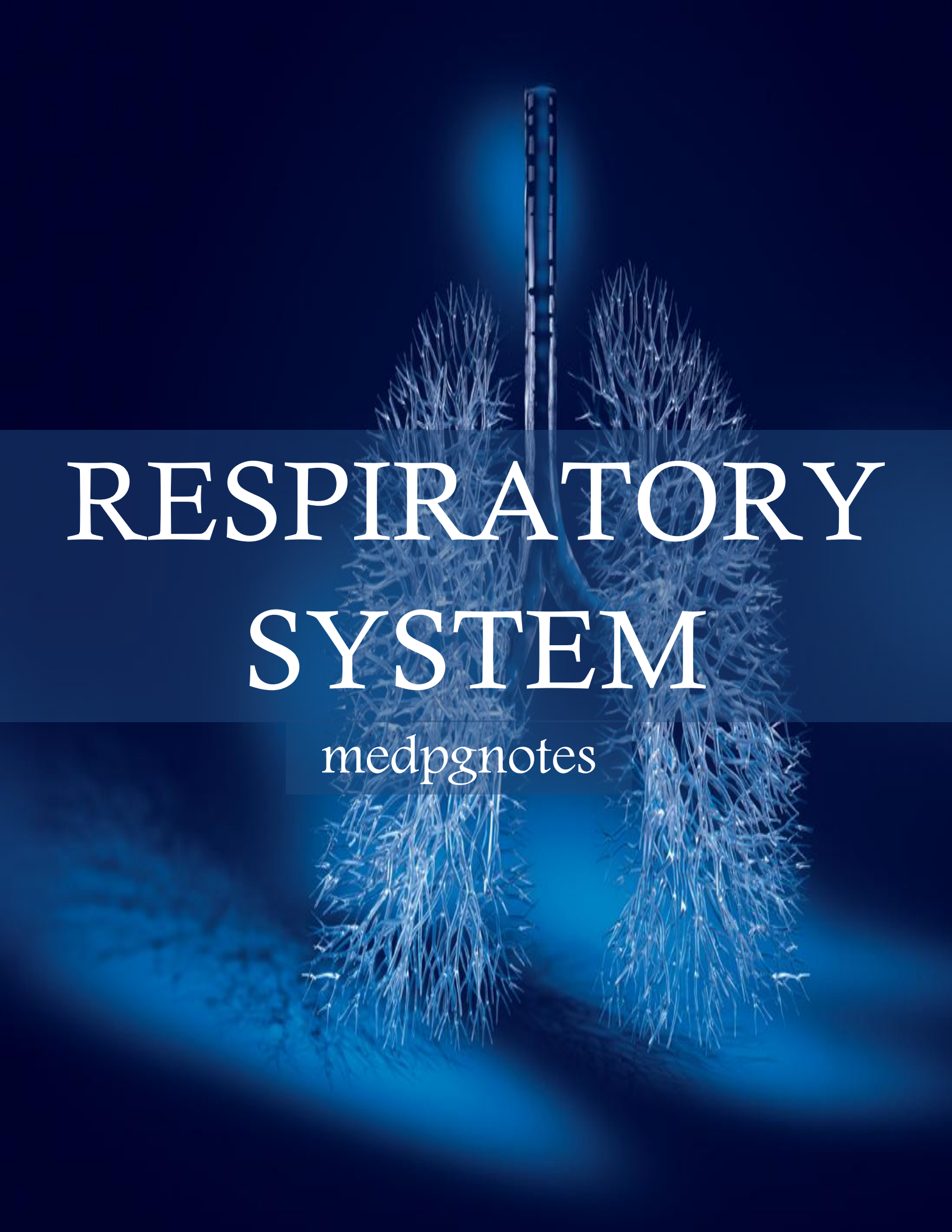

RESPIRATORY SYSTEM

medpgnotes

CONTENTS

RESPIRATORY SYSTEM

KEY TO THIS DOCUMENT

Text in normal font – Must read point. Asked in any previous medical entrance examinations

Text in bold font – Point from Harrison's text book of internal medicine 18th edition

Text in italic font – Can be read if you are thorough with above two.

