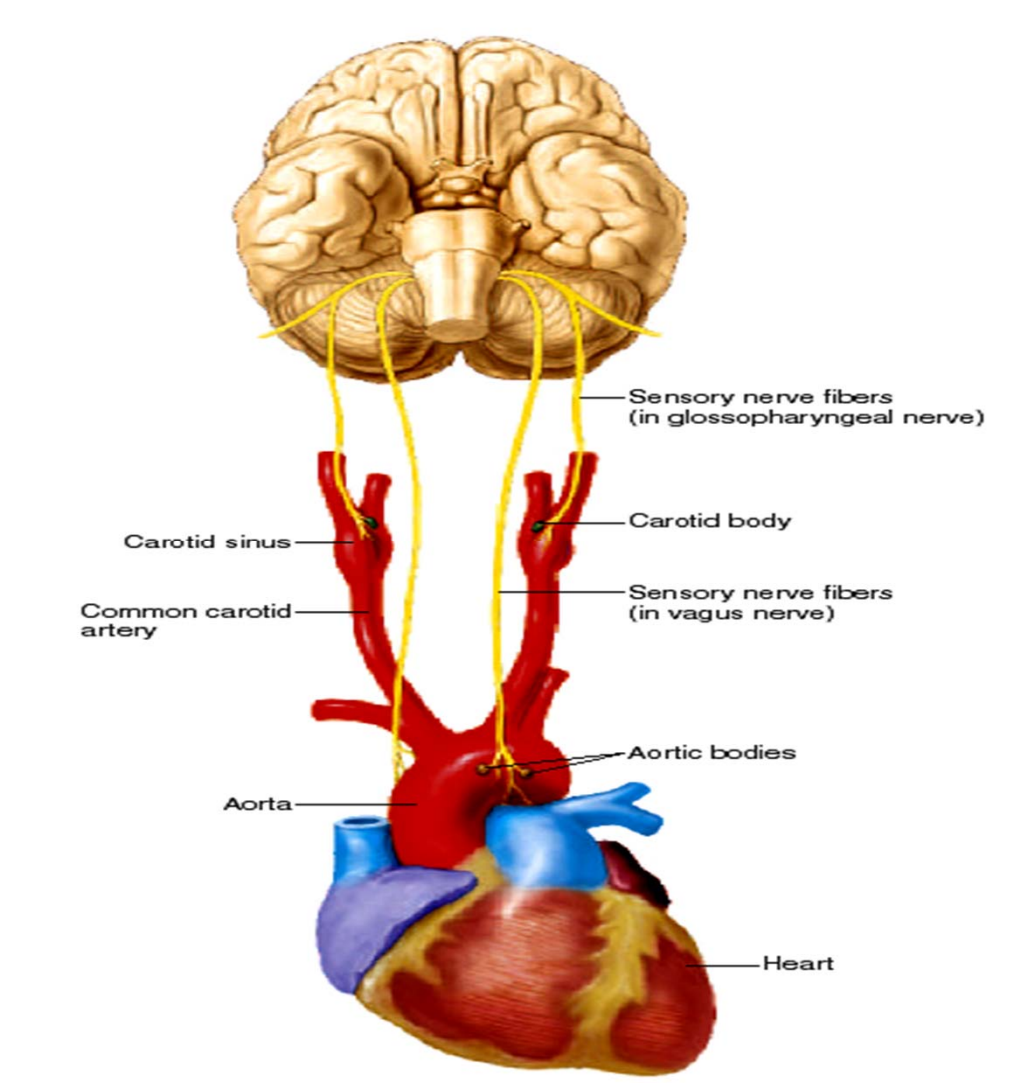


CONTROL OF RESPIRATION

- PERIPHERAL CHEMORECEPTORS
  - Located in – Bifurcation of Carotid artery

    Aortic arch
  - Connected to the respiratory centre In the medulla
  - Glossopharingeal nerve (carotid body)
  - Vagus nerve (aortic body)
  - Respond to ↓ed arterial PO_2 and ↑ed PCo_2 and H^+
  - Rapidly responding

CONTROL OF RESPIRATION



Peripheral chemoreceptors are the only sensors detecting a fall in PO2

LUNG RECEPTORS

- PULMONARY STRETCH RECEPTORS
  - Slow adapting
  - Lie in airway smooth muscles
  - Stimulated by distension of lung
  - Reflex action inhibits inspiratory activity & causes bronchodilatation
  - Determine rate & depth of breathing
  - Insensitive to "pathological" changes in the lungs such as
    - Micro embolism
    - Mild bronchoconstriction
    - Inhalation of irritants and dust
  - Weakly sensitized by pulmonary congestion /edema due to LVF

LUNG RECEPTORS

- IRRITANT RECEPTORS (Deflation or collapse receptors)
  - Rapidly adapting
  - Lie in airway epithelial cells
  - Stimulated by- noxious gases, cigarette smoke, inhaled dust & cold air
  - Cause bronchoconstriction , hyperpnea and hyperventilation

- J (JUXTACAPILLARY) RECEPTORS
  - Ending of nonmyelinated C fibers
  - In alveolar wall close to capillaries
  - Stimulated by hyperinflation /Inhalation of strong irritant gases, including halothane
  - Cause tachypnea , rapid shallow breathing , bronchoconstriction , apnea (intense stimulation)
  - Role in rapid shallow breathing and dyspnea associated With LHF and ILD

LUNG RECEPTORS

- BRONCHIAL C FIBERS
  - Supplied by bronchial circulation
  - Stimulated by hyperinflation /chemicals injected into bronchial circulation
  - Cause rapid shallow breathing, bronchoconstriction & mucus secretion

LUNG RECEPTORS

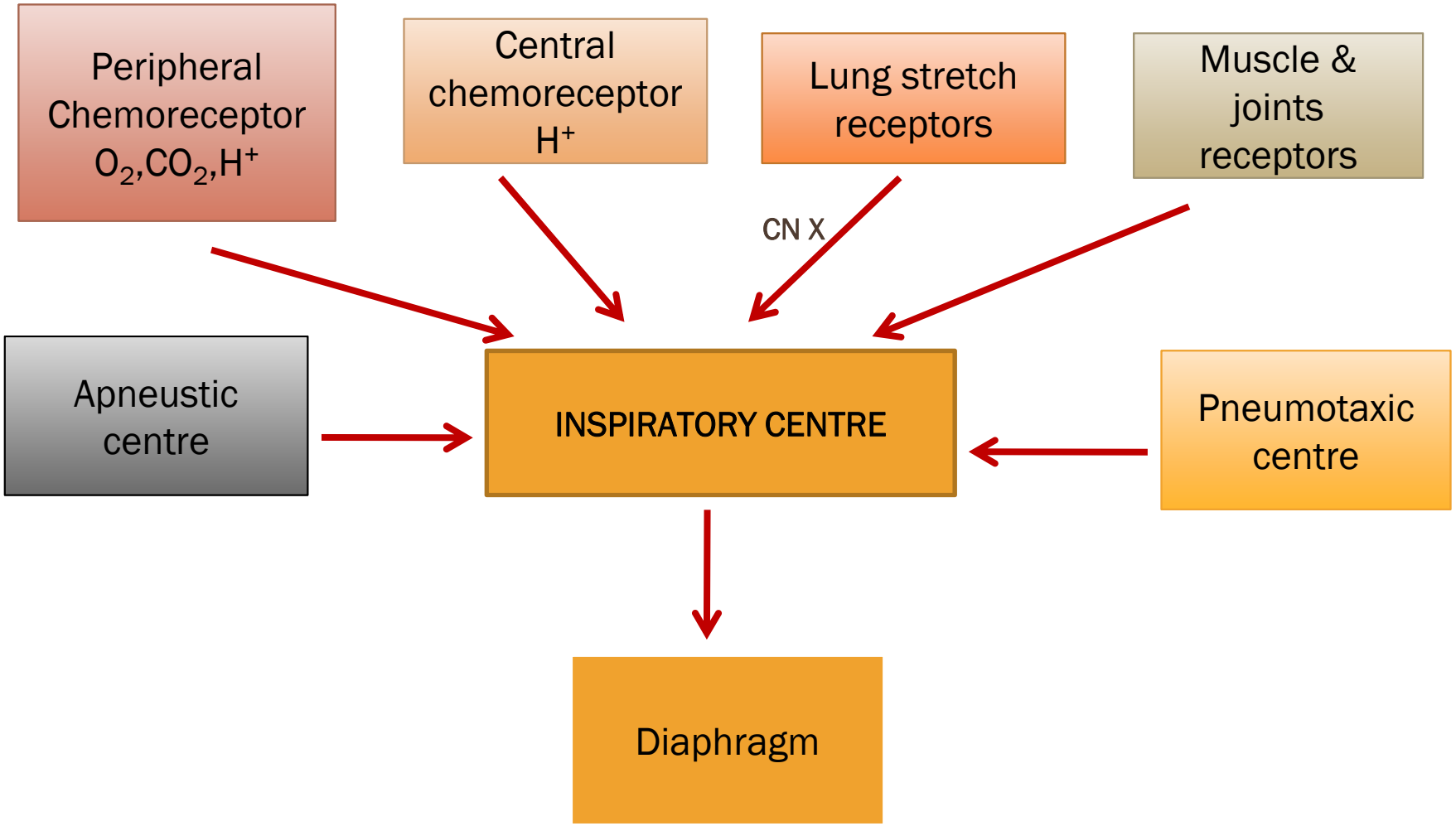
- Two other types of respiratory receptor
  - Cough receptors in the tracheal epithelium
  - Pulmonary arterial baroreceptors.

