

RESPIRATION

CH-1

Structure and function of resp system

Qn) 1) Respiratory and non-respiratory functions of lungs.

Ans: 1) Functions served by lung defence mechanisms

- IgA - Secreted in bronchial secretions and protect against respiratory functions
- Ciliary escalator - dust particles and bacteria caught in mucus layer of resp passages are moved up towards pharynx by rhythmic upward beating action of cilia
- Pulmonary alveolar macrophages } - defend against bacteria & virus by phagocytosis
- Cough reflex - laryngeal, tracheal and bronchial mucus membrane contain irritant receptors
→ expulsion of foreign material.

NOTE → Impaired ciliary motility seen in smoking, Kartagener's syndrome, cystic fibrosis.

2) Functions served by pulmonary circulation

- Reservoir for left ventricle
- Pulmonary vessels act as a filter
- lung contain heparin → remove small emboli from circulation

- Removal of fluid from alveoli
 - due to low hydrostatic P, fluid entering alveoli is absorbed by capillaries
- Role in absorption of drugs

3) Metabolic functions

- Synthesize surfactant
- Fibrinolytic system in lungs $\rightarrow$ lyses clot in pulmonary vessels
- Conversion of Angiotensin I $\rightarrow$ II is by ACE of capillary endothelium
- Secretion of histamine, kallikrein, PGs to systemic circulation
- Syn of biologically active amines $\rightarrow$ VIP, Sub P

4) Fun by resp muscles $\rightarrow$ during laughing and singing

5) Acid-base balance $\rightarrow$ maintain pH by regulating CO_2 content of blood

6) Water balance $\rightarrow$ water evaporates through expired air

7) Regler of body temp

8) Factory function

Exercise lower airway temp

$\downarrow$
Cool the airway

$\downarrow$
causes bronchoconstriction $\rightarrow$ asthmatic attack

NOTE

Factors affecting vital capacity

1) Size of thoracic cavity	More in males $\rightarrow$ large chest size and more muscle power than females
2) Age	Old age - VC $\downarrow$ due to $\downarrow$ in elasticity of lungs
3) Strength of resp muscles	Swimmers and divers VC more due to $\uparrow$ strength of resp muscles
4) Gravity	Standing $\rightarrow$ $\uparrow$ chest size $\rightarrow$ VC more
5) Pregnancy	VC reduced due to pushing of diaphragm (reduced size of thoracic cavity)
6) Ascites	VC reduced - pushing up of diaphragm
7) Pulmonary diseases	Pulmonary fibrosis, emphysema $\rightarrow$ decreased VC

▶ Residual volume (RV) $\rightarrow$ cannot be measured by spirometry

1) RV acts as buffer in b/w breaths to aerate the pulmonary capillary bed

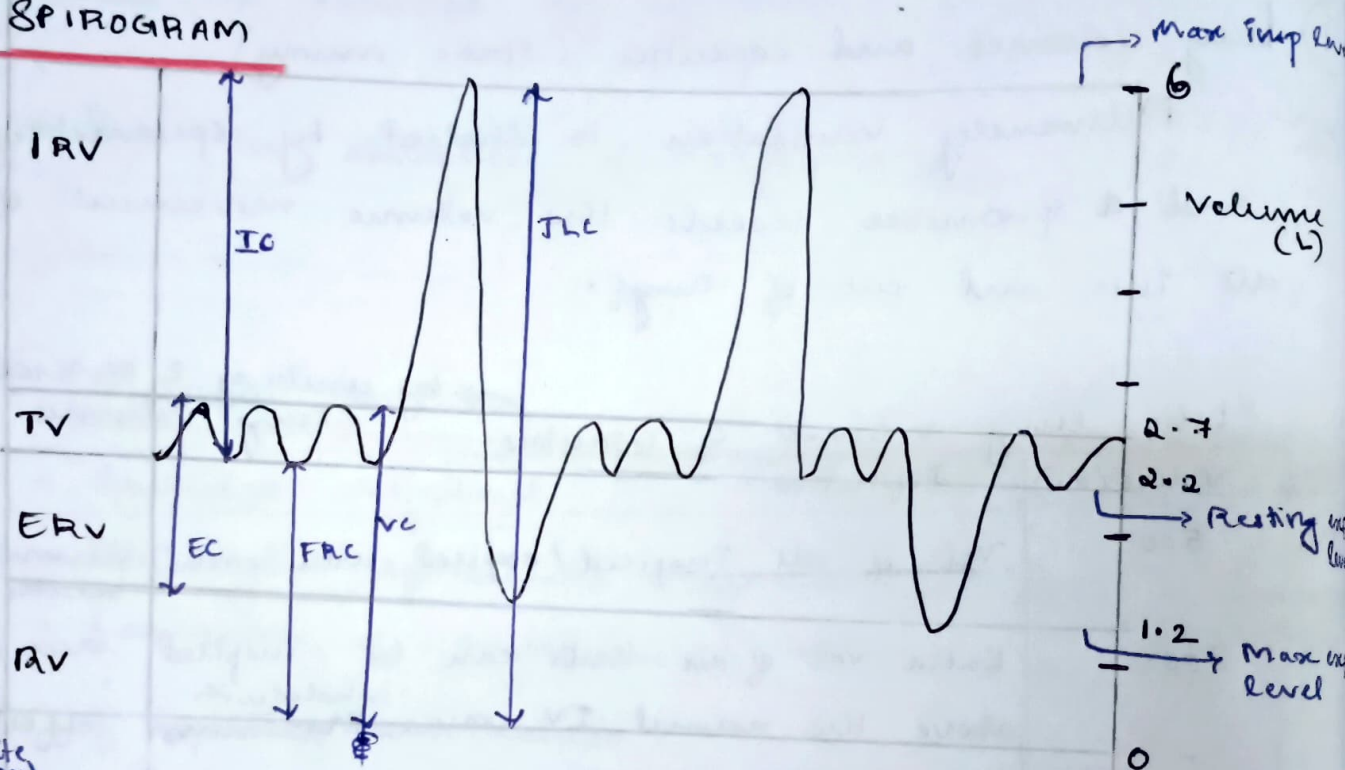
2) Medicolegal importance

- If the baby has taken 1st breath, minimal air volume will be there and lungs float in water.

- Some amt of air remains in lungs even after collapse of lungs because proximal airway collapse before the distal airways, trapping air.

The 5800 Vol of air present in lungs after maximum inspiration (VC + RV)

▶ SPIROGRAM



Write full form below

▶ Functional residual capacity (FRC) → can't be measured by spirometer

- 1) Continuous exchange of gases → maintains a constant concentration of O_2 and CO_2 in blood
- 2) Breath holding is possible
- 3) Risk in load on resp mechanism = without FRC, lungs would have been collapsed at end of expen

▶ Vital Capacity (Sheet note) FVC

- 1) Gives info about strength of respiratory muscles; $\therefore$ Maximal inspiratory and expiratory efforts can be assessed
- 2) It gives useful info about other aspects of function through FEV₁.
- 3) If VC ↑ es with treatment of a patient, that means the patient is responding to treatment.

20 Factors affecting vital capacity

- | | |
|-----------------------------|---|
| 1) Size of thoracic cavity | More in males $\rightarrow$ large chest size and more muscle power than females |
| 2) Age | Old age - VC less due to $\downarrow$ in elasticity of lungs |
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| 4) Gravity | Standing $\rightarrow \uparrow$ chest size $\rightarrow$ VC more |
| 5) Pregnancy | VC reduced due to pushing of diaphragm (reduced size of thoracic cavity) |
| 6) Asthma | VC reduced - pushing up of diaphragm |
| 7) Pulmonary disease | Pulmonary fibrosis, emphysema $\rightarrow$ decreased VC |

► Residual volume (RV) $\rightarrow$ cannot be measured by spirometry

1) RV acts as buffer in b/w breaths to re-oxygenate the pulmonary capillary bed

2) Medicolegal importance

- If the baby has taken 1st breath, minimal air volume will be there and lungs float in water.

- Some amt of air remains in lungs even after collapse of lungs because proximal airway collapse before the distal airways, trapping air.

- RV prevents lungs from collapsing at very low lung volumes.

2) DYNAMIC LUNG VOLUMES

Essay TIMED VITAL CAPACITY (Imp) / Forced ^{expiratory vol.} ~~Vital Capacity~~ (FEV)

- timed vital capacity or FEV is the fraction of forced VC (FVC) that is expired ^{forcefully} during 1st second (FEV₁), during 1st 2 seconds (FEV₂) and during 1st 3 seconds (FEV₃). ~~of forced expir.~~

Forced vital capacity (FVC)

- It is the largest vol of air that can be expired forcefully after a maximal inspiratory effort.