DEVELOPMENT OF RESPIRATORY SYSTEM

Fetal respiratory movements	12 weeks
Respiratory bronchioles are formed during	*Pseudoglandular stage of lung development*
Common Lung anomalies	Pulmonary hypoplasia, Foregut cysts, Pulmonary sequestration
Bronchopulmonary dysplasia is seen with	Prematurity, Barotrauma, Oxygen therapy
Long term complications of bronchopulmonary dysplasia	*Small airway disease, decreased FRC, interstitial lung disease*
Use of steroids in neonates is required in	Bronchopulmonary dysplasia

ANATOMY OF RESPIRATORY SYSTEM

Respiratory cilia	*9 microtubular doublet with central singlets (9 + 2)*
Number of rings in trachea	*16 to 17 rings*
Length of trachea	*10 – 15 cm*
Diameter of trachea	*1.2 cm*
Right hilum is	*Lower than left*
Inferior most structure in right hilum	Inferior pulmonary vein
Hilum of right lung is arched by	Azygous vein
Mediastinal Surface of Right lung is associated with	Superior vena cava
Hilum of left lung is arched by	Arch of aorta
Uppermost structure in left lung hilum	Pulmonary artery
Most cranial structure in root of left lung	Pulmonary artery
Bronchopulmonary segment	Surgically resectable, Named according to segmental bronchus supplying it, It is drained by INTERsegmental branch of pulmonary vein, Largest subdivision of a lobe, First segment drains more than 1 pulmonary vein
Number of bronchopulmonary segments in right lung	*10*
Number of bronchopulmonary segments in left lung	*9*
Segment absent in left lung	*Medial basal segment*
Eparterial bronchus is NOT present in	*Left lung*
Parts of lower lobe of lung	Superior, Medial basal, Posterior basal
NOT a part of lower lobe of lung	Anteromedial basal
Lingual	Left upper lobe
Pulmonary segments in middle lobe of right lung	Medial, Lateral
In lungs, bronchial arteries supply bronchopulmonary tree	Till respiratory bronchioles
Bronchial arteries supply bronchopulmonary tree till	Respiratory bronchioles
Blood supply of lungs	Pulmonary artery, Pulmonary vein, Bronchial artery
Blood supply of Lungs	Two bronchial veins on each side
Sensory supply of trachea	Vagus

Pulmonary plexus	*Cell bodies of post ganglionic parasympathetic fibres*
Normal diameter of Trachea	2 – 6 cm
Lining cells of alveoli	Kulchitsky cells, Clara cells, Brush cells
NOT lining alveoli	Langerhan cells
MC cells in bronchoalveolar lavage	Macrophages
Clara cells are found in	Terminal bronchioles
Clara cells in bronchoalveolar lavage seen in	Bronchoalveolar carcinoma
Lepidic pattern	*Bronchoalveolar carcinoma*
Variants of bronchoalveolar carcinoma	*Clara cell, mucinous, type II pneumocyte*
Canals of Lembert in alveolar spaces in lung	Bronchoalveolar connections, Prevent atelectasis, Delay in collapse
Pleural reflection on left mid axillary line is in	10th intercostal space
Pleural extends up to which rib in mid axillary line	10
Pectus carinatum	**Pigeon chest**
Pectus excavatum	**Funnel chest**
Pectus excavatum	Decrease in lung capacity, Cosmetic deformity, Depression in chest
Pectus excavatum	Inferior part of sternum depressed in
NOT true about pectus excavatum	Gross CVS dysfunction

PHYSIOLOGY OF RESPIRATORY SYSTEM

GENERAL FEATURES OF RESPIRATORY PHYSIOLOGY

Normally lungs are kept dry by	*Osmotic pressure in interstitium*
Normal intrapleural pressure	*-3 to -5 cm of H2O*
Small airways have laminar flow because	Extremely low velocity
Cough receptors is seen in	Trachea
Type of receptors in bronchial smooth muscle	Beta 2
Normal intrapleural pressure is negative because	Chest wall and lung recoil in opposite direction
Negative intrapleural pressure is maintained by	Absorption lymphatics
Negative intrapleural pressure is maintained by	Lymphatic drainage of pleura
Intrapleural pressure is negative during both inspiration and expiration because	Thoracic cage and lung are elastic structure
Inflated state of lung in maintained by	*Negative intrapleural pressure*
A person is having normal lung compliance and increased airway resistance. Most economical way of breathing	Slow and deep
Effort during normal respiration is due to	Lung elasticity
During inspiration, intrapleural pressure	More negative
Airway obstruction if auscultation over trachea during forced inspiration	Breath sounds more than 6 seconds
Normal expiration	At the end of normal expiration of air in lungs is ERV
Greatest proportion of airway resistance	*Mid stem bronchi*
More resistance in expiration is due to	Increased compression of airway
Increased airway resistance due to	Forced expiration, Dense air, Low lung volume