LUNG RECEPTORS

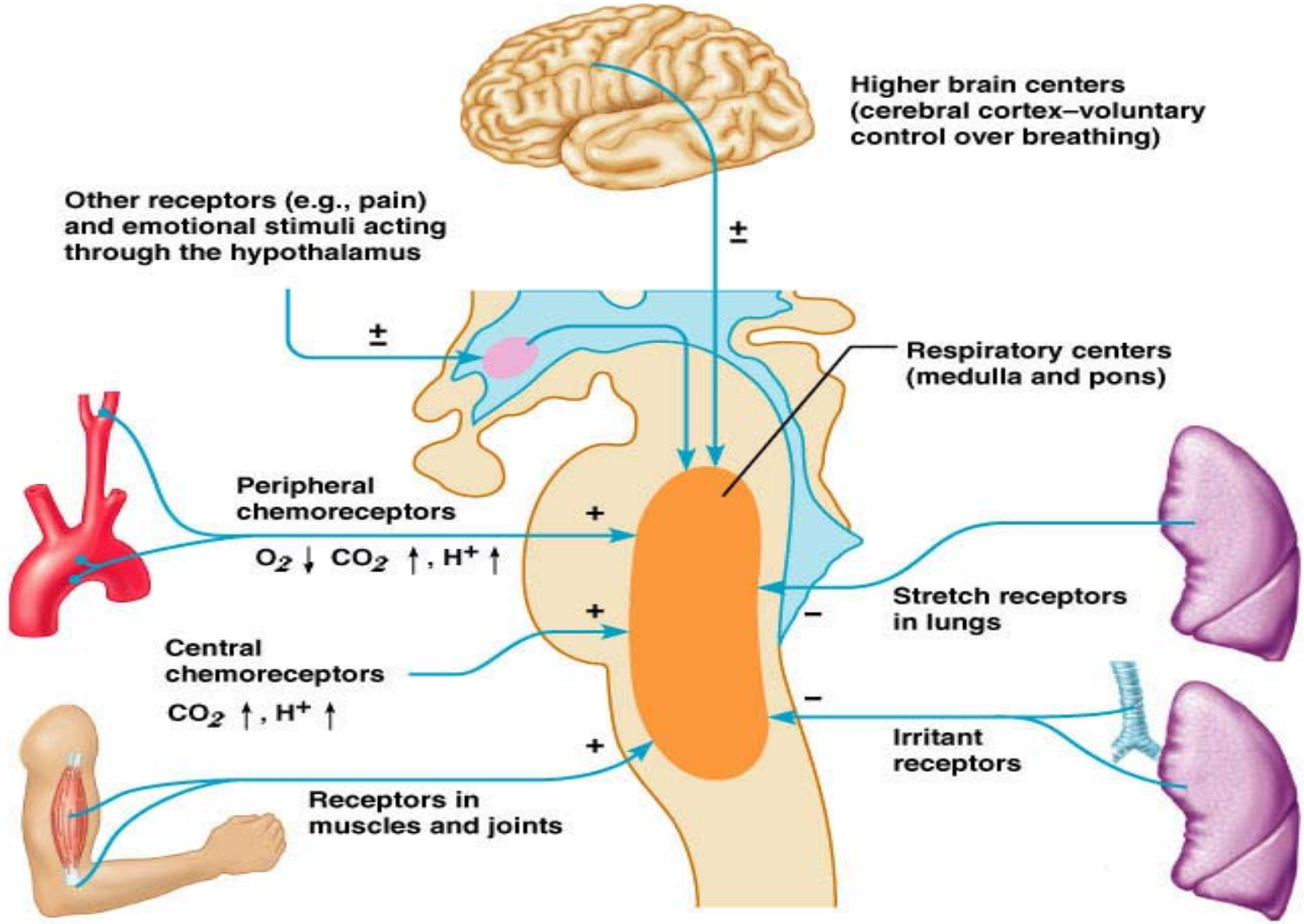
Summary of the responses of three types of lung receptor in various physiological and pathologica conditions. The brackets mean weak or not clearly established effect.

Stimulus	Receptor response		
	Pulmonary stretch	Type-J	Irritant
Lung inflation	+	−	+
Lung deflation	−	−	+
Dust	−	−	+
Chemical irritants	−	+	+
Halothane	(+)	+	−
Ether	(+)	+	+
Phenyl diguanide	−	+	+
Bronchoconstriction	−	−	+
Microembolism	−	+	+
Pulmonary congestion	(+)	+	+

COMPONENTS OF INVOLUNTARY CONTROL SYSTEM



SUMMARY:OVERALL CONTROL OF ACTIVITY OF RESPIRATORY CENTRE



RESPIRATORY TRACT REFLEXES

- HERING & BREUER INFLATION REFLEX
  - Inflation of the lungs inhibit further inspiratory ms. Activity
  - Mediated by pulmonary stretch receptors
  - Uncommon at quiet breathing
  - Barbiturate depress this reflex
- DEFLATION REFLEX
  - Deflation of the lungs tends to initiate inspiratory activity

RESPIRATORY TRACT REFLEXES

- HEAD'S PARADOXICAL REFLEX
  - Paradoxically stimulates a deeper breath rather than inhibiting further inspiration
  - Responsible for
    - Deep Breath (Sighs)
    - First breaths of Infants

FACTORS AFFECTING RESPIRATION

- CO_2 : Most imp. Stimulus
  - Most of the stimulus from central chemoreceptos but peripheral chemoreceptors also contribute
  - Magnified effect if PO2 is low
  - ↓ ed response – sleep, ↑ ing age, trained athletes, drugs

- O2 : Hypoxia
  - Only peripheral chemo. Involved
  - Negligible control during normoxia
  - Imp. In high altitude & chronic hypoxia

FACTORS AFFECTING RESPIRATION

- pH
  - Reduction stimulates ventilation
  - Site of action : peripheral chemoreceptors

- EXERCISE
  - Ventilation increases
  - **THEORIES**
    - Passive movements increases ventilation
    - Increase in body temp.
    - Impulses from motor cortex
    - Oscillation in arterial Po_2 , Pco_2

FACTORS EFFECTING BREATHING

Factors	Receptors Stimulated	Response	Effect
Stretch of tissues	Stretch receptors in visceral pleura, bronchioles, and alveoli	Inhibits inspiration	Prevents overinflation of lungs during forceful breathing
Low plasma Po_2	Chemoreceptors in carotid and aortic bodies	Increases alveolar ventilation	Increases plasma Po_2
High plasma Pco_2	Chemosensitive areas of the respiratory center	Increases alveolar ventilation	Decreases plasma Pco_2
High cerebrospinal fluid hydrogen ion concentration	Chemosensitive areas of the respiratory center	Increases alveolar ventilation	Decreases plasma Pco_2

STATIC CHARACTERISTICS OF THE LUNGS

COMPLIANCE

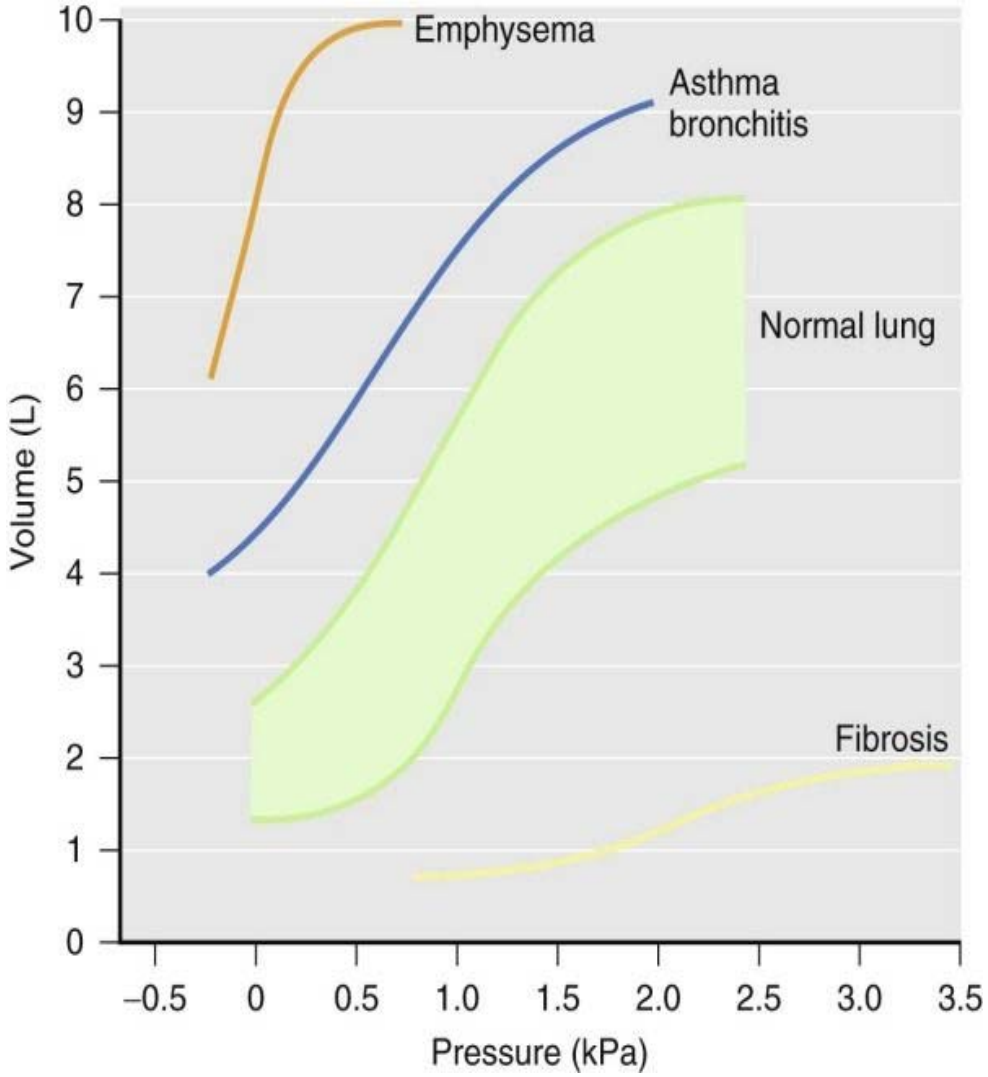
Compliance - Effort needed to stretch lungs

$$C_T\,(L/cm\ H_2O) = \frac{\Delta V(L)}{\Delta P\ (cmH_2O)}$$

Normal : 0.2-0.3 L/cm H20

COMPLIANCE contd.