NORMAL VALUES OF FEV%

FEV% is fraction of vital capacity ^(not insp) expressed in %.

$$FEV_1\% = \frac{FEV_1}{FVC} \times 100 \quad (\text{similarly } FEV_2, FEV_3)$$

- FEV₁% → 83% of ~~total~~ FVC

- FEV₂% → 93% of FVC

- FEV₃% → 97% of FVC

Other dynamic lung volumes

Viva

1) Peak Expiratory Flow Rate (PEFR)

- Expiratory flow rate during peak of vital capacity
- Using :- Wright's Peakflow meter

IMPORTANCE

- 1) Reflects large airway fn
- 2) Bedside test
- 3) In obstructive lung diseases $\rightarrow$ marked redn

NORMAL VALUE $\rightarrow$ 400 - 450 L/min

2) Forced Exp Flow Rate / Max Mid Exp Flow rate

- Flow of air through middle 50% of forced expiration

IMPORTANCE

- 1) Reflects small airway fn
- 2) Bronchial asthma test

EQUATION BASED QNS

1) Minute ventilation / Pulmonary ventilation

- Vol of air insp/exp in 1 minute

$$\begin{aligned} \text{MV} &= \text{TV} \times \text{RR} \\ &= 500 \times 12 / \text{min} \end{aligned}$$

$$\boxed{\text{MV} = 6000 \text{ mL/min}} = 6 \text{ L/min}$$

2) Alveolar ventilation

- Vol of air ventilating alveoli / gas exchange zone in 1 minute

$$\begin{aligned} \text{AV} &= (\text{TV} - \text{Dead space}) \times \text{RR} \\ &= 350 \times 12 \end{aligned}$$

$$\boxed{\text{AV} = 4.2 \text{ L/min}}$$

3) Maximum voluntary ventilation (MVV)

- Max vol of air that can be moved in and out of lungs by maximum voluntary effort

$$\text{Normal} : \boxed{80 - 170 \text{ L/min}} \text{ Avg} = 100$$

4) Pulmonary reserve / Breathing reserve

$$\text{PR} = \text{MVV} - \overset{\text{MV}}{\text{Pulmonary}}$$

$$= 100 - 6$$

$$\boxed{\text{PR} = 94 \text{ L/min}}$$

5) Dyspnoeic index $\rightarrow$ to find dyspnoea

$$\bullet \text{ DI} = \frac{\text{PR}}{\text{MVV}} \times 100$$

• Normal $> 70\%$

• If less $\rightarrow$ dyspnoea

1) Intrapulmonary pressure / Intra alveolar p.

- It is the pressure inside alveoli
- Alveoli are in connection with atmosphere at beg and end of insp & exp, so $p. \text{ alveoli} = p. \text{ atm} = 0$ (atm)
- During insp, (Mid) $\rightarrow$ I.P.P = -1 mmHg

Reason: Thoracic cage size $\uparrow$ es

$\downarrow$
Volume $\uparrow$ es

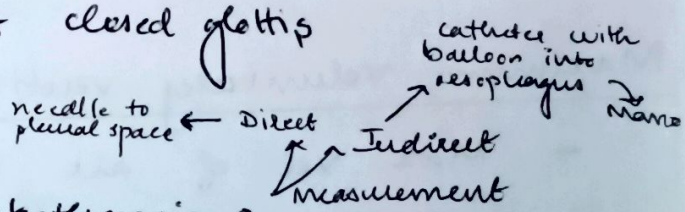
$\downarrow$
Pressure $\uparrow$ es (By Boyle's Law)

1) Müller's Manoeuvre $\rightarrow$ -80 mmHg

- Forced insp against closed glottis

2) Valsalva's manoeuvre $\rightarrow$ +100 mmHg

- Forced exp against closed glottis



2) Intrapleural pressure / Intrathoracic p.

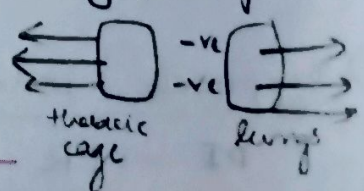
- Pressure b/w 2 layers of pleura
- It is subatmospheric (i.e., always -ve)

(due to elasticity and surface tension of pleura)

REASON: I) - Recoil prop of lungs is inward but
- Thoracic cage tends to expand

(When 2 surfaces move away from each other, -ve pressure is created which tends to suck and attract)

Eg: Water b/w 2 glass slides, when we try to separate them it is difficult)



II) More rapid drainage by pulmonary capillaries

1) Müller's manoeuvre $\rightarrow$ -40 mmHg

2) Valsalva's manoeuvre $\rightarrow$ +40 mmHg

Regional variation in pressure

- Apex $\rightarrow$ -6 mmHg
- Middle $\rightarrow$ -2.5 mmHg
- Base $\rightarrow$ -1 mmHg



$\downarrow \downarrow$ Lungs down in standing in standing posture
values at end of expiration

Normally (in middle)

- At beg of insp / end of expn $\rightarrow$ -2.5 mmHg
- At end of insp / beg of expn $\rightarrow$ -6 mmHg

Mechanism: Greater the expansion of lung



Greater the PE stored for recoil



Greater recoil $\rightarrow$ $\uparrow$ $-ve$ I pleural P

(Max expansion is at end of insp $\rightarrow$ $\therefore$ Recoil max $\Rightarrow$ Max $-ve$ IPP)

IMPORTANCE OF $-ve$ IPP

- 1) Increased venous return
- 2) maintains alveolar stability $\rightarrow$ prevents collapse
- 3) keeps airways open

CASES OF DIFF I pleural pressure

-2.5 to -6 mmHg	-4 to -1	0	$+ve$
Normal	less $-ve$ Emphysema, COPD	- At birth - Stab wound (without valve) $\downarrow$ pneumothorax	- Valsalva manoeuvre • defecation, micturition, child birth - Tension pneumothorax

Applied

1) Pneumothorax

- Air accumulates in pleural cavity
- Causes > 1 cm chest, chest wall injury etc
- Above conditions cause air to enter pleural space till

$$\text{Intrapleural } p = \text{APP}$$

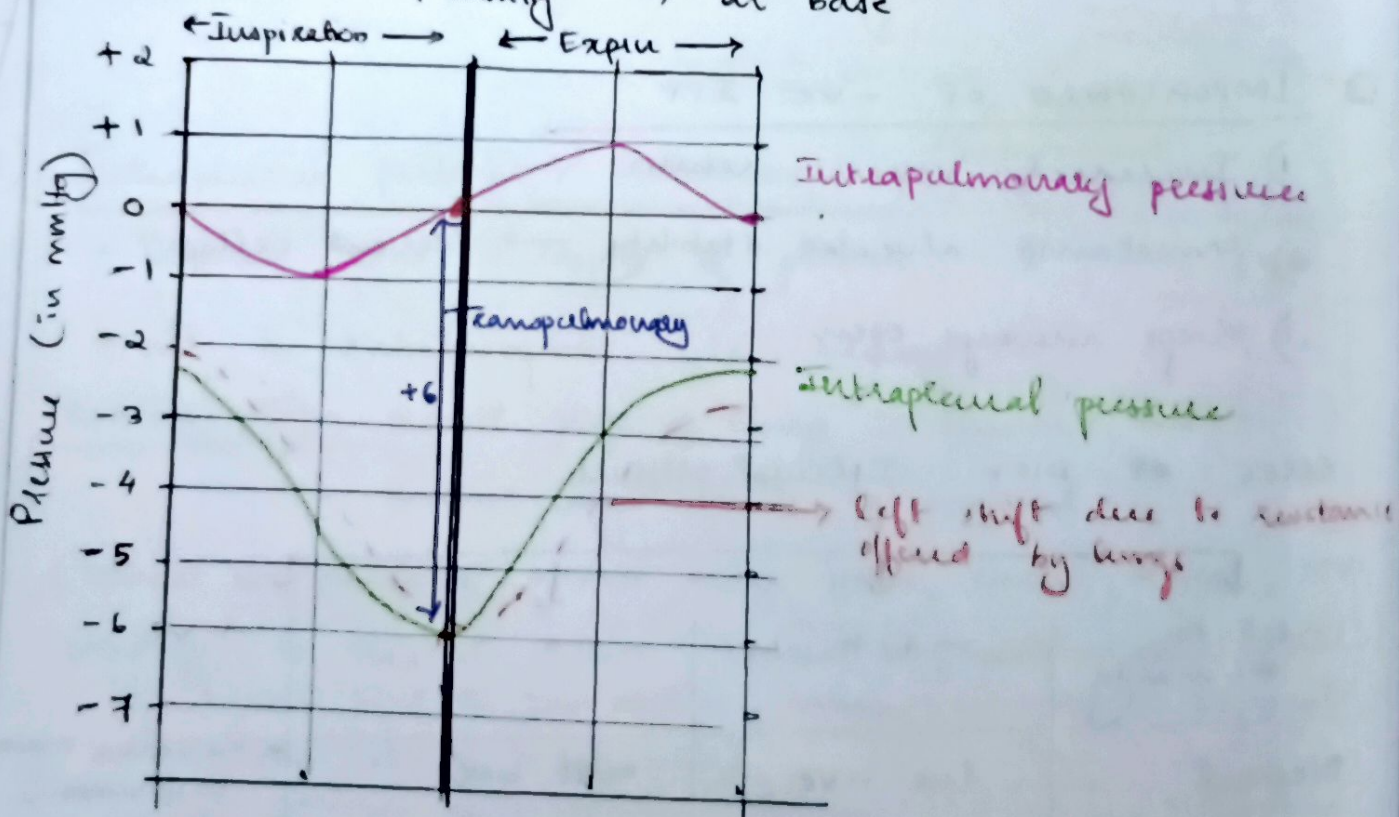
- Loss of -ve IPP $\longrightarrow$ collapse of lungs