Respiration stops in last stage of expiration, in forced expiration because of	Dynamic compression of airway
Neutral position of Chest	End expiratory
Plateau pressure	**End expiratory pressure**
Measurement of intravascular pressure by a pulmonary catheter should be done	At end expiration
Water fall effect in	*Middle lung*
Blood flow to apex of lung during	*Systole*
Flow volume curve in RS	Extrathoracic obstruction
Child's respiratory physiology differs from adult because of	Smaller airways
WRONG statement about Compliance	Compliance is affected only by surfactant
Pulmonary circulation	Hypoxia cause vasoconstriction, Blood volume in lung is 450 ml, Low resistance
Pulmonary circulation differ from systemic circulation	Pulmonary vasoconstriction in hypoxia, Resistance low, Capillary pressure low
Lung circulation	V/P ratio is 0.8 at rest, In apex ventilation is less than base
Pulmonary vascular resistance is decreased by	*Increase in cardiac output*
Bronchial circulation	Contribute 2% of systemic circulation, NO gaseous exchange, Causes venous admixing of blood, Provide nutritive function to lung
Recruitment is seen in	*Lung*
During heavy exercise, cardiac output increases up to five fold while pulmonary arterial pressure is very little. Physiological ability of pulmonary circulation is best explained by	Increase in number of wide open capillaries
Pulmonary circulation in hypoxia	Vasoconstriction
Vascularity of lung	Distended pulmonary veins in lower lobe
NOT true about lung circulation	Decreased vital capacity in supine position, Most blood in pulmonary capillary
Physiological dead space in lung	Zone 1
Physiological dead space	150 ml
Normal ratio of physiological and anatomical dead space	1:1
Anatomical dead space by	*Single breath nitrogen curve*
Best known metabolic function of lung	Conversion of angiotensin I to angiotensin II
Important non respiratory function of lung	Sodium balance

INSPIRATION AND EXPIRATION

Lattisimus dorsi used in	Forced EXPIRATION
Inspiratory muscles	*Diaphragm, external intercostal*
Accessory muscles of inspiration	Serratus anterior, Serratus posterior, Scalene
Muscle that does NOT contract during forced expiration	*External intercostalis*
Discharge spontaneously during quiet breathing	*Inspiratory neuron*

SURFACTANT

Surfactant is produced by	Type II pneumocytes
Size and number of inclusions in type II alveolar epithelial cells producing surfactant is increased by	*Thyroxine*
Accelerates maturation of surfactant in lung	*Glucocorticoid*
Surfactant production in lungs start at	28 weeks
Surfactant is made up of	Phospholipid
Major constituent of Surfactant	Dipalmityl Phosphotidyl Choline
Action of surfactant in human body is done by	Lipid and protein
Hyaline membrane contains	Fibrin
Functions of surfactant	*Increases compliance of lung, reduces surface tension of alveolar fluid, prevents collapse of alveoli*
Mechanism of action of surfactant in alveoli	Break the structure of water in alveoli
Pulmonary surfactant	Maintains alveolar integrity
Stability of alveoli is maintained by	Increase in alveolar surface area by surfactant
Stability of alveoli maintained by	Reduced surface tension by surfactant
Blood air barrier	Type II pneumocytes

GASEOUS EXCHANGE

Oxygen cascade	*Oxygen cascade describes the process of declining oxygen tension from atmosphere to mitochondria*
In a normal healthy person, arterial oxygen is considered satisfactory if spO2 is more than	*90%*
When blood passes through systemic capillaries	Increased protein content, Increased hematocrit, Decreased Ph, Shift of hemoglobin dissociation curve to right
PCO2 in atmospheric air	0.3 mm Hg
Alveolar CO2	40 mm Hg
Arterial carbon dioxide level	40 mm Hg
Least pCo2	Arterial blood
In alveolar gas, Mixed venous PCO2 is more than	Alveolar PCO2
Movement of CO2 from pulmonary capillaries to alveoli	Simple diffusion
CO2 diffuse more easily than O2 because	More soluble in plasma
CO2 is primarily transported in blood as	Bicarbonate
Percentage of O2 carried in chemical combination	97%
$PAO2 = FiO2 * (PB - PH2O) - \frac{PaCO2}{R}$	Alveolar gas equation, Barometric pressure = 760 mm Hg, Water vapor pressure = 47 mm Hg, Respiratory quotient = 0.8
Alveolar gas pressure is equal to	Body surface pressure