- Reduced compliance
  - Pulmonary fibrosis
  - Alveolar edema
  - Atelectasis
- Increased Compliance
  - Emphysema



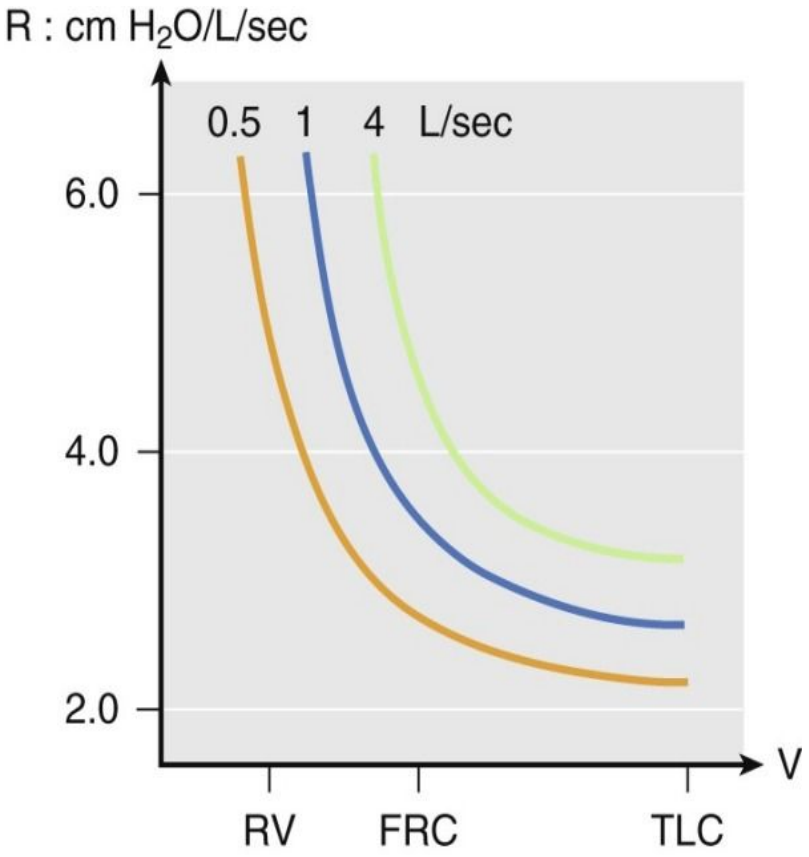
STATIC CHARACTERISTICS OF THE LUNGS

RESISTANCE

Relationship between pressure gradient & rate of air flow

- $R(cmH_2O/L/sec) = \frac{\Delta P(cmH_2O)}{\Delta V(L/sec)}$

- ΔP depends on – airway caliber, rate & pattern of airflow



PATTERN OF AIRFLOW

- Laminar flow – airway below main bronchi
- Turbulent flow - trachea

STATIC CHARACTERISTICS OF THE LUNGS

RESISTANCE contd.

- Normal : 1 cm H20/ L/ sec.
- Maximum resistance in medium sized bronchi
- Increases with
  - Low lung volumes
  - Increased gas density
  - Decreased arterial PCO2
  - Cholinergic drugs

SURFACE TENSION

- S.T. is the molecular force present on the surface of a liquid that tends to make the exposed surface area as small as possible
- Laplace law – pressure across a curved surface is equal to twice the surface tension at liquid interface divided by radius

$$P = 2T / R$$

SURFACE TENSION contd.

- As alveoli ↓ in size during expiration, pressure tending to collapse them ↑ & a vicious cycle is established
- Role of surfactant – conc. of surfactant ↑ on the surface of liquid as S.A ↓
- Surfactant lower S.T at air liquid interface in the alveolus & prevent collapse at low lung volumes

PULMONARY SURFACTANT

- Reduces the surface tension of the alveolar lining layer
- Produced by type II alveolar epithelial cells
- Contains dipalmitoyl phosphatidylcholine
- Absence results in reduced lung compliance, alveolar atelectasis, tendency to pulmonary edema

FACTORS DECREASING SURFACTANT

- Oxygen therapy
- IPPV with high pressure
- Pulmonary collapse
- ↓ Pulmonary circulation - embolism
- Anaesthetic agent
- Patient with valve replacement procedure

HUMIDIFICATION

- Normal humidifying mechanism- nose & mouth
- Bypassed – ETT
  - Tracheostomy
- Benefits of humidification-
  - Protect drying of mucosa
  - Reduce heat loss
  - Reduce incidence of coughing & breath-holding during inhalational induction

DRY AIR ENTERING THE TRACHEA

Inflammatory reaction

Dried & tenacious secretions

Difficult to remove cough out

Damage / inhibition of cilia

Loss of cilia & keratinization of tracheal epithelium

HUMIDITY

- Normally, air entering trachea is saturated with water vapour - humidity of 34 g /m^3

  i.e. fully saturated at 34°C

- Two methods of increasing humidity artificially
  - Humidifying the environment - in infant incubators
  - Humidifying the inspired gases- humidifiers

SIZE OF DROPLETS

- > 20 µm - form pool of water in tubing/upper resp. tract
- 5µm - fall in region of trachea
- 1µm - pass upto alveoli & get deposited
- < 1µm - ideal
- Extremely stable, can be
- Inspired & expired again

VENTILATION AND PERFUSION

VARIATION OF VENTILATION WITH POSTURE

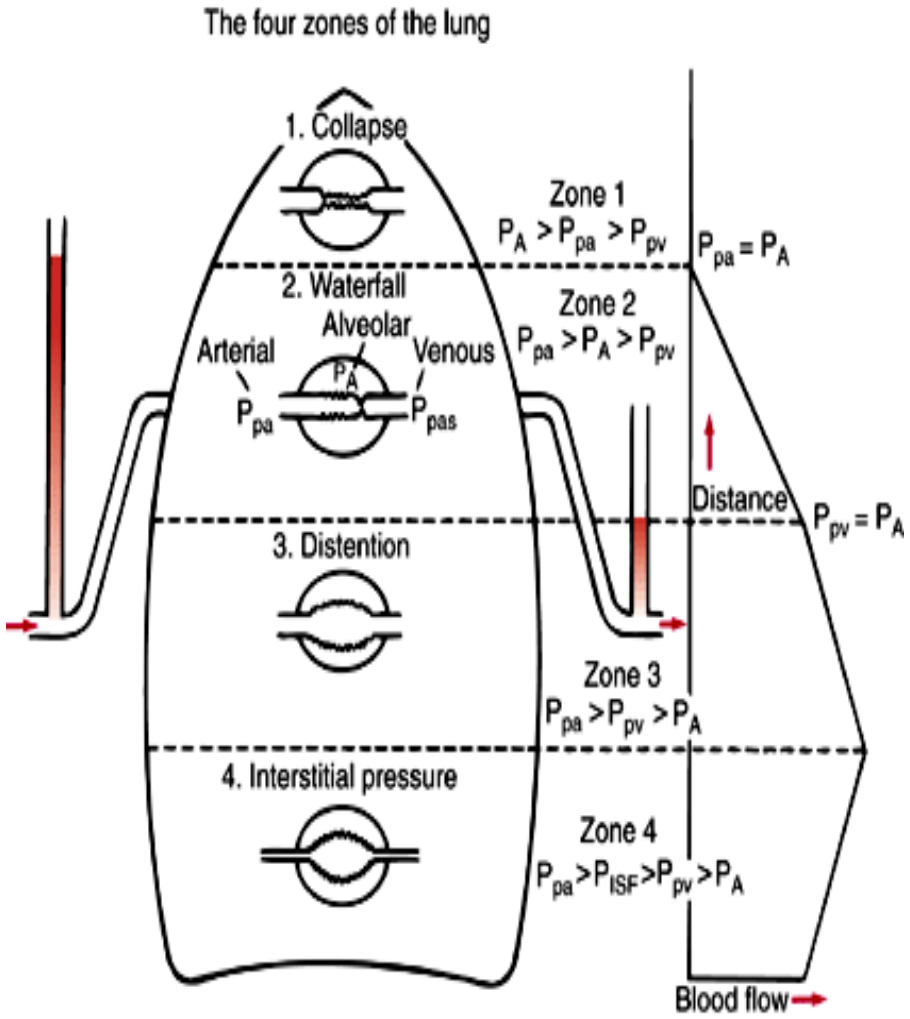
- Upright posture - ventilation is more in the base of the lung than at the apex