3) Transpulmonary pressure

$$= \text{Intrapulmonary } p - \text{intrapleural } p$$

- Numerically same as intrapleural p (but +ve)
- It is the pressure that actually expands the lung

It is +6 mmHg $\longrightarrow$ at apex
-1 mmHg $\longrightarrow$ at base



2) Respiratory dead space

Portion of TV that does not take part in gas exchange. 2 types $\begin{cases} \text{Anatomical} \\ \text{Physiological} \end{cases}$

1) Anatomical dead space

- Vol of air trapped in resp tract from trachea to terminal bronchioles (except alveoli)
- Value = 150 mL/min

2) Physiological dead space

- Anatomical dead space + alveolar dead space

Alveolar dead space

- Vol of air in alveoli that do not take part in exchange
- It is due to $\rightarrow$
 - Alveolar dysfunction
 - diseases $\rightarrow$ emphysema, pulmonary embolism
- In normal adult,
Anatomical DS = Physiological dead space

Variation

- 1) Age - old age DS $\uparrow$ (due to loss of elasticity)
- 2) Sex - ADS is less in females
- 3) Posture - less in lying down posture

Measurement

- $\rightarrow$ ADS : Single breath N_2 washout method
- $\rightarrow$ PDS : Bohr's eqn

3) Compliance $\begin{cases} \text{total compliance} \\ \text{specific} \end{cases}$

- measurement of ease with which lungs can expand (distensibility)

- Stiffer lungs, less compliance. Eg:- Fibrosis

$$\text{Lung compliance} = \frac{\text{change in lung volume}}{\text{change in intrapleural p}}$$

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

- Normal : 0.2 L/cm of water pressure change

1) TOTAL COMPLIANCE

$\nearrow 0.2 \text{ L/cm}$ $\nearrow 0.2 \text{ L/cm}$
= Compliance of thorax + lungs

= 0.1 L/cm of water pressure change

NOTE :-

1) Lung compliance is more during expiration

2) To produce same volume, more pressure req in inspiration

↓

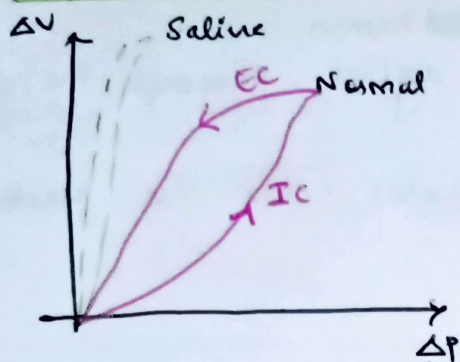
COMPLIANCE DIAGRAM $\rightarrow$ hysteresis loop

- relates lung volume changes to changes in pleural pressure

- IC curve lags behind EC curve

$\therefore$ hysteresis loop is formed

Graph



1) Lungs with air as interface
blow alveoli and lung
↓
Hysteresis

Reason

- Surface tension
- viscous and airway resistance

2) With saline interface

→ No hysteresis
(as no S.T.)

FACTORS affecting hysteresis

1) Gravity

- Apex less compliant as more lung vol present



2) Expir → compliance more

3) S.T. → ↑, comp ↓

4) lung vol → ↓, comp ↑

2) SPECIFIC COMPLIANCE

$$\bullet SC = \frac{\text{Compliance}}{FRC}$$

• It is the compliance / unit vol of lung

4) Surfactant

A. fluid

alveolar fluid

- surface tension lowering agent present in alveoli
- Synthesis : Type II alveolar cells in mitochondria

Composition

- 1) Mainly DPPC $\rightarrow$ dipalmitoyl phosphatidyl choline
- 2) Calcium
- 3) Carbohydrates
- 4) Proteins

FACTORS INFLUENCING PRODN

- 1) Hormonal : Glucocorticoids, Thyroxine stimulate
- 2) Insulin inhibits
- 3) Cigarette smoking $\downarrow$ surfactant

SIGNIFICANCE / Functions

- 1) $\downarrow$ S.T, $\uparrow$ compliance and $\downarrow$ work of breathing

• S.T. without surfactant - 50 dynes/cm²

S.T with surfactant - 5-30 dynes/cm²

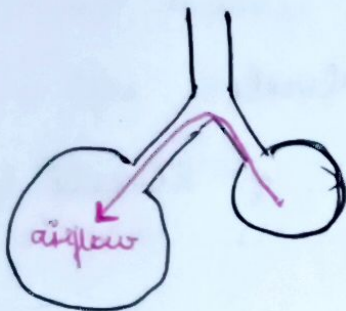
- By $\downarrow$ ing ST it makes expansion of alveoli easy and prevents collapse of (compliance $\uparrow$) lungs during expir.

2) Maintains alveolar stability $\begin{matrix} \swarrow \text{during inspiration} \\ \searrow \text{expiration} \end{matrix}$

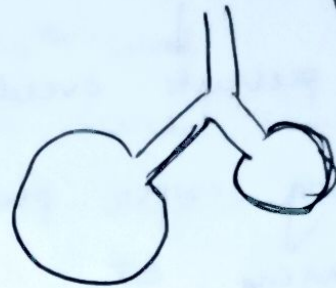
Law of Laplace

$$P = \frac{2T}{R} \quad \begin{matrix} \nearrow \text{Tension} \\ \text{in a distensible spherical organ} \\ \text{R} \rightarrow \text{radius} \end{matrix}$$

No surfactant



with surfactant

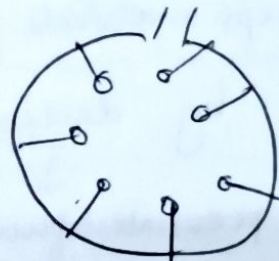


Small alveoli



ST $\downarrow$ well in small alveoli as surfactants are close

Large alveoli



ST $\downarrow$ does not happen much as surfactants are far apart

Eg: Let ST without surfactant be 50 mmHg.

Small alveoli

$$P = \frac{2 \times 50}{1}$$

$$= 100$$

Large alveoli

$$P = \frac{2 \times 50}{2}$$

$$= 50$$

This will result in P. difference. But this does not happen becoz surfactant pre

$$\therefore P = \frac{2 \times 25}{1}$$

$$= 50 //$$

$$P = \frac{2 \times 50}{2}$$

$$= 50 //$$

During inspiration

Alveolar diameter ↑



Surfactant molecules move apart



No. of surf molecules ↓ per unit area



ST not ↓ed much



Hence, prevents overdistention of alveoli

During expiration, prevents collapse of alveoli by decreasing ST.

3) Keeps alveoli dry / prevents pulmonary edema

1) - by decreasing S.T.



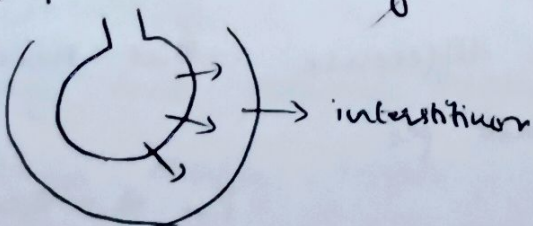
prevents fluid accumulation

REASONS other than surfactant

2) Inward pressure gradient (-ve p) in capillaries

3) -ve pulmonary interstitial fluid p,
sucks any fluid that collects in alveoli

4) Lymphatics in lungs drain excess fluid



5) low pressure circulation (pulmonary feature)

■ APPLIED PHYSIO - Infant Resp Distress Syndrome (IRDS) (Hyaline membrane disease)

- Infants (esp premature infants) born to diabetic mothers have def of surfactant in alveoli and there is $\downarrow$ $\downarrow$ $\downarrow$ ST.
- Surfactant normally appears at 28th week of gestation and mature just before 36th week.
- After birth, infant makes several strong inspiratory movements and lung expands. Surfactant keeps them from collapsing again.
- So def of surfactant causes collapsing of ^{alveoli} ~~lungs~~ (atelectasis) and pulmonary edema.

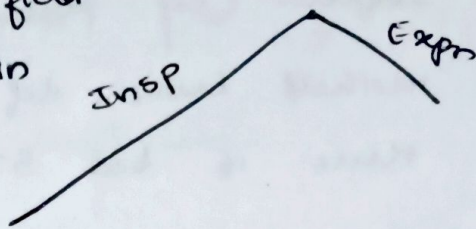
■ OTHER FACTORS CAUSING IRDS

- 1) low level of thyroid and glucocorticoids
- 2) • During fetal life, Cl^- is secreted with fluid by pulm epithelial cells.
 - At birth, there is shift to Na^+ absorption along with fluid (via ENaCs)
 - ∴ Prolonged ^{intensity} ~~abnormalities~~ ^{of} ENaCs can cause pulmonary abnormalities $\rightarrow$ like edema.