Po2 in atmospheric air	
Partial pressure of O2 at atmospheric pressure of 760 mm Hg	160 mm Hg
Atmospheric pressure 760mm Hg. O2 = 21%, partial pressure of 02?	159 mm Hg
Po_2 In Alveoli	104 mm Hg
Normal value of PO2 in healthy man is	80 mm Hg
At attitude of 6500 m, atmospheric pressure is 347 mm Hg. inspired pO2	73 mm Hg
pO2 in pulmonary capillary	*97 mm Hg*
pO2 of aorta	*95 mm Hg (because of physiological shunt)*
Partial pressure of oxygen in venous blood	*40 mm Hg*
Concentration of O2 in blood 0.0025 ml, atm 760 mm Hg, approximate oxygen tension	80 mm Hg
Normal level of Oxygen in blood when hemoglobin is saturated with O2	20 ml/dl
Arterial blood O2 in ml of O2 per dL	19.8
Amount of dissolved oxygen transported in 100 ml of plasma in a subject breathing 100% oxygen at 4 ATA	9 ml
Additional amount of oxygen transported in 100 ml of blood in a subject breathing 100 % oxygen under hyperbaric conditions of 4 ATA compared to normobaric conditions (1 ATA)	6 ml
If hemoglobin is completely absent, amount of plasma for basal oxygen requirement	*83 L*
Gas used to measure diffusion in lung	CO
Fraction of inspired air in mouth to mouth respiration	0.16 (16%)
Respiratory quotient	**VCO2/VO2**
Respiratory quotient of carbohydrate	1
Non Protein Respiratory Quotient	0.75
Rupture of mucosal blood vessels of trachea	40 mm Hg
Venous admixture by	*Thebesian veins, high V/Q areas of lung, bronchial vein*

VENTILATION PERFUSION RATIO AND COMPLIANCE

Normal respiratory system compliance	*0.2 L/cm H2O*
Specific lung compliance is decreased in	Pulmonary congestion, Pulmonary fibrosis, Decreased surfactant
Specific lung compliance is NOT decreased in	Chronic bronchitis
Pulmonary ventilation	PaO2 is maximum at apex
Ventilation perfusion ratio is maximum at	Apex of lung
High oxygen tension in alveoli is due to	Ventilation perfusion mismatch
Function of shunt	**Perfusion of non ventilated lung**

HYPERCARBIA AND ALVEOLAR HYPOVENTILATION

Gradient of alvelolar arterial oxygen tension in Hypoventilation is	Normal
Gradient of alveolar arterial oxygen tension is increased in	Diffusion effect, Right to left shunt, Ventilation perfusion abnormality
Alveolar hypoventilation in	Bulbar poliomyelitis, COPD, Kyphoscoliosis
Hypoventilation	**Excess of plasma bicarbonate in absence of volume depletion**
NOT associated with alveolar hypoventilation	Lobar pneumonia
Management of hypoventilation	**NIPPV**
Hypercarbia is characterized by	Hypertension, Tachycardia, Mydriasis due to sympathetic stimulation
CO2 retention is seen in	Respiratory failure, Ventilator failure, Pulmonary edema, Drowning
Apnoea is	Cessation of respiration
Sleep apnoea, temporary pause for at least	10 seconds

HYPERVENTILATION

Hyperventilation caused by	Decreased pH in CSF, decreased plasma HCO3, increased adrenergic levels
Voluntary hyperventilation at rest is associated with	*Washing out of CO2, alkalosis, convulsions, decrease in arterial CO2 pressure, decreases H+ ion (increases pH)*
NOT a cause of hyperventilation	CO poisoning
Initial change after Hyperventilation	Decreased PCo2 with Increased pH
In hyperventilation	P50 decreases and O2 affinity increases
Reduction in arterial oxygen tension caused by	Hypoventilation
Arterial blood gas determination in hyperventilation shows	Reduced PCO2
After hyperventilation for some time holding breath is dangerous, due to	*Lack of stimulation by CO2, anoxia can go into dangerous level*
Alveolar O2 tension is	Increased by hyperventilation
Solubility of CO2 is	20 times than that of O2

HIGH OXYGEN TENSION

Hyperbaric oxygen is dangerous because it	Is toxic to tissues
Toxic effects of high oxygen tension	Pulmonary edema, Retinal damage, CNS excitation and confusion
NOT an effect of high oxygen tension	*Hyperthermia*
Decreased cerebral blood flow in high oxygen tension is	Protective effect