- Supine posture –

  Posterior areas better ventilated than the anterior ones

  Lateral position - dependent lung best ventilated

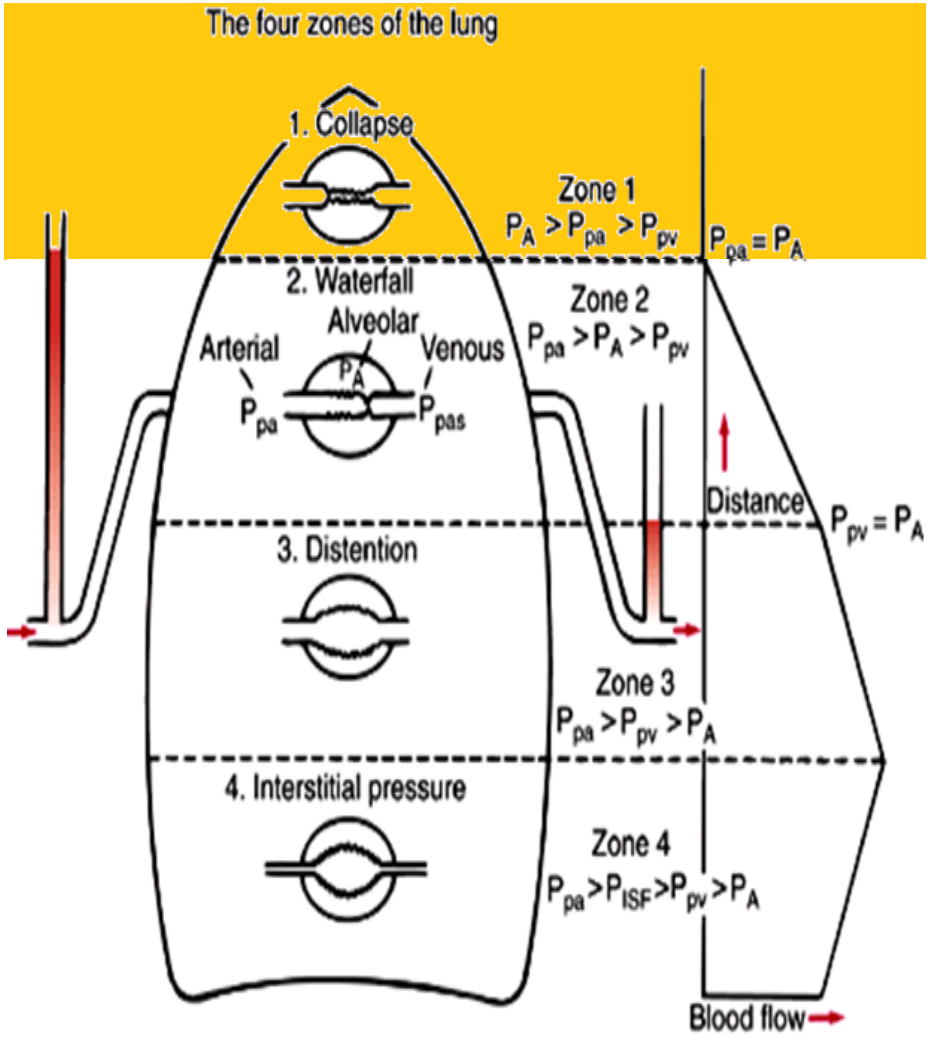
NORMAL PHYSIOLOGY OF UPRIGHT POSITION



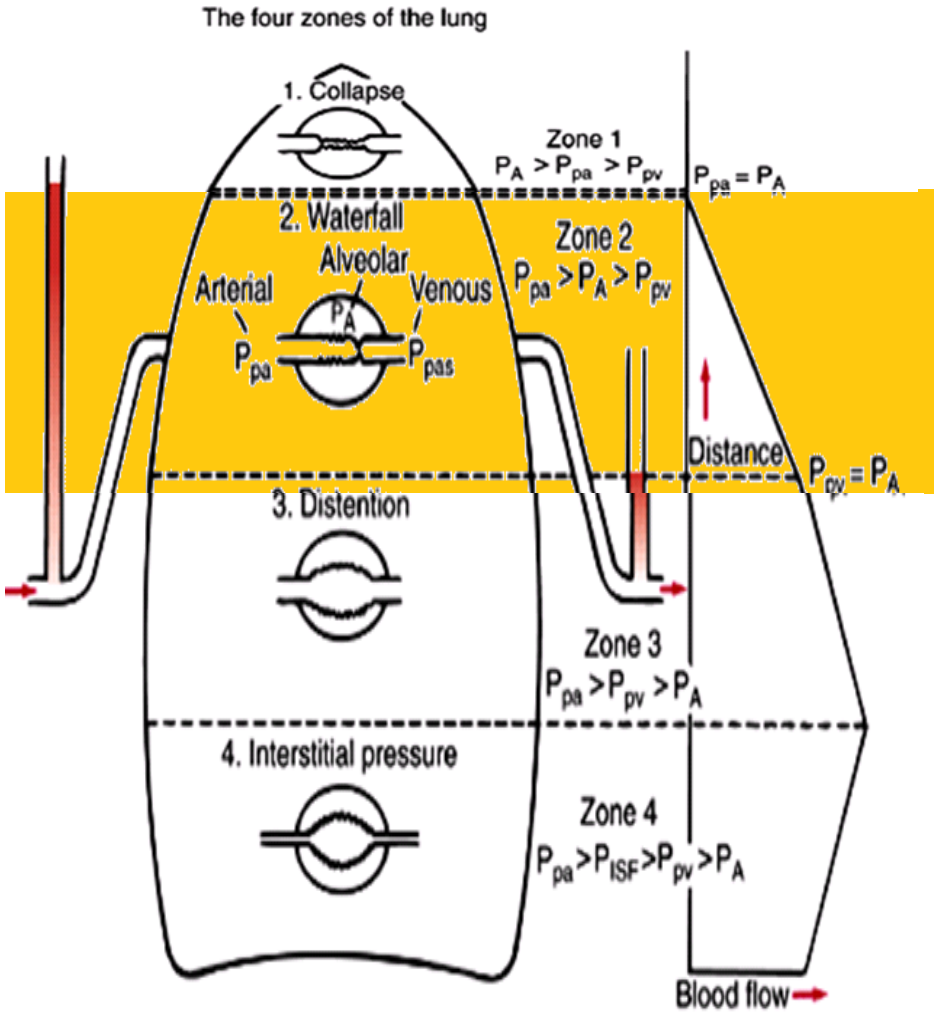
- 'WEST' zones
- Contraction of RV :
  - Propels blood into PA
- Absolute pressure : Decreases 1 cm of H2O for each cm travelled vertically up the lung

DISTRIBUTION OF PULMONARY PERFUSION

- Zone I -
  - **PA> Ppa > Ppv**
  - No blood flow
    - *Wasted ventilation*
    - Acts as alveolar dead space
    - Zone I increased in
      - Hypotension
      - Positive pressure ventilation

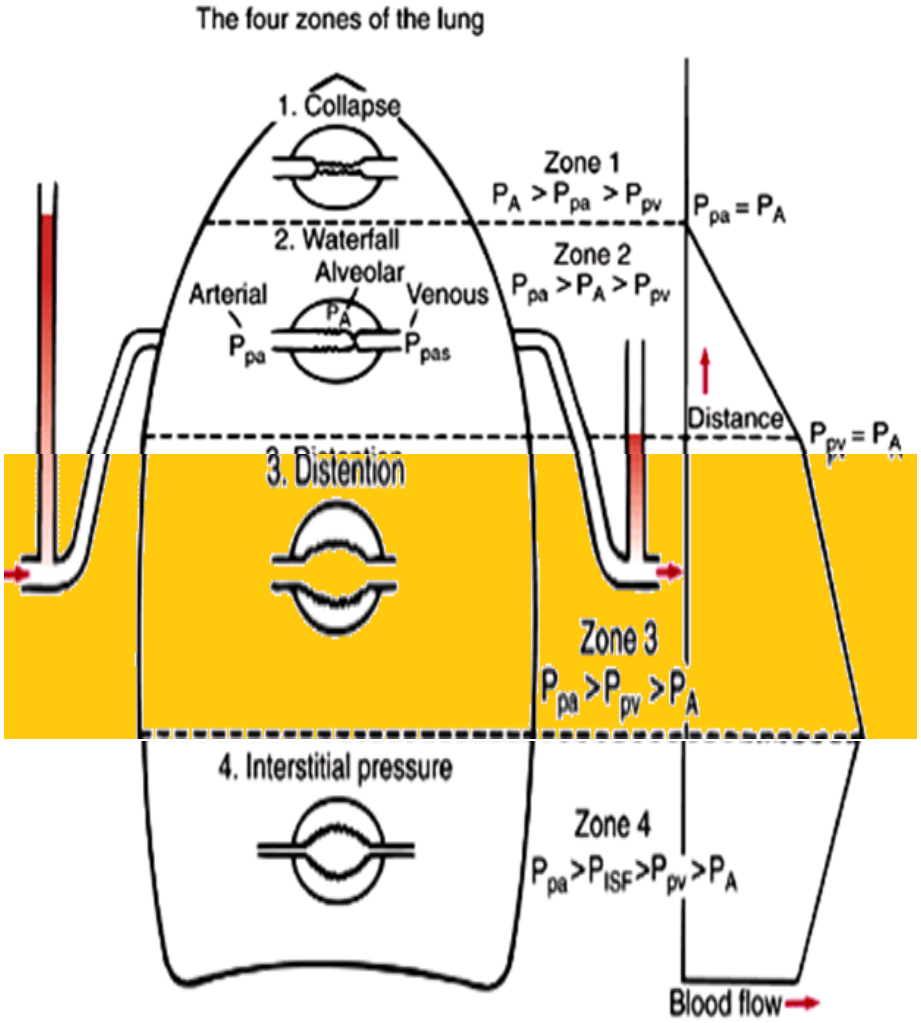


- Zone II –
  - Blood flow is determined by : **Ppa – PA**
  - *k/as – waterfall effect /Starling resistor / sluice / weir effect*
  - *Height of upstream river ~ Ppa*
  - *Height of Dam ~ PA*
  - Mean driving pressure increases linearly down the lung zone

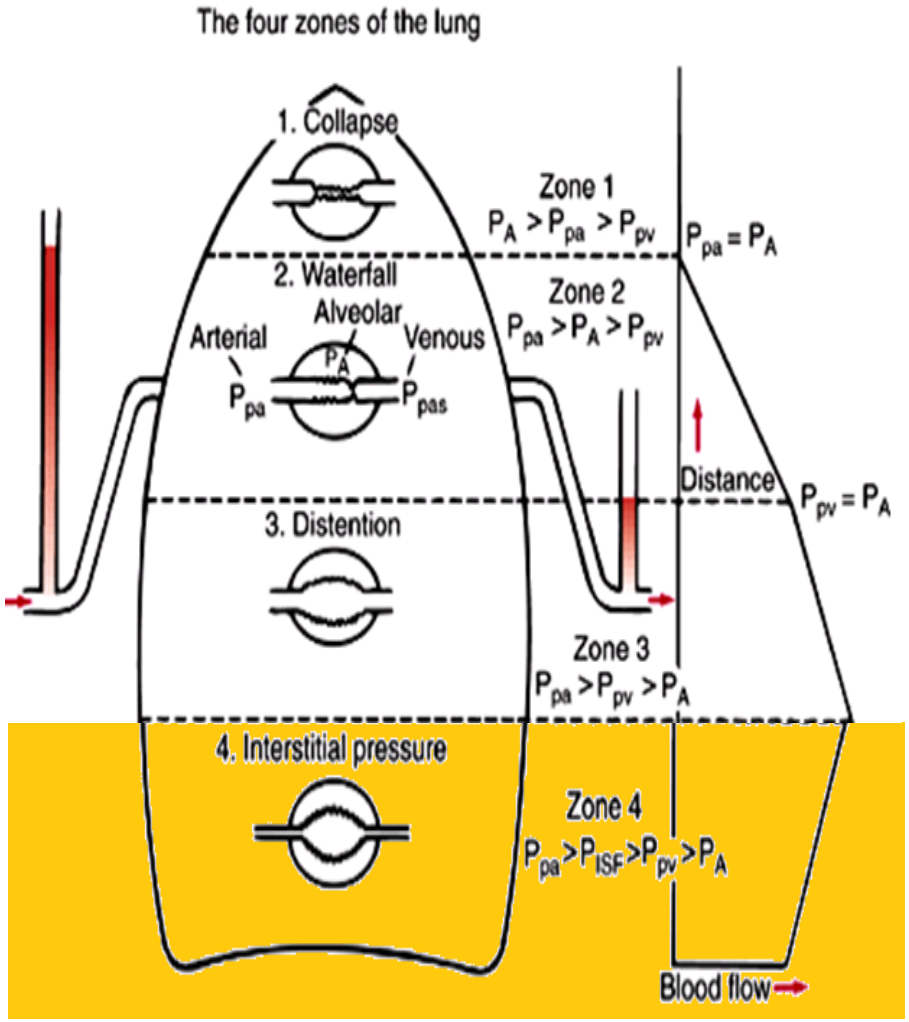


- Zone III –
  - Blood flow is determined by :