6) Breath sounds

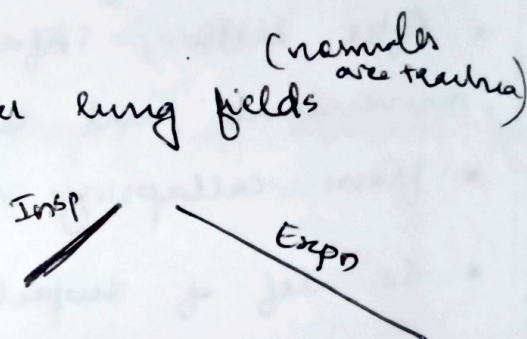
1) Vesicular

- Normal finding over lung field
- No gap b/w insp and expn
- Insp longer
- Quiet, low pitched, rustling



2) Bronchial

- Abnormal if auscultated over lung fields
- Gap b/w insp & expn
- Exp phase is longer
- Loud sounds



3) Added / Adventitious

- Wheeze / Rhengi
- dry, musical sounds Eg: In COPD
- Crepitation / crackles
- wet & bubbling sounds Eg: In pulm edema
- Pleural rub
- Rubbing produced by inflamed pleural surfaces
Eg: Pleurisy

6) Mechanics of Resp

INSPIRATION

• Active process

• Muscles involved

- 1) Normal quiet
- Diaphragm - phrenic n. (1, 4, 5)
 - Ext intercostals

2) Forced insp - Sternocleid, Serratus ant, scalene

• Movements :-

1) Vertical - diaphragm

- When diaphragm contracts, dome flattens
- Vertical diameter of thoracic cage ↑

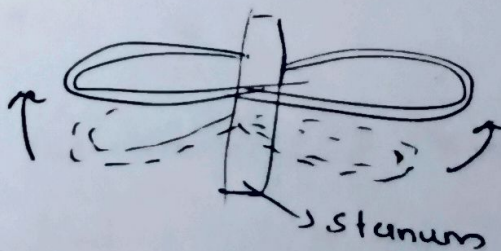
2) Anteropost - Sternum & 2-6 ribs

- by pump handle motion
- Contraction of external intercostals, pushing sternum ~~forward~~ outward and ↑ AP diameter



3) Transverse - (7-10) ribs

- Bucket handle motion



EXPIRATION

- In normal quiet breathing, it is ~~passive~~ (no muscles are actively involved)
- Muscles :-

1) Normal quiet → relaxation of insp muscles
→ elastic recoil of lungs

2) Forced expir → ant abdominal, internal intercostals
↓ ↓
↓ vertical diameter ↓ diameter of thorax
of thorax

Applied Physio

- Transection of SC above C_3 is fatal without artificial resp but transection below C_5 is not because it leaves phrenic n. that innervates diaphragm.

Pulmonary Circulation

SE

1) Ventilation - perfusion ratio

ANS: I) Definition

V/Q is the ratio b/w alveolar ventilation in 1 min and pulm perfusion in 1 min.

$$V/Q = \frac{4200 \text{ mL/min}}{5000 \text{ mL/min}} \approx 0.8 \text{ (middle of lung)}$$

Values :-

- Apex $\rightarrow 3$
- Base $\rightarrow 0.6$

II) FEATURES

NOTE

- Ventilation and perfusion are not uniformly distributed throughout lungs
- Gravity dependent edo is more marked in case of perfusion than ventilation.
- Apical alveoli $\rightarrow$ more underperfused than underventilated
 $\therefore \frac{V \uparrow}{Q \downarrow} \uparrow \text{ed} = 3 //$
- Middle $\rightarrow 0.8$
- Base $\rightarrow$ Alveoli are overperfused than over-ventilated
 $\therefore \frac{V}{Q} \downarrow \text{ed} = \underline{\underline{0.6}}$

III) Importance (clinical)

☐ Pulm tuberculosis affects apex of lung first. Bcz:-

A) high V/Q at apex

↓
↑ alveolar O_2 at apex
↓

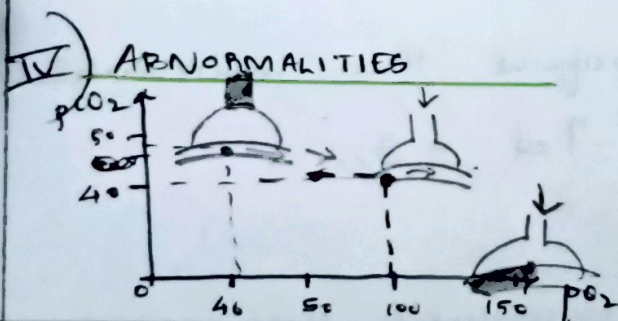
Mycobacterium tuberculosis can grow well (as it is aerobic)

B) Poor perfusion at apex

- Antibodies do not reach apex satisfactorily
- So, apex more vulnerable to bacterial attack.

OTHER IMP

- 1) Determining gas conc in alveoli
- 2) " effectiveness of gas exchange through alveolar capillary membrane
- 3) To assess if there is imbalance b/w alveolar ventilation and blood flow.



R/L Shunt	Normal	Dead space
$V/Q = 0$	$V/Q = 0.8$	$V/Q = \infty$
$pA_{O_2} = 45$	$pCO_2 = 40$	$pCO_2 = 0$
$pO_2 = 40$	$pO_2 = 100$	$pO_2 = 150$
- Impure blood goes to syst circs eg:- COPD, bronchial asthma, etc.		eg:- Pulm embolism, hyperventilation

1) Shunt (2)

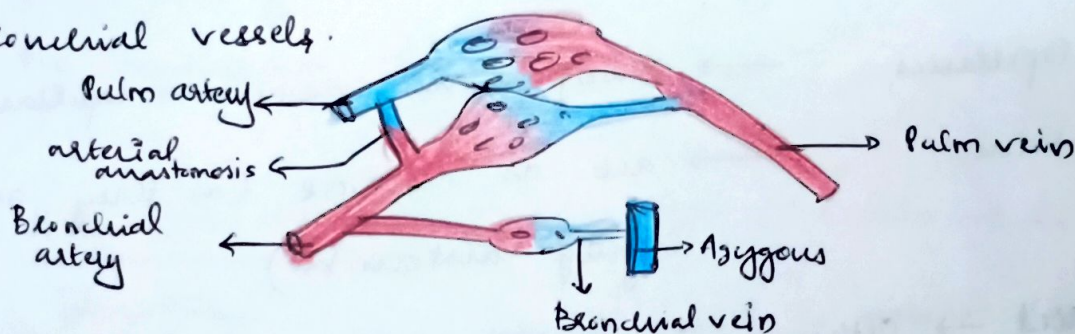
1) Physiological shunt

- is defined as any mechanism by which blood that has not been oxygenated in lungs is added to systemic circn.

CAUSES

1) Bronchopulm anastomosis

- anastomosis b/w capillaries of pulm vessels and bronchial vessels.



2) Some amt of coronary venous blood, which is drained from heart, enters left ventricle through thecorian veins. This constitutes 0.5% of blood in systemic circn.

2) Pathological shunt

Eg: R-L Shunt (septal defects) in patients with cyanotic congenital heart disease contribute to redn in systemic arterial P_{O_2}

Applied

- 1) Cause for redn in systemic arterial P_{O_2}
- 2) RV output is little less than LV output
- 3) as P_{O_2} ↓, saturation of Hb less in systemic arterial

2) Features of pulmonary circulation

- 1) Lung is the only organ receiving entire C.O.
- 2) Due to effect of gravity, regional perfusion is greater near base of lung than apex

3) Vessels :-

- Pulm arteries →
 - 1) thin-walled
 - 2) very little smooth muscles
 - 3) large diameter
- Capillaries → multiple anastomoses → capillary basket
- Veins → act as reservoir (as they are highly distensible)

Applied → Orthopnea in cardiac failure

- ^{In} Supine position (change in thoracic vol)