HYPOXIA

O2 content of arterial blood	19.4 ml/100 ml
Decrease in respiration causes	Decreased pH + Increased PCO2
Hypoxia	When it is severe, it causes stimulation of sympathetic nervous system, It leads to accumulation of hydrogen and lactate ions, If it is chronic, causes rightward shift of oxygen Hb curve.
Hypoxia is characterized by	Intense chemoreceptor response, Low arterial PO2,favourable response to 100 % CO2
Tachycardia in hypoxia is due to	Diffuse vasodilatation
Hypoxia causes	Decrease in cerebral blood flow
MC physiological cause of hypoxemia	Hypoventilation
Most prone for hypoxic injury	Hippocampus
Neurons may get irreversibly damaged if exposed to significant hypoxia for	8 minutes
No stimulation of ventilation by hypoxia until pO2 falls below	*60 mm Hg*
Hypoxia does NOT cause vasodilatation in	Lung
Hypoxemia does NOT depend on	Hb
Variant of hypoxia NOT stimulating peripheral chemoreceptors	Anemic hypoxia
Anemic hypoxia is due to	Decreased O2 content in arterial blood
Best test for anemic hypoxia	Oxygen content or Hb%
Hypoxia seen in general anesthesia	Hypoxic hypoxia
Best parameter for analysis of hypoxic hypoxia	Arterial pO2
Condition leading to tissue hypoxic without alteration of blood oxygen content	Cyanide poisoning
Stagnant hypoxia is due to	Reduced blood flow
Best test for stagnant hypoxia	AV difference
Histotoxic hypoxia	**CO and cyanide**
Best test for histotoxic hypoxia	AV difference of PO2 of venous blood
Oxygen therapy is NOT effective in	*Histotoxic anoxia*
Does NOT used to prevent hypoxia	Pin Index
Stimulus for pulmonary vasoconstriction	Hypoxemia, Hypercapnia, Thromboxane
Pulmonary Vasoconstrictor	Low PaO2
Primary pulmonary hypoventilation	Does not respond to chemical stimuli

FEATURES OF HEMOGLOBIN

Hemoproteins	Cytochrome c, Cytochrome 450, Myoglobin, Hemoglobin, Catalase
Hemoprosthetic group is found in	Myoglobin, Cytochrome oxidase
Heme synthesis require	Ferrous ion, Glycine, Succinyl coA
Initially important for hemoglobin synthesis	Glycine
First step of heme synthesis	Glycine + Succinyl CoA
Key enzyme in heme biosynthesis	ALA synthase
Hemoglobin is a buffer because of	Histidine residue

Buffer NOT involved in non rapid achievement of Renal pH	Hemoglobin
Allosteric protein	Hemoglobin
Quarternary Structure	Hemoglobin
Hemoglobin is present in	Hydrophobic pockets
Hemoglobin structure	Hb has 4 polypeptide, Iron is present in ferrous state, Hb is structurally similar to myoglobin, Ferrous ions are in porphyrin rings
In hemoglobin, iron is bound to	Histidine
In hemoglobin, the innate affinity of heme for carbon monoxide is diminished by presence of	His E7
Function of histidine E7 in hemoglobin	*Hindered environment, protects CO poisoning*
Fe++ is attached to	*Histidine F8 of globin chain*
HbM	*Histidine F8 to tyrosine*
HbF	*Histidine 21 to serine*
HbS	*Glutamate for valine*
Hb Sydney	*Valine for alanine*
In lung R state favors	*Oxygenation (breaks salt bridge)*
T structure is stabilized by	2,3 DPG
Decreased glycolytic activity impairs oxygen transport by hemoglobin due to	Decreased production of 2,3 bisphosphoglycerate
Embryonal hemoglobin	Zeta epsilon
Type of hemoglobin with least affinity for 2,3-DPG	HbF
ADT test for	HbF
NOT true about fetal hemoglobin	Strong affinity for 2,3 DPG
Hemoglobin unlike myoglobin shows	Sigmoid curve of oxygen dissociation, Positive co operativity
Feature common to both hemoglobin and myoglobin	Heme at hydrophobic pockets
Each gram of hemoglobin carry	*1.39 ml of O2*
Carbon dioxide is carried in blood as	*Carbaminocompounds, dissolved gas, bicarbonate*