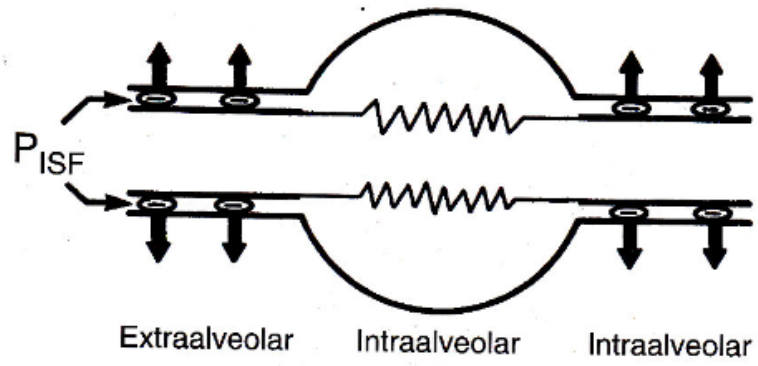
    Ppa – Ppv
  - Transmural distending pressures increase down zone 3
  - Blood flow is *continuous*

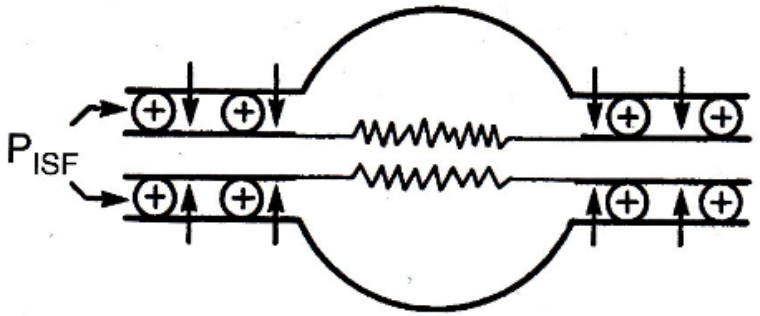


- Zone IV –
  - Vascular resistance of extra alveolar vessels increases
  - Pulmonary interstitial pressure > pulmonary venous pressure
  - *Blood flow is determined by* **Ppa – Ppisf**



EFFECT OF PISF ON EXTRA ALVEOLAR VESSELS

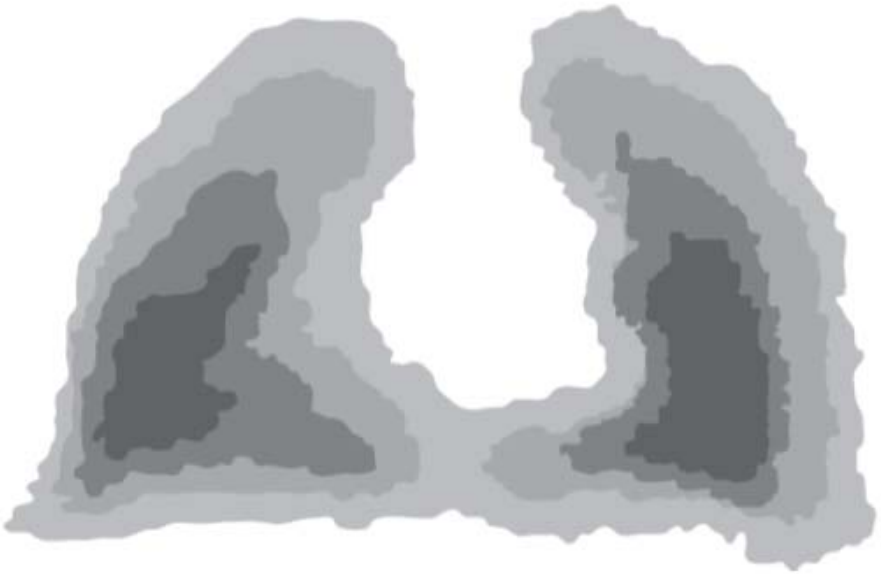




- Zone Four Increased in conditions of
  - Volume overload
  - Pulmonary embolism
  - Mitral stenosis

DISTRIBUTION OF PULMONARY PERFUSION

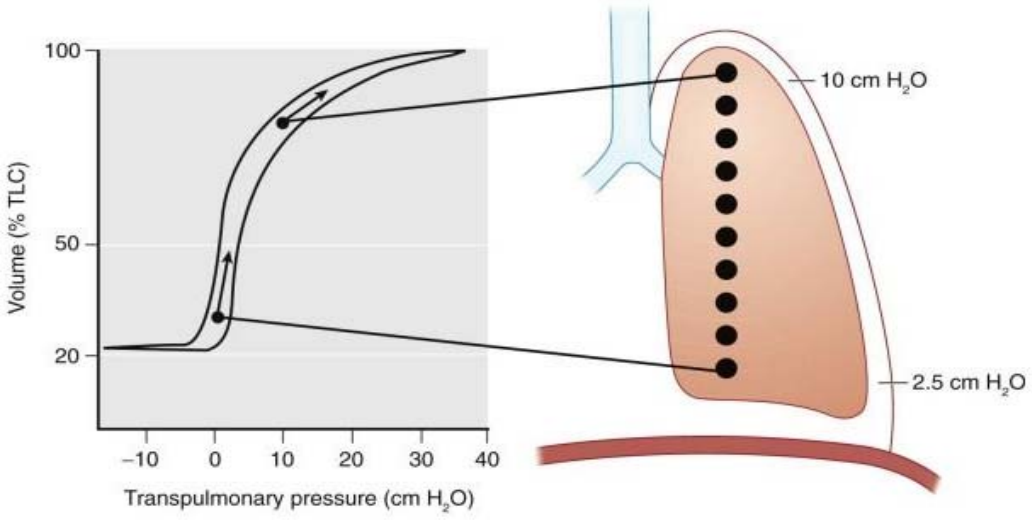
- Perfusion scanning:
- Gravitational distribution + onion-like layering



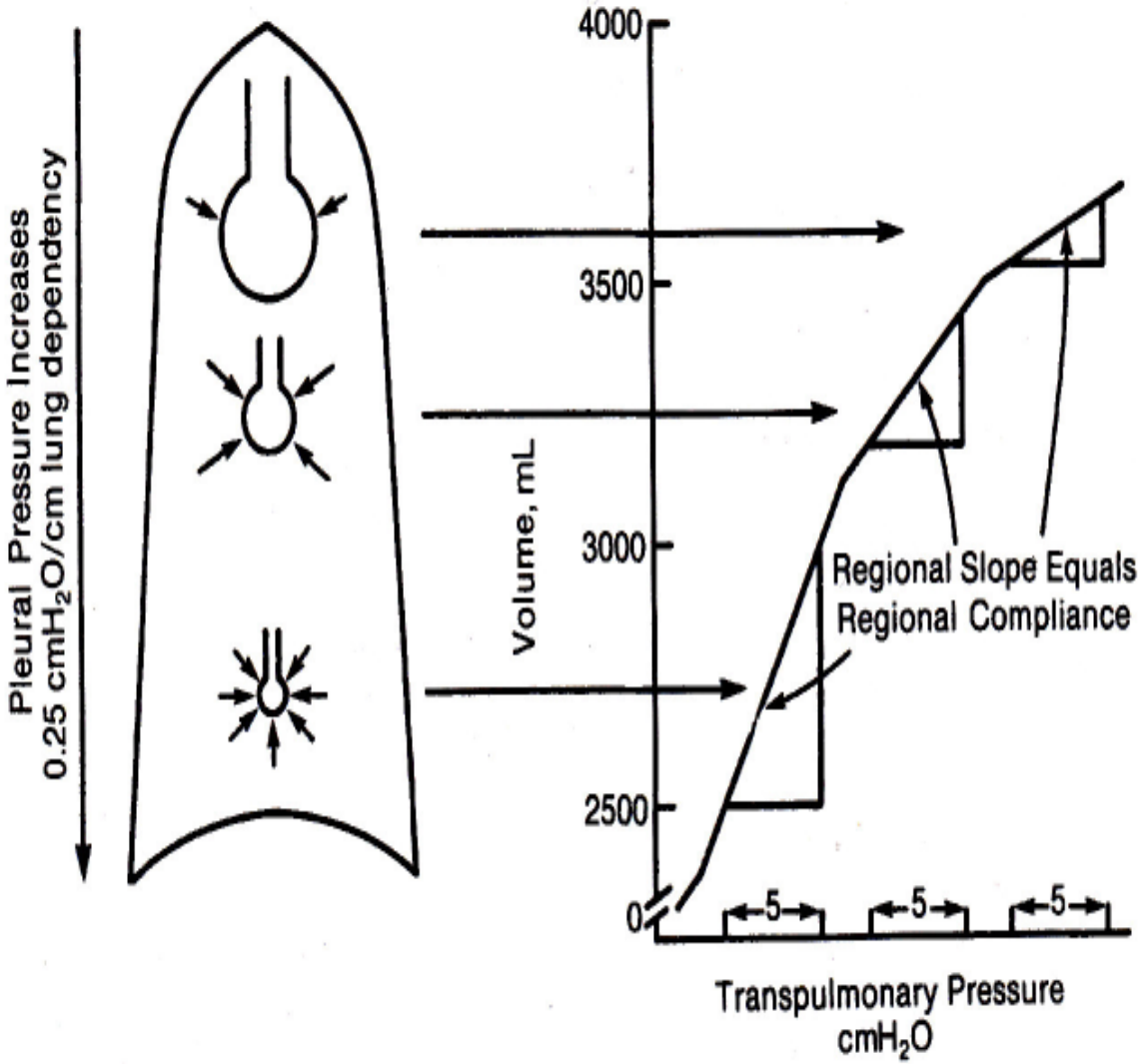
(reduced flow at the periphery of the lung than toward the hilum)