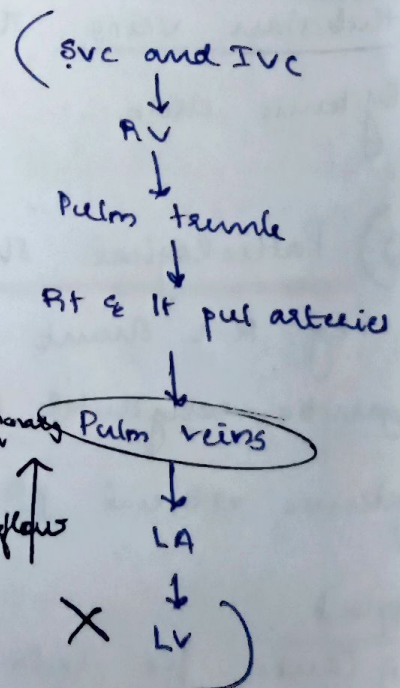
↓
↑ venous return

↓
Impaired LV can't handle extra preload

↓
↑ pulmonary venous pressure

↓
leads to transudation of fluid into alveoli → pulmonary edema

↓
Fluid in alveoli impairs oxygen diffusion → ↓ VC → causes distress of breath



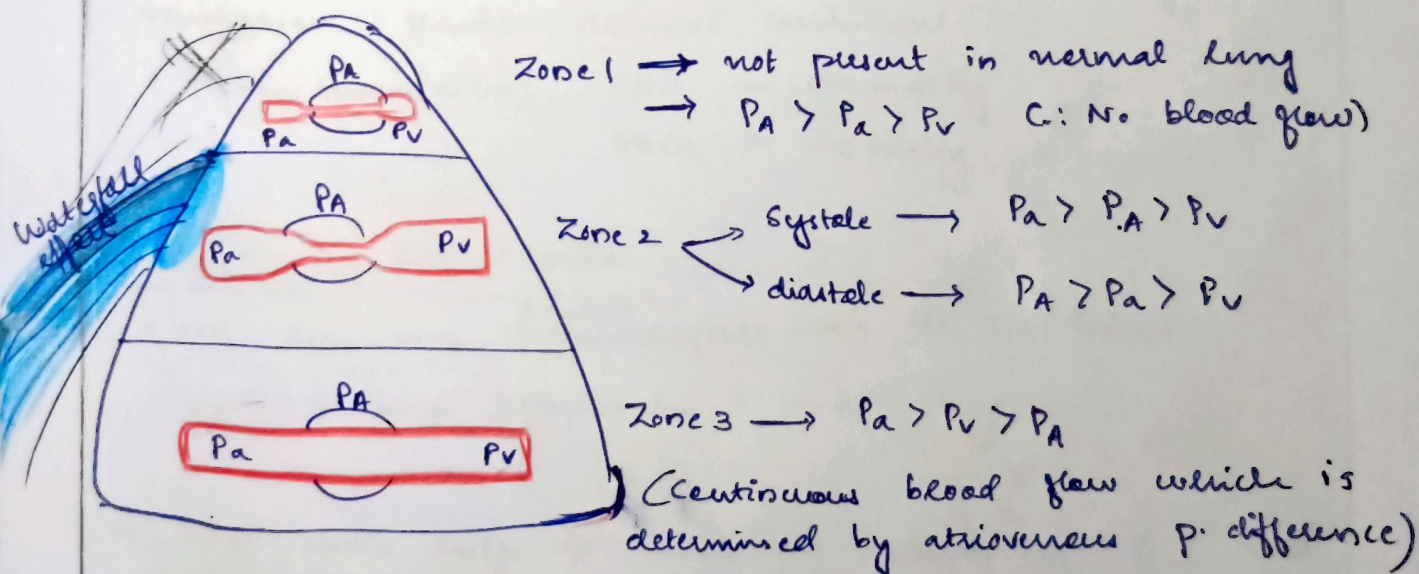
- Sitting up reduces venous return and promotes better lung expansion

4) Since the pulm vessels are highly distensible, pulm circn is a low-pressure, low-resistance, high capacitance system with high compliance.

5) Pulmonary circulation acts as a filter that prevents emboli from reaching systemic circn due to pres. of fibrinolytic system in lungs.

6) P.c maintains dryness of alveoli

2) REGIONAL VARIATION EXPLAINED (For circn $\rightarrow P_a > P_A > P_v$)



$P_a \rightarrow$ alveolar pressure

$P_A \rightarrow$ arterial p.

In Zone 1, think of a hose being squeezed in middle

- If outside p. (i.e. alveolar P) is stronger than inside blood p., the hose collapses and blood can't flow
- That's waterfall effect, blood won't flow unless arterial pressure overcomes alveolar pressure

other (other than gaseous)
7) Factors affecting pulm circulation

- 1) Nervous $\begin{cases} \text{symp} \rightarrow \text{vasoconstriction} \\ \text{par} \rightarrow \text{vasodilation} \end{cases}$
- 2) Chemicals $\rightarrow$ hypoxia, hypocapnia, acidosis $\rightarrow$ constriction
- 3) Hormonal $\begin{cases} \text{vasoconstrictors} \rightarrow \text{Ang II, epi, NE} \\ \text{dilators} \rightarrow \text{Ach, NO} \end{cases}$
- 4) Posture
- 5) $\text{CO} \propto \text{pulm flow}$
- 6) $\text{pulm flow} \propto \frac{1}{\text{vascular resist}}$
- 7) Inspiration $\rightarrow$ dilation
Expiration $\rightarrow$ constriction

CH-4

Pulmonary Gas Exchange

PA

1) How alveolar air is kept constant?

Ans - 1) Slow replacement of alveolar air

- Only about 350ml of air enters alveoli with each breath (TV - dead space)
- FRC $\rightarrow$ air left in lungs $\rightarrow$ 2300ml
- So, fresh air mixes slowly with a large volume of old air, leading to gradual change (not abrupt swings)
- This keeps O_2 and CO_2 conc with each breath

2) Diffusion balance across alveolar membrane

- Rate of gas exchange is matched to rate of alveolar ventilation under normal conditions.
- This maintains: $\text{PAO}_2 \approx 104 \text{ mmHg}$
 $\text{PACO}_2 \approx 40 \text{ mmHg}$

3) Regln by resp centres

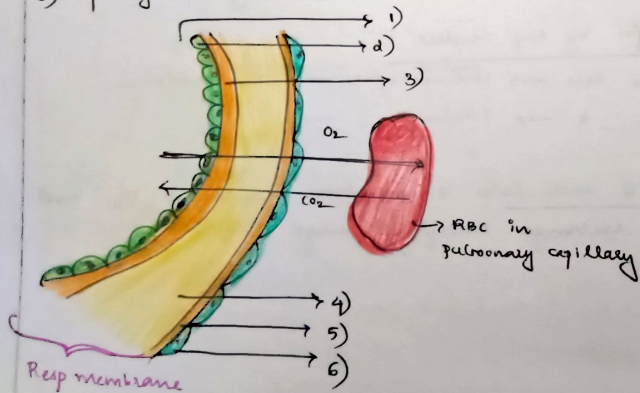
- CO_2 rise $\rightarrow$ ^{central} chemoreceptors $\rightarrow$ $\uparrow$ ventilation
- O_2 $\downarrow$ $\rightarrow$ peripheral $\rightarrow$ $\uparrow$ ventilation

4) V/Q ratio helps to maintain consistent O_2 and CO_2 exchange throughout lungs

2) Respiratory membrane / Alveolar capillary membrane (Blood-gas barrier)

- Membrane through which gas exchange occurs
- Forms blood-air interface that separates blood in pulmonary capillaries from air in alveoli.
- Total SA across which gas exchange occurs is 70 m²
- The resp membrane is very thin (0.2 - 0.4 μ m) and has the following layers (from air to blood)

- 1) A layer of fluid lining alveoli
- 2) Alveolar epithelium (Type-I pneumocytes)
- 3) Epithelial basement membrane
- 4) Interstitium
- 5) Capillary basement membrane
- 6) Capillary endothelium



3) Factors affecting diffusion across membrane

$$\text{Rate of Diffusion} \propto \frac{\Delta P \cdot A \cdot S}{d \cdot \sqrt{MW}}$$

thickness of memb

- 1) Pressure gradient (ΔP) across RM
 - Greater gradient for O_2
 - More gradient, more diffusion
- 2) SA of resp memb (A)
 - directly proportional
 - CA $\rightarrow$ In emphysema, there is destruction of alveolar wall, hence $\downarrow$ SA to $\frac{1}{3}$ of normal
- 3) Solubility of gas (S)
 - Although ΔP of CO_2 less than O_2 , (also O_2 has lower mol wt)
 - But CO_2 is 24 times more soluble
 - Hence, diffuses with ease.
- 4) Molecular wt of gas (MW)
 - inversely prop
- 5) Diffusion coefficient (D)
 - Rate of diffusion of a gas through a given area for a given dist for a given ΔP in unit time.
 - D of CO_2 is 20 times more than O_2 .

3) Diffusing capacity (DC) & Lung

- It is volume of gas in mL that is transported across cap membrane in time per 1mmHg ΔP

$$DC_x = \frac{A}{t} = \frac{dC_x}{\Delta P}$$

$DC_x \rightarrow$ DC of O_2

$dC_x \rightarrow$ diffusion coefficient

$A \rightarrow$ area of cap mem

$t \rightarrow$ thickness of membrane

Variations

i) DC is $\uparrow$ ed in exercise

Factors

- Opening up of closed capillaries (recruitment)
- Vasoilation
- Stretching of alveolar mem cause decrease in thickness of mem $\rightarrow$ more DC

ii) DC $\downarrow$ ed in pulm edema, fibrosis etc.