OXYHEMOGLOBIN DISSOCIATION CURVE

Amount of oxygen consumed per minute under basal condition	*250 ml*
Oxyhemoglobin dissociated curve is	**S shaped**
Oxyhemoglobin dissociation curve is sigmoid shaped because	Binding of one oxygen molecule increases the affinity of binding other O2 molecules
Oxygen dissociation curve is sigmoid in shape because of	Shifting affinity for Oxygen
Myoglobin dissociation curve	*Hyperbolic*
Myoglobin is	*8 alpha helix*
Myoglobin does NOT use oxygen	*Because p50 is low*
True about conversion of deoxy hemoglobin to oxyhemoglobin	Binding of O2 cause release of H+
Normal value of P50 on oxyhemoglobin dissociation	3.6 pKa

curve in an adult	
During exercise, increase in O2 delivery to muscle increase because of	Oxygen dissociation curve shifts to right, Increased stroke volume, Increased extraction of oxygen from blood, Increased blood flow to muscles
Role of 2,3-DPG	Unloading oxygen to tissues
Major role of 2,3-DPG	Release of oxygen
Feature of 2,3-DPG	Higher concentration in adult blood
Increase in 2,3-DPG seen in	Anemia, Hypoxia, Inosine
In anemia concentration of 2,3-DPG	Increased
Fetal hemoglobin has higher affinity for oxygen due to	Reduced 2,3 DPG concentration
Shift of Oxygen dissociation curve to right is by	Temperature, pH, DPG concentration
Oxygen curve shift to right	Decrease pH, increased temperature, increase in 2,3 DPG
Compound shifting curve to right	2,3 DPG
Shift to right in	Hypercarbia, Sickle Hb
Acidosis shift curve to	Right
Right shift in oxygen dissociation curve does NOT occur in	Transfusion
Oxygen dissociation curve does NOT shift to right in	Blood transfusion, Metabolic alkalosis
Does NOT shift ODC to right	Increased pH
Curve shift of left by	Increased oxygen affinity of hemoglobin
Increased pH causes O2 dissociation curve to	Left
What causes O2 curve to left	Decreased temperature
Oxygen dissociation in peripheral tissues is NOT altered by	Anemia
Does NOT influence dissociation curve	Chloride ion concentration
Oxygen affinity is increased by	Alkalosis, Increased HbF, Hypothermia
Oxygen affinity is NOT increased by	Hypoxia
Oxygen affinity is NOT increased in	Hyperthermia
O2 delivery to tissue does NOT depend on	Type of fluid administered
Decrease in affinity of hemoglobin when pH of blood falls	Bohr Effect
O2 delivery to tissue is decreased by	Decreased hemoglobin level, Decreased PaO2, Increased Ph

REGULATION OF RESPIRATION

Pacemaker of respiration	Pre Botzinger complex
Spontaneous rhythmic respiration is initiated in	Pre Botzinger complex
Rhythmic control of respiration lies at	Dorsal respiratory centre
Rhythm of Respiration is maintained by	Dorsal medulla
Most important stimulus of respiratory centre	Decreased PaO2
Respiratory centre is stimulated by	Hypercarbia
Respiratory centre	Inhibited during swallowing
Respiratory centre depression NOT caused by	Strychnine
Complete transaction of brain stem above the pons	*Prevent any voluntary holding of breath*
Section above pons inhibit	*Apneustic centre*
Pneumotaxic centre	Pons

Inhibition of Pneumotaxic centre causes	Prolonged Inspiratory spasm
Lesion of pneumotaxic centre	*Deep gasping as if tidal volume is high*
In cat apneustic centre is destroyed along with cutting of vagi	Prolonged inspiratory spasm
Lesion of prebotzinger complex	*Ondine curse (involuntary respiration is affected)*
Transection at mid pons level result in	Apneusis
Transaction at mid pons level with intact vagii	*Slow and deep breathing*
Apneusis is caused by	*Parabrachial nucleus and vagus*
What will be effect of respiration if transaction made between pons and medulla	Irregular and gasping
Lesion below medulla	*Total loss of respiration*
NOT a stimulus for pulmonary vasoconstriction	PGI2
Central chemoreceptors are most sensitive to	Increased PCO2
Chemoreceptor reflex primarily causes	Bradycardia, Vasoconstriction
Primary direct stimulus for excitation of central chemoreceptors	Increased H+
Central and peripheral chemoreceptors respond to	Increased arterial CO2
Peripheral chemoreceptors stimulated by	Hypoxia, Acidosis, Low perfusion pressure
Administration of pure O2 to hypoxic patients is dangerous because	Apnea occurs due to hypostimulation of peripheral chemoreceptors
Does NOT stimulate peripheral chemoreceptors	Hypocapnia
Does NOT stimulate peripheral chemoreceptors	Anemic hypoxia
Tidal volume excessive load is prevented by activation of	Bronchial stretch receptors
Inflation of lung induce further inflation	Head's paradoxical reflex
Herring Breuer inflation reflex	*Protective, involves pulmonary stretch receptor, inhibition of inspiratory centre*
Affect resting ventilation	Stretch receptors, Oxygen, PCO2
Does NOT affect resting ventilation	J receptor
J receptors are present in	*Pulmonary interstitium*
Stimulation of J receptors cause	*Apnea followed by tachypnea*
J receptor stimulation causes	*Apnea, hyperapnea, hypotension, bradycardia*
J receptor reflex	A.S.Paintal (India)
J receptor reflex	Sensitive to pulmonary congestion, Stimulated by Bradykinin
Lung reflexes are mediated by	*Myelinated nerve fibres*