DISTRIBUTION OF VENTILATION

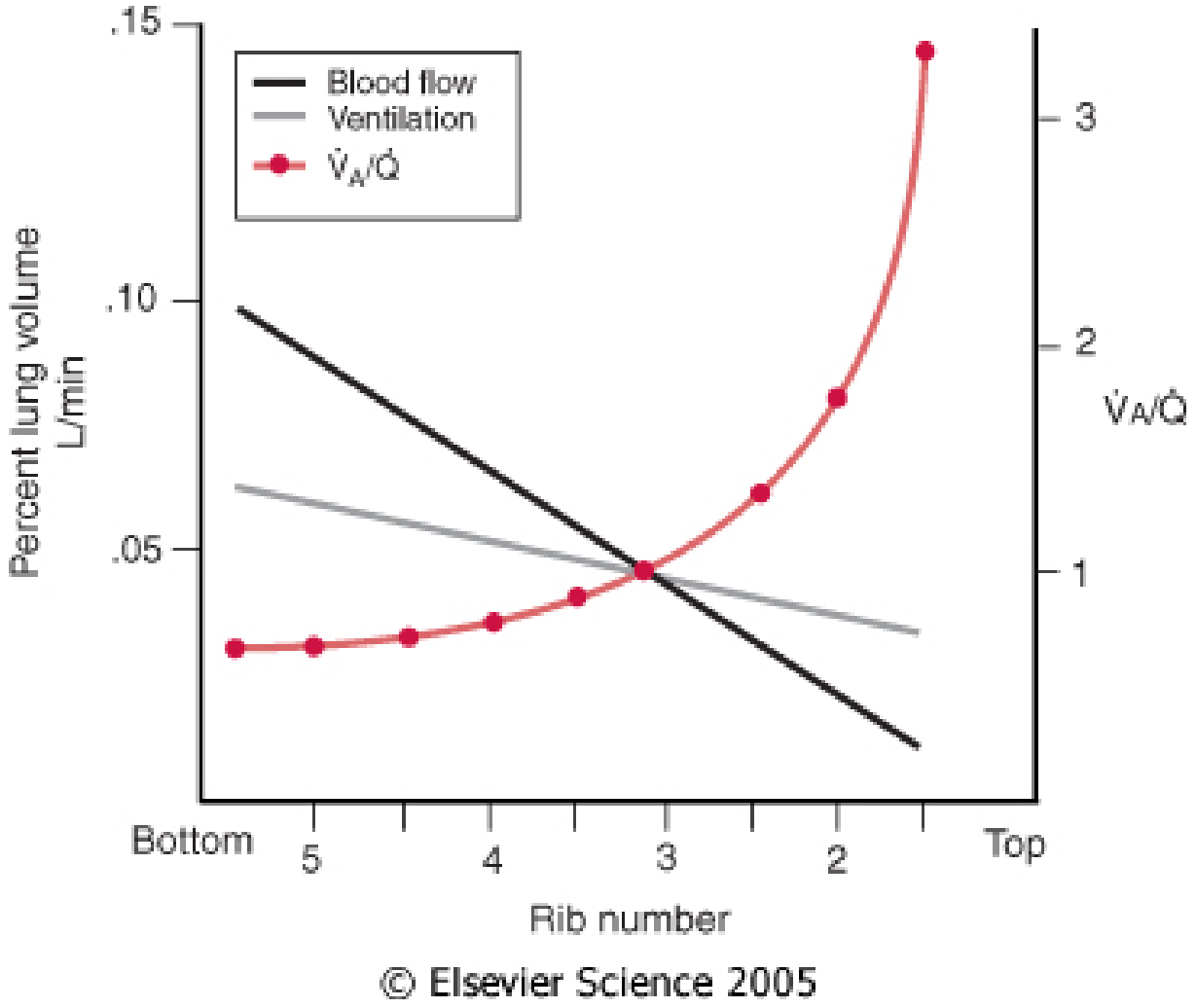
- Pleural pressure *increases* down the lung
- Fourfold decrease in alveolar volume
- *Transpulmonary pressure decrease from* top to bottom of lung
- Dependent alveoli are more compliant (*steep slope*)
- Non dependent alveoli are relatively non complaint (*flat slope*)
- Basal regions are more ventilated



DISTRIBUTION OF VENTILATION



VENTILATION PERFUSION MISMATCH



V/Q RATIO AND REGIONAL COMPOSITION OF ALVEOLAR GAS

- **Alveoli (Bottom)**

  Retain CO2

  Does not take enough O2

- **Alveoli (Top)**

  Gives off excessive CO2

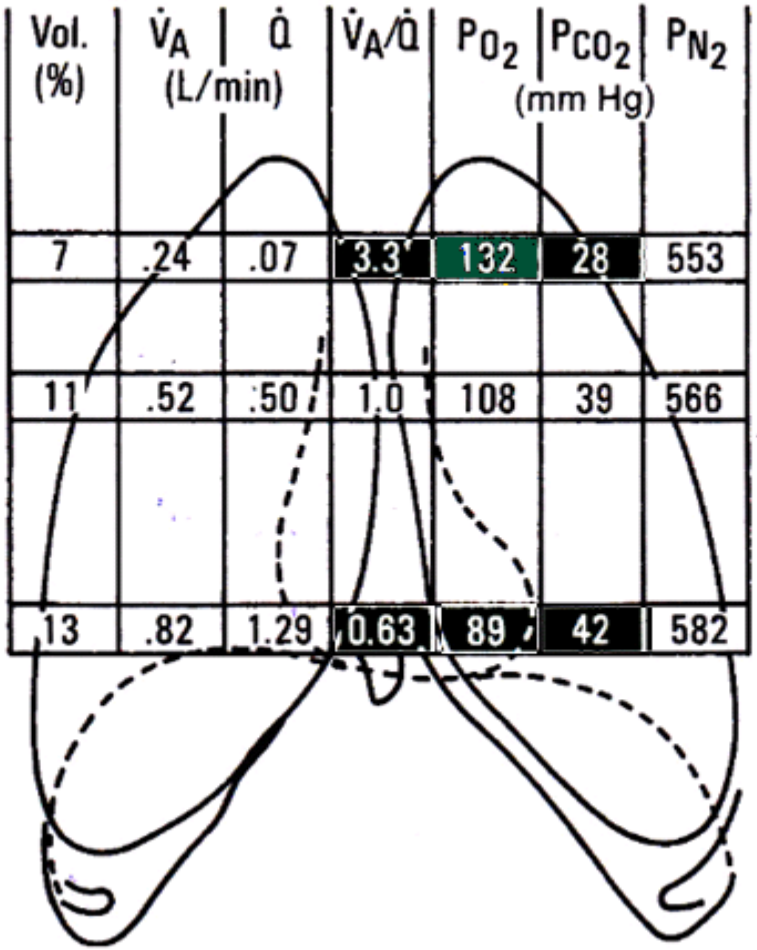
  Cannot take up enough O2

  (Due to flatness of ODC curve in this region)

PACO2 – PaCO2 : GRADIENT SMALL

PAO2 – PaO2 : GRADIENT LARGE

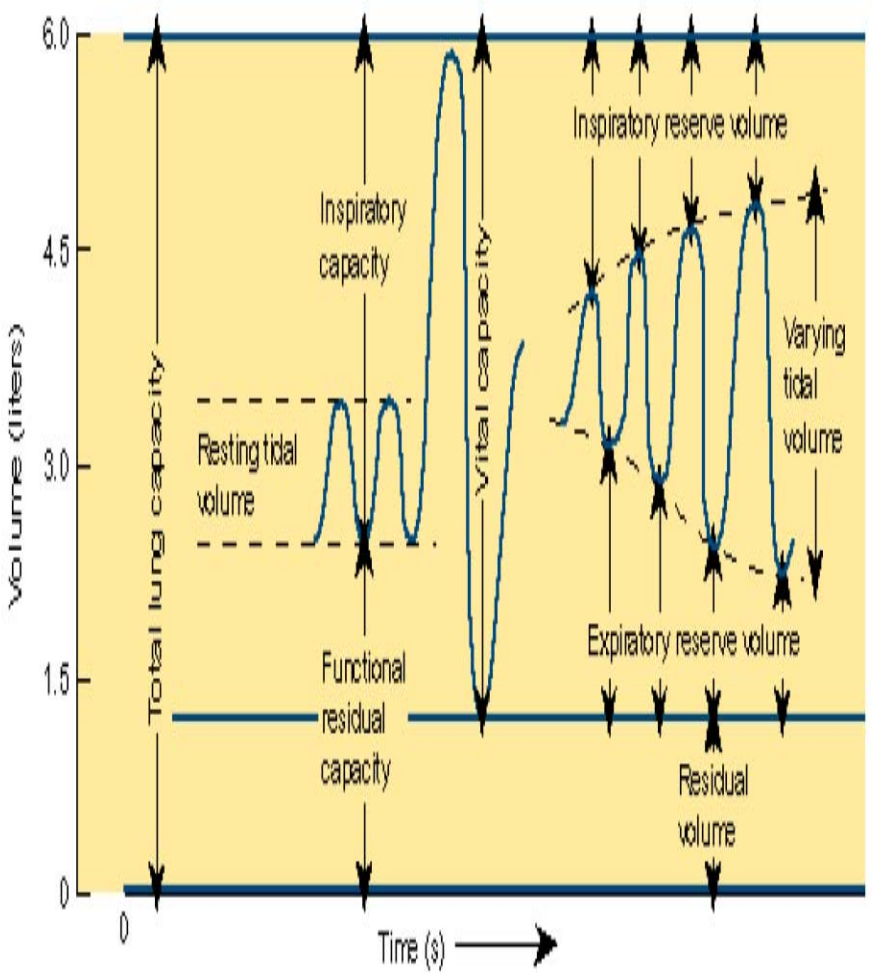
Final Composition : Which Zone occupies the Major lung portion ?