CH - 5

Transport of Gas

Essay

1) Transport of O_2 and CO_2

Ans: ∇ Transport of O_2

- I) Steps:-
- a) Uptake of O_2 in lungs by pulm blood
 - b) Transport of O_2 in arterial blood
 - c) Release of O_2 from blood to interstitial fluid in tissues

A) Transport Uptake of O_2

- PO_2 alveolar air $\rightarrow$ 100mmHg
- PO_2 at arterial end $\rightarrow$ 95mmHg of capillary

B) Transport of O_2

- Arterial blood contains - 20mL O_2 / 100mL blood
- Venous blood - 15mL O_2 / 100mL
- 5mL of O_2 is transported per 100mL from lungs to tissues

$\Rightarrow O_2$ carrying capacity of blood

- 1g Hb binds with 1.34 mL of O_2
- 15g Hb carries $\rightarrow$ 20.1 mL of O_2

$\Rightarrow O_2$ content

- Only 77% of Hb is available (due to shunt)
- $\therefore$ 100mL of arterial blood carries only 19.8 mL of O_2

$\Rightarrow$ % Saturation of Hb

$$\% \text{ Saturation} = \frac{O_2 \text{ content}}{O_2 \text{ capacity}} \times 100$$

= 97%

$\sim$ % of Hb combined with O_2

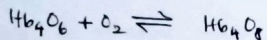
• Transport of O_2 is in 2 forms: $\begin{cases} \text{dissolved (8\%)} \\ \text{Combined (92\%)} \end{cases}$
 i.e., out of 19.8 mL $\begin{cases} 0.3 \text{ mL} \\ 19.5 \text{ mL} \end{cases}$

- dissolved form determines partial pressure

- Combined form (oxy-Hb)

- 1) Heme moiety can only combine with CO_2
- 2) 1 Hb molecule (4 Heme) $\rightarrow 4 O_2$
- 3) Loose and reversible
- 4) Only oxygenation (not oxidation)
- 5) 0.01 seconds

Reactions: $Hb_4 + O_2 \rightleftharpoons Hb_4O_2 + O_2 \rightleftharpoons Hb_4O_4 + O_2 \rightleftharpoons$



Here, affinity of Hb for O_2 increases with each step.

-(Heme-Heme interactions)

Reasons- Config in $Hb_4 \rightarrow T$ config (tense state)

• In this state, there are only minimal O_2 binding sites. Heme portion is covered by globin chains

In $Hb_4O_8 \rightarrow T$ state changes to R state (relaxed)

• Here, O_2 binding sites get exposed

(Co-operative binding kinetics operates here.)

$\downarrow$
 Resulting in sigmoid (S-shaped) curve

Oxygen Dissociation Curve (ODC)

- Curve obtained when reln b/w pO_2 and % Satn of Hb are plotted

- S/Sigmoid shaped

- PHASES (3) $\begin{cases} \text{Flat top} \\ \text{Steep fall} \\ \text{Flat bottom} \end{cases}$

1) Flat top (loading zone/Association curve)

• O_2 uptake in lungs

• At $pO_2 \geq 100 \text{ mmHg}$, Hb is $\frac{94\%}{100\%}$ saturated

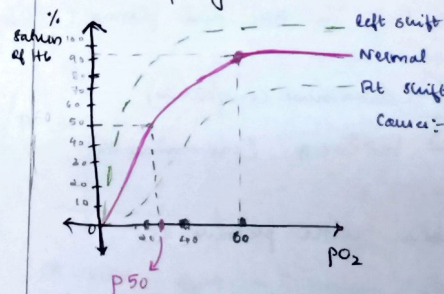
• Even if pO_2 falls to 60 mmHg, Hb saturation is still 90%.

2) Steep fall (unloading zone/dissociation curve)

• Occurs at pO_2 below 60 mmHg at tissues (unloading)

• In tissue, $pO_2 = 40 \text{ mmHg}$ $\therefore O_2$ Hb released

O_2 rapidly.



(Bohr effect)
 Affinity $\downarrow$, O_2 released

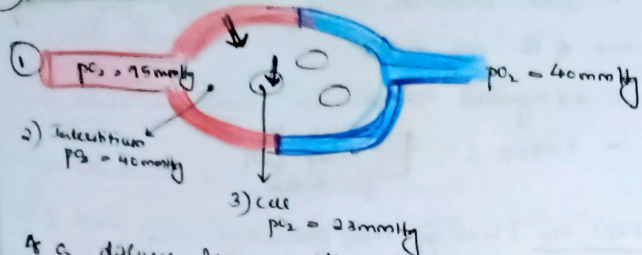
- Causes:
- 1) $\uparrow$ in temp
 - 2) $\uparrow$ in $d, B - DPG$
 - 3) $\uparrow pCO_2$
 - 4) $\uparrow H^+$ ($\downarrow$ in pH)

• Refers to pO_2 that produces 50% satn with Hb

• Normal $\rightarrow 25 - 27 \text{ mmHg}$

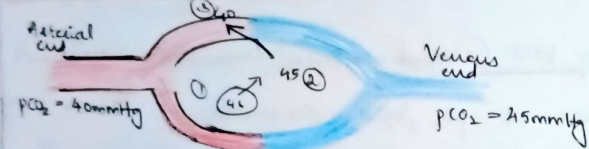
• Hb affinity $\propto \frac{1}{P_{50}}$

② O_2 transport from tissue capillaries to tissue



As O_2 diffuses from capillary blood to tissues, till it becomes 40 mmHg at venous end.

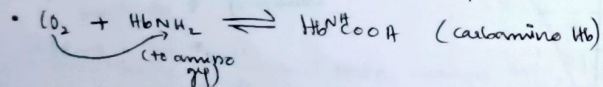
▷ TRANSPORT OF CO_2



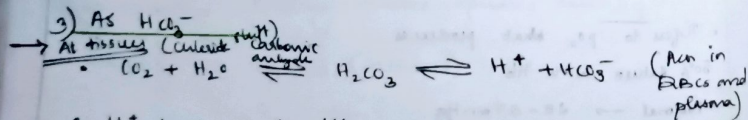
Three forms

- 1) dissolved (7%)
- 2) Combined with Hb (23%)
- 3) As HCO_3^- in RBC and plasma (70%)

a) Combined with Hb (Carbaminic compounds)



• CO_2 can also combine with proteins



• H^+ buffered by Hb

• HCO_3^- diffuses into plasma in exchange for Cl^-

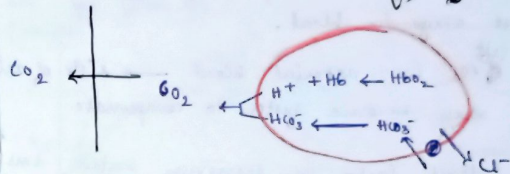
which is facilitated by Band 3 Anion exchanger
→ Chloride shift / Hamburger phenomenon

• For each CO_2 molecule added to RBC, an H^+ of 1 osmotically active particle (Cl^-) occurs

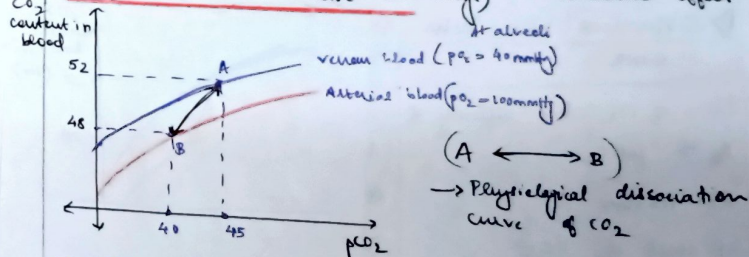
Applied: Hematocrit of venous blood is normally 3% greater than arterial blood

- Also, more fragile.

→ At lungs (Reverse chloride shift)



→ CO_2 dissociation curve (in lungs) - Haldane effect



② Haldane effect - "Oxygen helps remove CO_2 from blood"

• For every 100ml blood, 4ml CO_2 released because of Haldane effect.

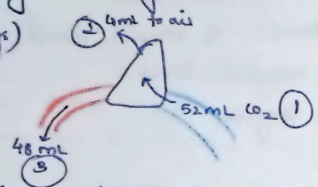
• O_2 entering lung pushes CO_2 out of blood more efficiently.

• Bind of O_2 with Hb, tends to displace CO_2 from blood
→ shift of COC to right.

• Point A in graph, denotes that at pCO_2 of 45 mmHg CO_2 content in venous blood is 52 mL.

• At point B, CO_2 content $\downarrow$ to 48 mL
(This is by releasing out 4 mL CO_2 from venous blood to lungs)

Diagram:



• If Haldane effect not present, only 2 mL of CO_2 released, rest stays in blood.

• So 50 mL CO_2 in arterial blood $\rightarrow$ risk of acidosis
 $\therefore$ Breathing may become faster to compensate.

$\Rightarrow$ Haldane effect helps in removing extra 2 mL of CO_2 from blood to air (lungs).

Differences b/w Bohr and Haldane effect

BOHR	HALDANE
1) P.e. of CO_2 $\downarrow$ affinity of Hb for O_2	P.e. of O_2 $\downarrow$ affinity of Hb for CO_2
2) At tissues	At alveoli and pulm capillaries
3) At shift of ODC helps release O_2 into tissues	At shift of ODC helps remove CO_2 in ~~blood~~ ^{lungs}

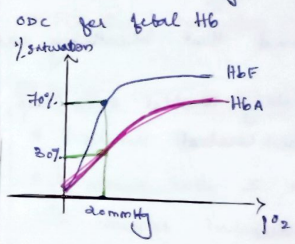
Applied physiology

1) Stored blood is not safe for transfusion to a hypoxic patient

Ans: • 2,3-DPG $\downarrow$ in stored blood with ACD (Acid citrate dextrose) as anticoagulant
• ODC to left, O_2 release will be less
• Prevented by using CPD as anticoagulant (Citrate phosphate dextrose)

2) A/c count is high in fetus

Ans:



Advantageous for O_2 transfer (not delivery) to

- HbF has higher affinity for O_2 than HbA
- So, release of O_2 will be decreased.
- To counteract this, A/c count is higher in fetus.