LUNG VOLUMES, CAPACITIES AND ALVEOLAR VENTILATION

PFT	Total lung volume increases in emphysema, Compliance decreases in interstitial lung disease, Compliance is total lung distensibility
Best indication of alveolar ventilation is provided by measurement of	Tidal Volume
Volume of air taken in and given out during normal	Tidal volume

RESPIRATORY SYSTEM

respiration	
Tidal volume calculated by	Inspiratory capacity minus inspiratory reserve volume
Tidal Volume in both Men and women	500 ml
Resting tidal ventilation	5 L/min
Minimal tidal volume for adult resuscitation	600 ml
Maintenance of tidal volume	Bronchial stretch receptors
Expiratory reserve volume	*1000 ml*
Inspiratory reserve volume	*3300 ml*
Residual volume	*1200 ml*
Inspiratory capacity (TV + IRV)	*3800 ml*
Normal vital capacity (TV + IRV + ERV)	*4800 ml*
Functional residual capacity (ERV + RV)	*2200 ml*
Total lung capacity	*6000 ml*
Amount of air in lungs at the end of tidal breath	FRC
Volume of air in Lungs when respiratory muscles are at rest	Functional Residual capacity
Functional residual capacity is	Volume remaining of normal respiration
Functional residual capacity	ERV + RV
Normal functional residual capacity	*2.2 L*
Functional residual capacity is measured following	Normal expiration
At functional residual capacity, trans respiratory pressure system	**Zero**
Nitrogen washout method for	Functional residual capacity
During quiet inspiration, alveolar pressure	*0 cm H2O*
Alveolar ventilation	(tidal volume – dead space volume) X respiratory rate
Total alveolar volume in litre per minute	4.2
Alveolar Ventilation if an adult shows tidal volume 600 ml, dead space of 150 ml and respiratory rate of 15/min	6.75 L/min
Alveolar PaO2	100 – 120 mm Hg
FEV1	Forced expiratory volume in first second
FEV1 is	80% of Vital capacity
Instrument used for measuring vital capacity and FEV	Vitalograph
Vital Capacity	TV+IRV+ERV
Critical Closing volume is	Close to Residual Volume
Closing Capacity depends of	Dependent Small Airways
Breathing reserve	*Maximum breathing capacity – respiratory minute volume*
Hyaline membrane disease	FRC below closing volume
Decreased maximum mid expiratory flow rate indicates obstruction in	Small airway
Used to measure resistance to smaller airways	Mid respiratory flow rate
Total lung capacity depends on	Compliance of lung
Normal Vd/Vt ratio in adult	0.3
Better vision in video assisted thoracoscopic surgery created by	Collapse of Ipsilateral Lung
Spirometry used in diagnosis of	Asthma
Volume that can NOT be measured by spirometer	Functional Residual capacity
Spirometry does NOT measure	Residual volume
Routine spirometry can NOT measure	RV, FRC

In body plethysmography, a person is asked to expire against closed glottis. change in pressure in the lung and the box	Increase in lung and decrease in box
Man connected to body plethysmograph for estimation of FRC	inspired against closed glottis