SHUNT – VENOUS ADMIXTURE

- Venous admixture is said to occur when blood passes through the lung without being properly oxygenated
- Anatomic Shunt- portion of CO that bypasses pulmonary capillaries (2% of CO) - bronchial, pleural, thebesian, anterior cardiac veins
- Capillary Shunt- portion of CO that perfuses nonventilated alveoli - atelectasis, pulmonary edema, consolidated pneumonia
- Hypoxemia (not responsive to increased FiO2)

LUNG VOLUMES AND CAPACITIES



4 VOLUMES:

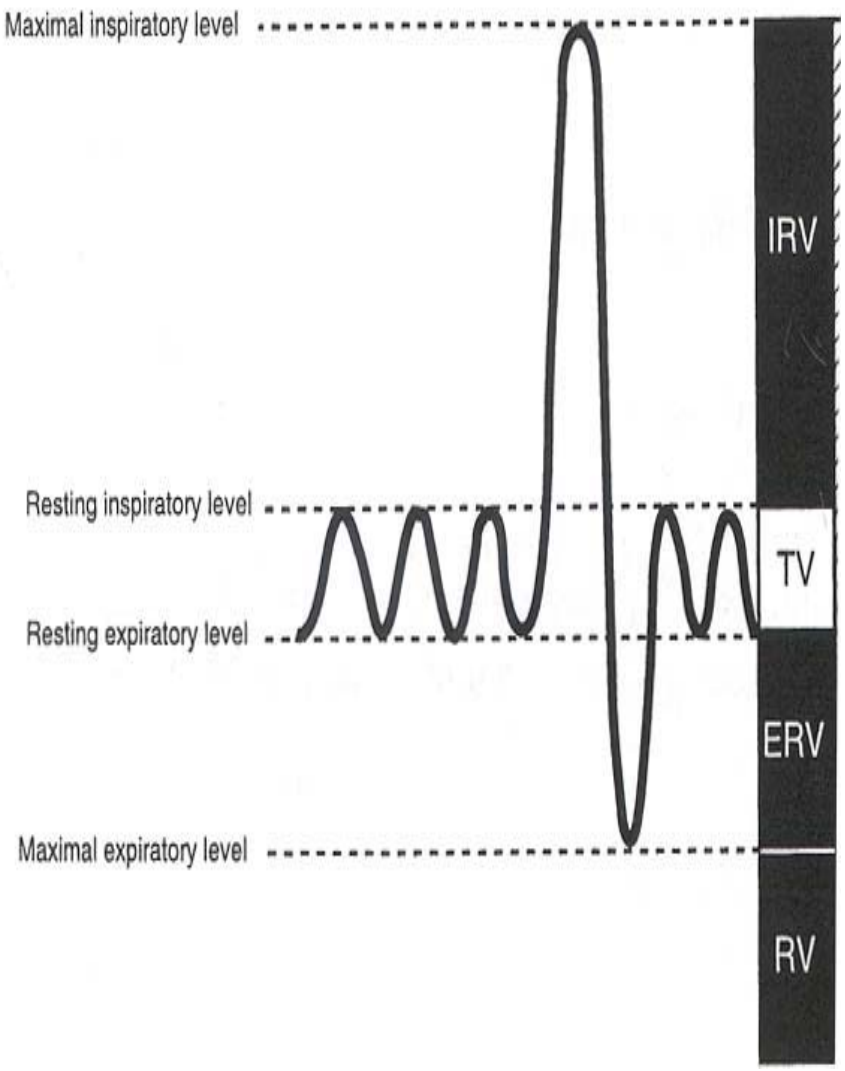
- Tidal volume
- Inspiratory reserve volume
- Expiratory reserve volume
- Residual volume

2 or more volumes comprise a capacity

4 CAPACITIES:

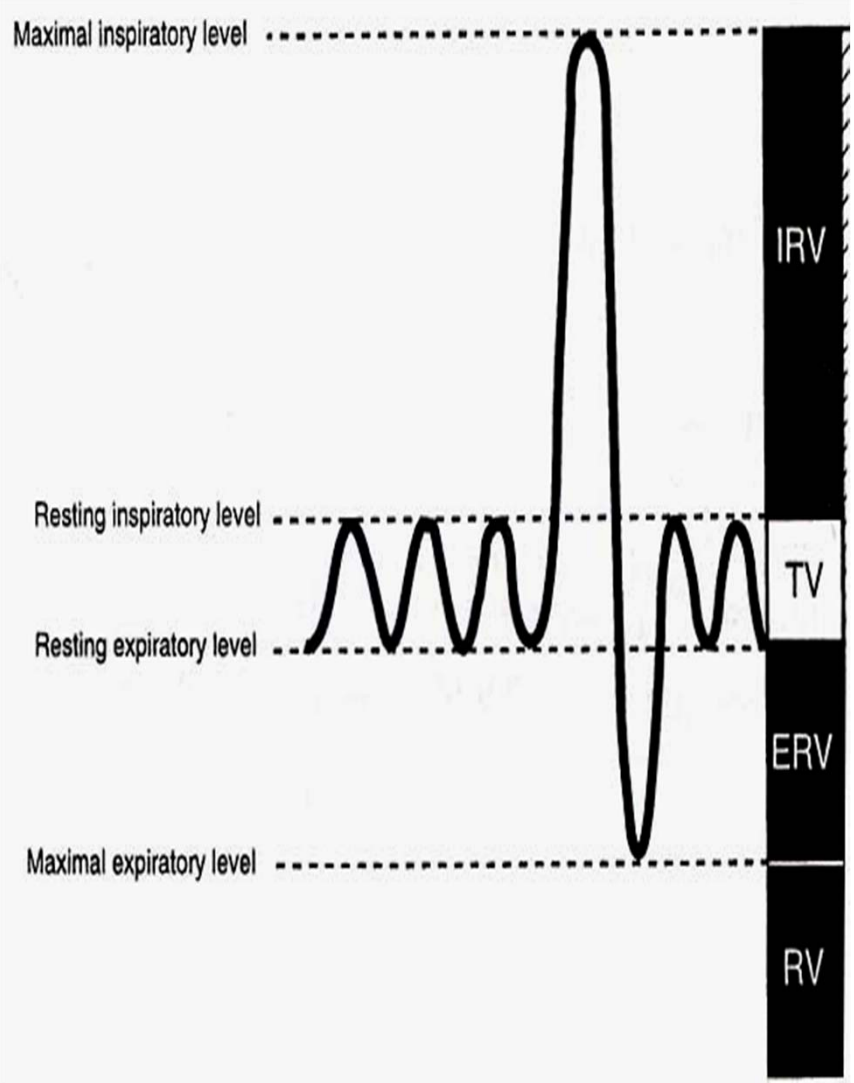
- Vital capacity
- Inspiratory capacity
- Functional residual capacity
- Total lung capacity

LUNG VOLUMES



- Tidal Volume (TV): vol. of air inhaled or exhaled with each breath during quiet breathing. N – **500ml**
- Inspiratory Reserve Volume (IRV): maximum volume of air inhaled from the end-inspiratory tidal position. N ~**3000ml**
- Expiratory Reserve Volume (ERV): maximum volume of air that can be exhaled from resting end-expiratory tidal position. N ~**1100ml**

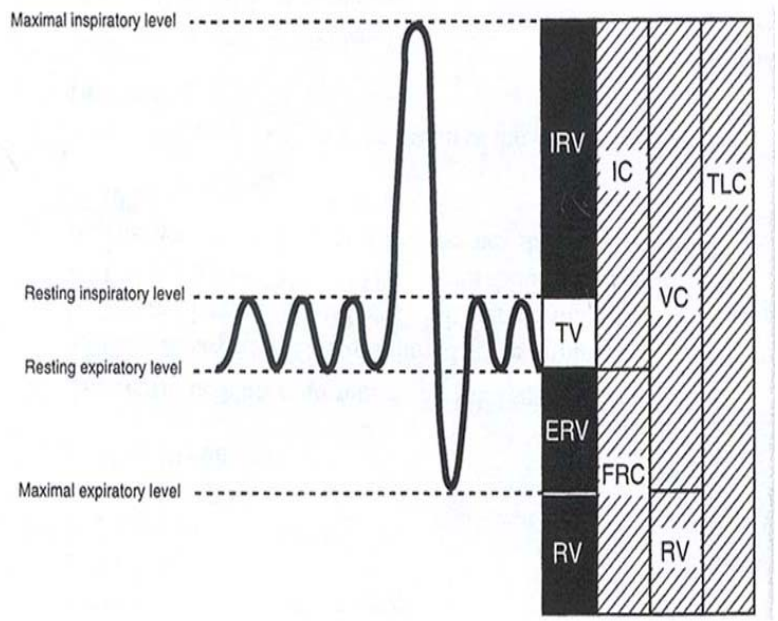
LUNG VOLUMES



- Residual Volume (RV):
- Volume of air remaining in lungs after maximum exhalation. N ~1200ml
- Indirectly measured (FRC-ERV) not by spirometry

N ~**1100ml**

LUNG CAPACITIES



- Total Lung Capacity (TLC): - volume of air in lungs after maximum inspiration
  - Sum of all volume compartments

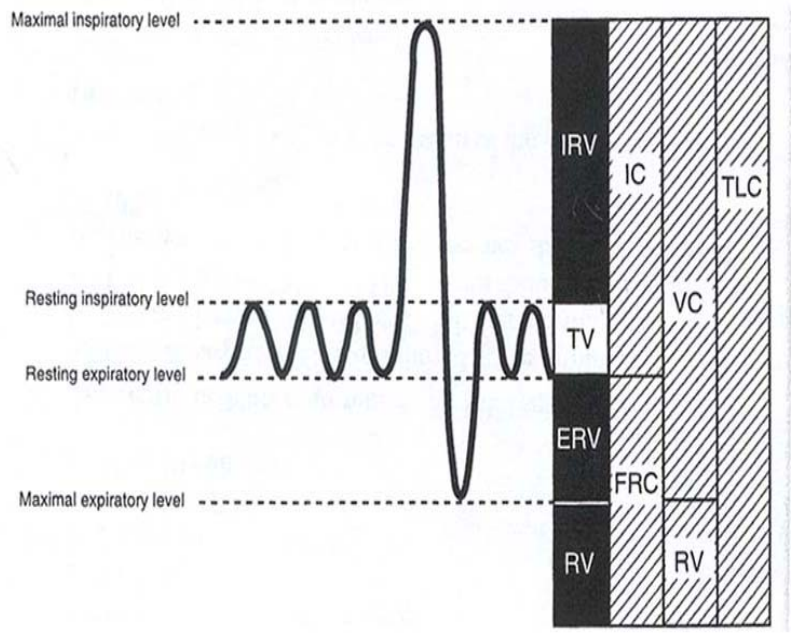
- Vital Capacity (VC): maximum volume of air exhaled from maximal inspiratory level

  VC = TLC - RV

- Inspiratory Capacity (IC): maximum volume of air that can be inhaled from the end-expiratory tidal position

  IC = IRV + TV

LUNG CAPACITIES



Functional Residual Capacity (FRC):

- Volume of air in the lungs at end-expiratory tidal position
- FRC = RV + ERV
- TLC, FRC, RV measured by:
  - Helium dilution
  - Body plethysmography

RESP. FUNCTION DURING ANAESTHESIA

- Anesthesia causes impairment in pulmonary function, whether pt. is breathing spontaneously or is ventilated mechanically

- Impaired oxygenation of blood occurs during anaesthesia, hence FiO2 is maintained at 0.3-0.4

- Clinically significant pulmonary complications is seen 1-2 % after minor surgery & upto 20% after upper abdominal & thoracic surgeries.