CH-6

Regulation of Respiration

▷ NEURAL REGULATION (essay)

- 1) Voluntary aspect
- 2) Automatic
- 3) Reflexes

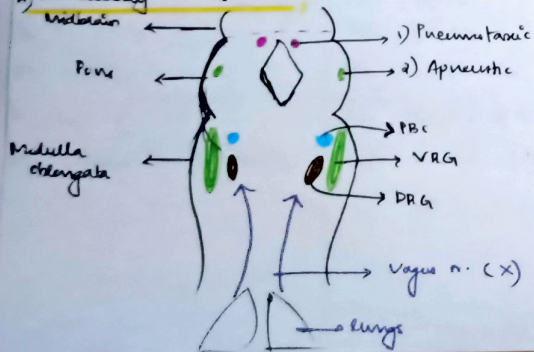
1) Voluntary

- Centre → cerebral cortex
- Breathing point → 40s (beyond that breathing can no)
- Online's case → Only voluntary control over resp, no automatic control

2) Automatic - Involves resp centres

- 1) Medullary centres → PBC, DRG, VRG
- 2) Pontine → Pneumotaxic, apneustic centre

1) Medullary centres



1) PBC (Prebotzinger complex)

- b/w NA and lateral reticular nucleus
 - Pacemaker of expn
 - produces activity in phrenic and intercostal n.
- CA → Any transaction b/w phrenic and PBC, stops action

2) DRG (close to NTG)

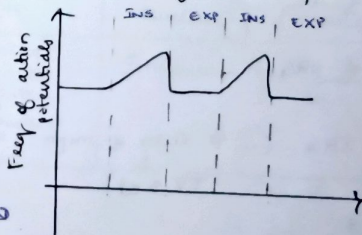
- Made of I neurons (inspiratory) → Active in inspi
- Controlled by PBC
- Receive impulses from lung chemoreceptors (via vagus)

3) VRG (ventrolateral part of medulla)

- Extent from NA to NRA
- Contains both I and E neurons, myelinated
- E neurons inhibit I neurons

3) IRS (Insp ramp signal)

- Pattern of action pot produced by resp centres



- Nervous signals that is transmitted to inspiratory muscles begins weakly and rises steadily in a ramp manner for 2 seconds

- 1) Stops abruptly for approx next 3 seconds (turns off excitation of diaphragm and allows elastic recoil of lungs and chest wall to cause expn)
- New cycle begins

B) PONTINE CENTRES

1) Pneumotaxic (Upper part of pons)

Function → Modify spontaneous discharge of medullary centres

→ Inhibitory effect on I neurons

↓
Decreases duration of IRS

MCA → PC is stimulated

↓
I neurons inhibited (Switch off IRS)

↓
Rate of resp ↑ and filling vol ↓ (2° effect)

∴ Resp → Shallow and rapid

NOTE → PC inhibits apneustic centre

Damage to PC leads to prolonged inspir/apneus breathing

2) Apneustic centre (Lower part of pons)

Function → Opposite to PC

MCA → Shrink of AC

↓
Excitation of DRG

↓
Prolonged IRS (∴ ↑ duration of insp
↓
TV ↑)

↓
Apnoea

3) Reflex control

1) Hering-Breuer inflation reflex

Inflation of lungs ($TV > 1.5L$)

↓

Stretch of stretch receptors

↓

Vagus (afferent)

↓

Pont-medullary resp centre

↓

Inhibit resp

2) Irritant receptors in resp tract

1) Sneezing reflex

- deep insp followed by forceful expir with open glottis

- Receptor → Nasal mucosa

2) Cough reflex

- deep insp followed by forceful expir with closed glottis

- R → trachea, bronchi

3) Defluation reflex

- temporary apnoea in pharyngeal phase of swallowing (Prevents food entry to resp tract)

4) Hering-Breuer deflation reflex R → Bronchial epi

- Responsible for yawns and sighs

CHEMICAL REGULATION (Essay)

I) Def → changes in PO_2 , PCO_2 , pH of blood act on resp centre through a set of receptors → chemoreceptors
→ These receptors are sensory n. endings sensitive to changes

II) Types of chemoreceptors (based on locn)

- Central chemoreceptor - CO_2 , acidosis
- Peripheral " - O_2 (hypoxia)

A) Central chemoreceptor

• Locn → beneath ventral surface of medulla oblongata

• Stimulus → ↑ in CO_2 and H^+ conc in surrounding interstitial fluid and CSF

• Action

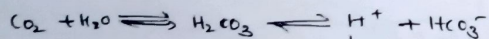
- CO_2 readily crosses BBB (as it is small, soluble, uncharged molecule)

- ↑ in blood pCO_2

↓
cerebral vasodilation

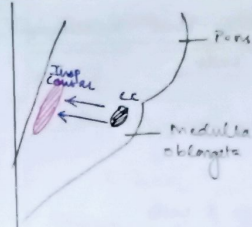
Enhanced diffusion of CO_2 into ^{brain} interstitium and CSF

- In CSF and brain tissue,



↑ H^+ conc stimulates central chemoreceptor

Stimulates resp → Reach resp centre

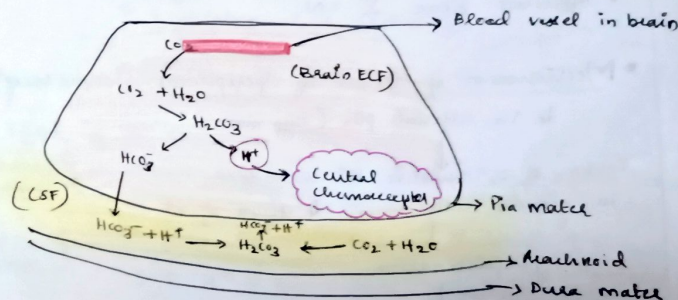


NOTE:

- BBB does not allow charged ions (H^+ , HCO_3^-) to cross readily
- C.C., not stimulated by hypoxia
- C.C. inhibited by - anaesthesia, cyanide, during sleep

4) If pCO_2 of brain and IF persistently high, action of H^+ on C.C. stops.

Reason: • Normally CSF has fewer buffers than blood
• But if hypercapnia prolonged, HCO_3^- diffused into CSF and neutralises H^+ , pH in CSF → alkaline



B) PERIPHERAL CHEMO RECEPTOR

• Locn → carotid and aortic bodies

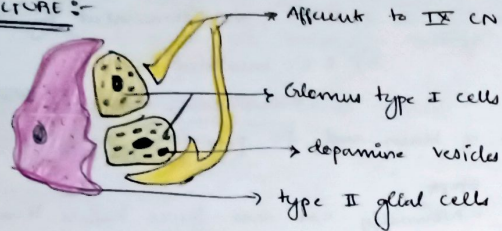
• Stimulus → Most potent: changes in PO_2
(Sensitive to ↑ CO_2 , H^+ but lesser extent)

▣ CAROTID BODY

- located at bifurcation of common carotid artery
- Has 2 types of cells
 - Type I → has granules containing dopamine (stimuli ab PO_2 receptors)

- Type II cells/ → supporting cells that surround glial cells
4-6 type I cells

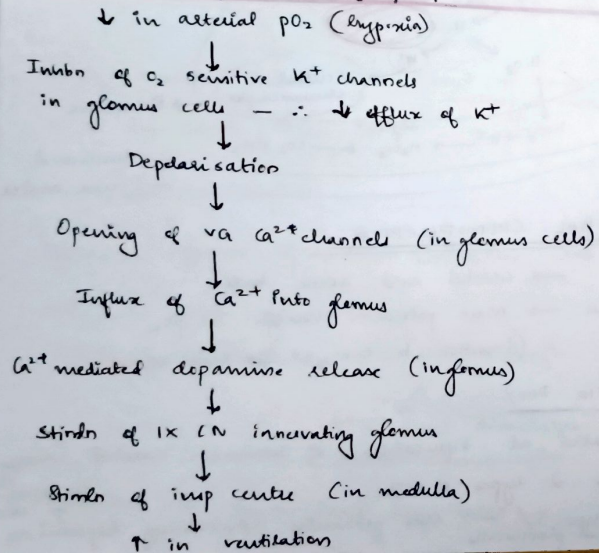
STRUCTURE:-



AORTIC BODY

- Located near arch of aorta
- Afferents from IX CN

Mechanism of action of peripheral chemoreceptors



III) Ventilatory responses

1) pCO_2 (response to $led\ pCO_2$)

• Hyperventilation (CO₂ washed out)

↓ $PaCO_2$

Depression of resp centre

↓ Apnoea (shortness of breath)

↓ $PaCO_2$, ↓ PaO_2 → Stimn of resp centre

2) Response to hypoxia

↓ ↑ ventilation (CO₂ washed off)

↓

↓ $PaCO_2$ → Inhibn of resp centre

3) Effect of H^+ conc on resp

1) Metabolic acidosis } — ↓ HCO_3^- secondary to ↑ in H^+ . Eg: Diabetic ketoacidosis

2) Resp acidosis } — 1° pulm hypoventilation → elevation of pCO_2 (↑ H_2CO_3)
∴ ↑ H^+ . Eg: Narcotic poisoning

3) Metabolic alkalosis } — ↑ HCO_3^- secondary to ↓ in H^+
Eg: Excess vomiting (loss of gastric acid)

4) Respiratory alkalosis } — 1° pulm hyperventilation → ↓ of $PaCO_2$
↓ in H^+ ← ↓ in H_2CO_3
Eg: Voluntary hyperventilation

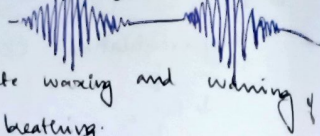
1) Periodic breathing

2) Cheyne - Stokes breathing

A type of abnormal breathing

- When resp shows alternate waxing and waning & TV, it called Cheyne - Stokes's breathing.