ACCLIMATISATION

During acclimatization	Increase in minute ventilation, increased in sensitivity of central chemoreceptors, increase in sensitivity of carotid body to hypoxia
Features of acclimatization	*Polycythemia, increased diffusion capacity of lung, increased pulmonary ventilation, pulmonary vasoconstriction*
Earliest change in high altitude	*Hyperventilation*
pH and arterial pCO2 in a climber	*pH will rise and pCO2 will fall (respiratory alkalosis)*
Adaptation will be apt to increase the work capacity at high altitude	Decreasing work load, increasing duration of exercise
Mountaineer ascents 18000 feet in 2 days without supplemental oxygen.	Decreased barometric pressure, Decreased PaO2, Increased pH
Seen in high altitude climbers	Hyperventilation, Decreased PaCO2, Pulmonary edema
Feature of pulmonary edema in high altitude climbers	increased pulmonary capillary pressure, Normal left atrial pressure
Compensating mechanisms involved in acclimatization to altitude	Hyperventilation, Respiratory alkalosis
A person goes to mountains, when he reaches about 5000 feet, he develops dyspnea.	CO2 washout
Does NOT occur in High altitude acclimatization	Increased Blood glucose

MOUNTAIN SICKNESS

Acute mountain sickness is associated with	**Sleep desaturation**
Treatment of acute mountain sickness	**Acetazolamide**
Monge's disease	**Chronic mountain sickness**

CAISSON'S DISEASE

For every 20 meter depth	**3 atm pressure (1 atm due to atmosphere, 2 atm due to water level)**
Decompression sickness	**1 in 10,000 divers**
Decompression sickness seen in	Diver, pilot
Caisson disease	Gas embolism
Feature of Caisson disease	*Myonecrosis, paraplegia*
Pathological changes in Caisson disease is due to	N2
Main danger in deep sea divers is due to	Oxygen and nitrogen
Nitrogen narcosis is due to	Increased solubility of nitrogen in nerve cell membrane

SIGNS AND SYMPTOMS OF RESPIRATORY SYSTEM

GENERAL SIGNS AND SYMPTOMS OF RESPIRATORY SYSTEM

Hypoxemia	Hypoventilation, Decreased FiO2, Myasthenia gravis, Pulmonary emboli
MC fetal response to acute hypoxia	Bradycardia
Platypnea	Pleural effusion, Pulmonary embolism, Cirrhosis, COPD
Wheeze in children is caused by	Foreign body, Bronchial asthma
Bilateral rhochi	Pulmonary edema, Bronchiectasis, Emphysema
Rhonchi NOT in	Pulmonary embolism
Vocal Resonance is increased in	Consolidation
Vocal resonance is increases in	Lobar pneumonia, localized fibrosis of lung, cavity in apex
Cavernous respiration	Cavity
Consolidation	Trachea midline, Dull percussion note, Bronchial breath sounds, Increased vocal resonance
Tubular breathing	*Consolidation*
NOT a finding of consolidation	Dullness
Homogenous opacification of right hemithorax with right sided shift of mediastinum may be caused by	Collapse of right lung
Golden S sign	*Right upper lobe collapse*
Fever malaise, on examination tracheal shift to right side, VR, VPL heard, percussion note dull	Apical fibrosis
Emphysema COPD	INCREASED static compliance. Decreased dynamic compliance
Decreased static compliance	ARDS, Pulmonary edema, Interstitial fibrosis, Fibrosing alveolitis, Pulmonary congestion, Decreased surfactant
Wide Alveolar O2 gradient	ARDS, Bronchiectasis, Intestinal fibrosis
Bronchial hyperplasia caused by	Smoking, prematurity, allergy
Bronchial hyperplasia NOT caused by	Theophylline
Lung granuloma with necrosis	Tuberculosis, Histoplasmosis, Cryptococcosis, Wegener granulomatis
Bull's eye granuloma	**Pulmonary granuloma**
Large granuloma is seen in	Berylliosis, sarcoidosis
Bagassosis is most likely caused due to inhalation of	Sugar cane
MC cause of secondary tracheomalacia	*Aberrant innominate artery*
Sandstorm appearance on chest X ray	*Pulmonary alveolar microlithiasis*
Diffuse parenchymal lung disease	*Activation of macrophages leads to laying down of fibrous tissue and irreversible lung scarring*
Cause of pulmonary renal syndrome	Leptospirosis, Hanta virus, Paraquat poisoning
Pulmonary renal syndrome is seen in	Goodpasture syndrome, Leptospirosis, Hanta virus infection, Wegener's granulomatosis
APUD cells seen in	Bronchial carcinoid
Vascular ring causing external airway compression can be diagnosed by	Angiography of aortic and pulmonary circulation