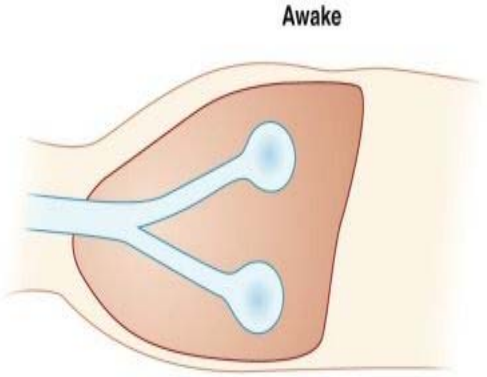
LUNG VOLUME & RESP. MECHANICS DURING ANESTHESIA

- **FRC is decreased around 20% of awake.**

  Loss of respiratory muscle tone.

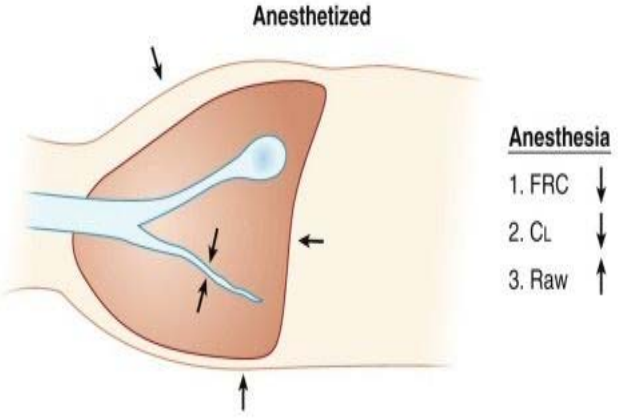
  Cranial shift of diaphragm

  ↓ in transverse diameter of thorax



- **Lung compliance is reduced**

  ↓ ed ventilation vol.

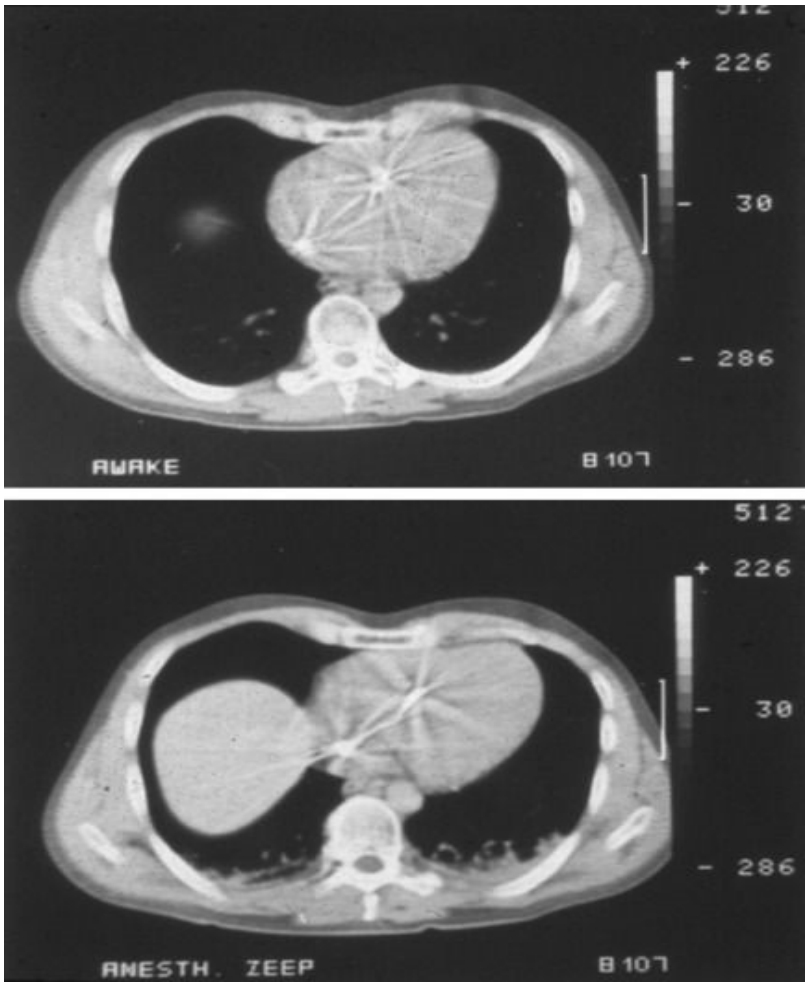


- **Resistance is increased**.

  ↓ ed airway dimensions

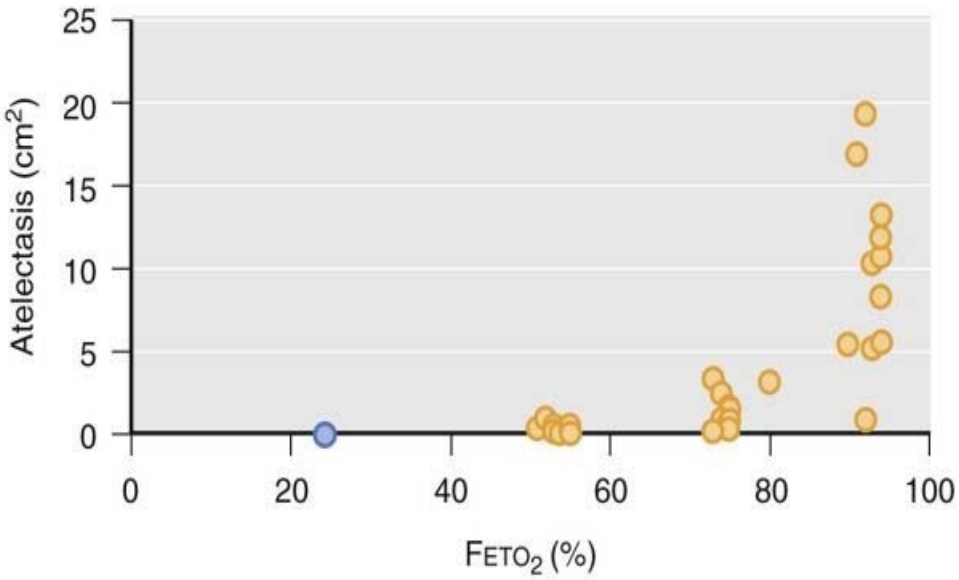
ATELECTASIS DURING ANAESTHESIA

- Seen in 90% of anaesthetized pts.
- Both in spontaneous breathing & after muscle paralysis.
- Development depends on preoxygenation, FiO2 during surgery, PEEP, postanaesthesia O2, BMI.
- Obese pt. - larger atelectasis
- Independent of age



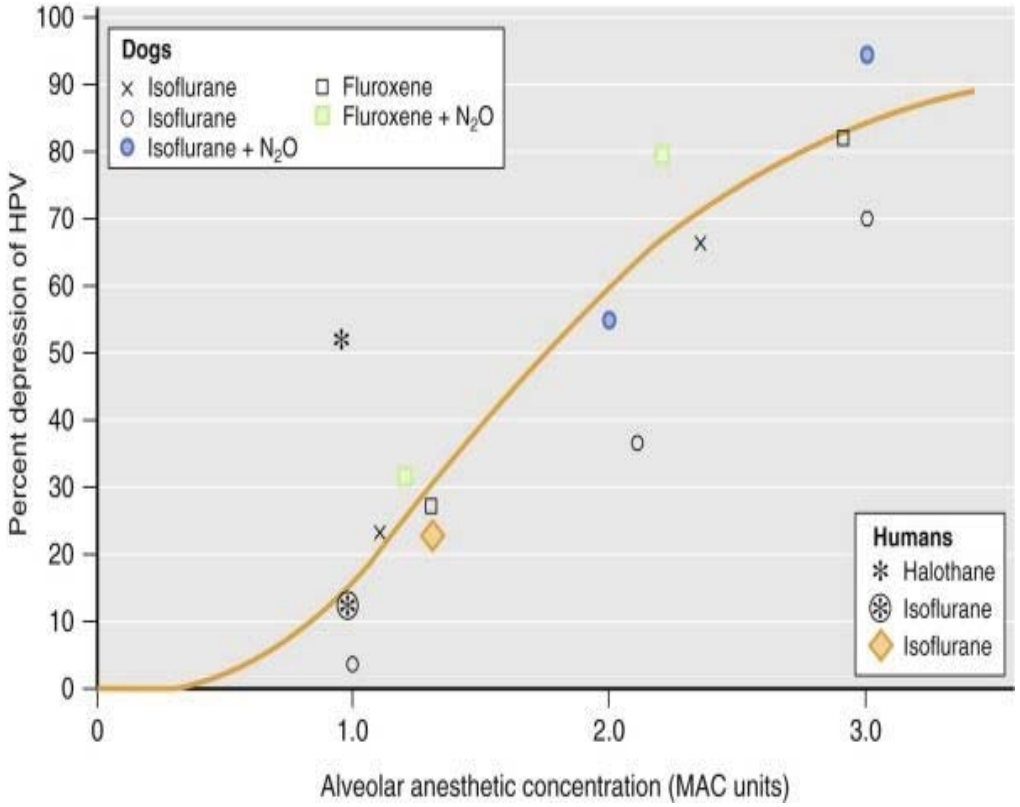
PREVENTION OF ATELECTASIS

- Application of PEEP
- Recruitment maneuvers
- Minimizing gas resorption- use of Low FiO2 during anaesthesia
- Use of low FiO2 in postanesthetic oxygenation.



Hypoxic pulmonary vasoconstriction (HPV)

- Physiological mechanism that optimizes V-P matching and pulmonary gas exchange by diverting blood flow from poorly ventilated areas of lung



- Inhaled anaesthetics inhibit HPV

Thank you