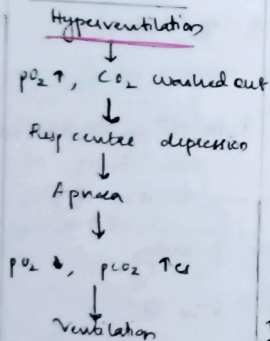
Regular apnoea with hyperventilation



→ Occur (Physiologically) in

- 1) Vol hyperventilation
- 2) Premature babies
- 3) During sleep
- 4) High altitude

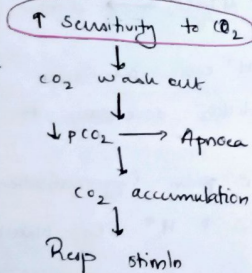
Mechanism:



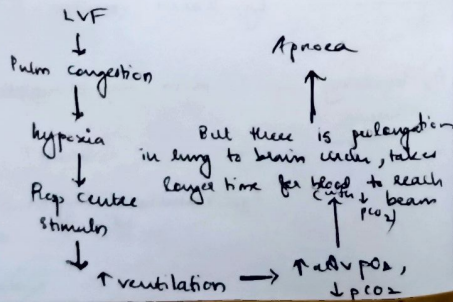
→ Pathologically in:-

- 1) CCF
- 2) Renal failure and uremia
- 3) Brain damage
- 4) ↑ intracranial tensions

2) Mechanism (Brain damage)



II) Cardiac Failure



2) Biot's breathing

- Varying periods of apnoea alternate with hyperventilation
- The change from one to other is abrupt
- Not seen physiologically
- Gradual ↑ or ↓ of resp amplitude is absent
- Apnoeic interval differs

→ Causes:- Head injury, meningitis (Road traffic accident)

2) Hypoxia

- It is defined as the O₂ def at tissue level
- 4 types:-

1) Hypoxic hypoxia

- pO₂ of arterial blood is ↓ (PaO₂)

- Causes:-

High altitude

- breathing in closed space
- ↓ in ventil → bronchial asthma
- Weakness of resp muscles → bulbar palsy, myelitis
- Abnormalities of thoracic cage
- Defect in O₂ transport across memb → edema, fibrosis
- Anatomical shunt → congenital cyanotic heart disease
- Damage of resp centres

FEATURES

- 1) pO_2 of arterial blood $\downarrow$
- 2) Normal Hb - O_2 carrying capacity is normal
 - Hb not saturated with O_2
 - O_2 content in blood $\downarrow$
- 3) Abnormal O_2 conc diff $\downarrow$

2) Anemic hypoxia

- Hb available to carry O_2 $\downarrow$
- May be due to quantitative (Hb conc) or qualitative defect of Hb
 - $\rightarrow$ CO poisoning
 - $\rightarrow$ Altered Hb (meth Hb)

FEATURES

- 1) pO_2 is normal - (Peripheral chemoreceptors not stimulated)
- 2) O_2 carrying capacity is $\downarrow$
- 3) O_2 content $\downarrow$ in blood
- 4) A-V O_2 conc diff is normal (O_2 is present in art amt but can't be carried)

NOTE = Anemic hypoxia not severe at rest

- $\uparrow$ 2,3-DPG
- $\uparrow$ CO
- Redistribution of blood (dynamic flow)

CO poisoning

- Symptoms
- Headache, nausea, vomiting, dyspnoea, fainting, paralysis, loss of consciousness
 - Cherry red discoloration of skin, nail bed

$\rightarrow$ Death - 70-80%

$\rightarrow$ Chronic exposure to sublethal dose $\rightarrow$ brain damage, Parkinsonism

3) Stagnant hypoxia

- due to $\downarrow$ ed blood flow to tissues - O_2 not delivered to tissues

- Causes:

- Generalised circulatory def as CCF, shock
- Localised " " $\rightarrow$ peripheries, coronary, cerebral vessels

FEATURES

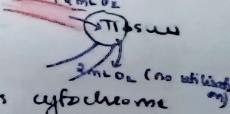
- pO_2 is normal
- Normal Hb
- O_2 carrying capacity normal
- O_2 content normal
- A-V O_2 conc difference $\uparrow$ (as in venous blood O_2 is decreased)

4) Histotoxic hypoxia

- Tissues unable to utilise the O_2 supplied to them

- Causes:

- due to inhibition of oxidative processing
- Cyanide poisoning $\rightarrow$ cyanide inhibits cytochrome oxidase
- A-V difference is $\downarrow$ (as venous O_2 $\uparrow$ ed)



TYPE	pO_2	% saturation	O_2 content	A-V O_2 conc diff
1) Hypoxic	$\downarrow$	$\downarrow$	$\downarrow$	$\downarrow$
2) Anemic	N	$\downarrow$	$\downarrow$	$\downarrow$
3) Stagnant	N	N	N	$\uparrow$
4) Histotoxic	N	N	N	$\downarrow$

❑ Symptoms of Hypoxia

Early → R - Restlessness

A - Anxiety

T - Tachycardia

Late → B - Bradycardia

E - Extreme restlessness

D - Dyspnea

❑ EFFECTS :-

1) On cells → Prols of transcription → Produce angiotensin factors and erythropoietin

2) On nervous system → Rapid severe hypoxia can cause sudden loss of consciousness and death in 4-5 min

3) On resp system → Hyperventilation and stimuli of receptors

4) Digestive system → Loss of appetite, nausea, vomiting

5) CVS → initially → ↑ rate of contraction, CO, BP, coronary and cerebral circulation
late → ↓ " " " "

❑ TREATMENT :- O_2 therapy

- Treat underlying cause

- ↑ ing concentration of inspired O_2

- Usual in :-

1) Hypoxic hypoxia (except R-L shunt)

⇒ Hyperbaric O_2 therapy

• Adm of O_2 at ↑ pressure 2-3 atm

• Indications → CO poisoning, anemic and stagnant hypoxia

→ decompression sickness

→ Gas gangrene, diabetic ulcers

Complication → O_2 toxicity

3) Cyanosis

- It is the bluish discoloration of skin and mucous membrane when the amt of reduced Hb in capillary blood is $> 5g/100ml$ of blood.

- 3 types :-

A) Central cyanosis

- due to imperfect oxygenation at level of lung or heart (Eg: C heart disease, heart failure etc)
- Extremities → warm (due to peripheral vasodilation)
- Pulse → bounding
- Affects → tongue and lips

B) Peripheral cyanosis

- due to excessive redn of oxygns in capillaries
- Seen in

C	→ cold
O	→ obstruction (venous)
L	→ LVF and shock
D	→ bad CO

4) Asphyxia

- condition where acute hypoxia and hypercapnia occur together

- causes :- strangulation, choking, drowning, hypotension, tetany

- Stages :-

- 1) Stage of exaggerated breathing
- 2) " of convulsions
- 3) Stage of coma

3d Stage of coma (H-7)

a. Environmental Physiology

Essay

1) Physiological adjustment in high altitude and deep sea

Ans: High Altitude (↓ barometric p)

- Dalton's law $P = p_{O_2} + p_{CO_2} + p_{N_2} + p_{H_2O}$

At high alt, p_{O_2} and p_{N_2} is decreased.

1) Acute effects of hypoxia

Altitude	% Saturo	Symptoms
Upto 8000 ft	93%	- No ↑ in pulm ventilation
10,000 - 20,000 ft	Hypoxic stimuli of chemoreceptors	- Hyperventilation Resp alkalosis
> 20000 ft	50%	- LOC → COMA ↓ level of consciousness

2) Acute mountain sickness → Acute cerebral edema Acute pulm edema

- People who ascend rapidly to high altitudes develop symptoms by from few hours to 2 days after ascent.

ACUTE CEREBRAL EDEMA
Hypoxia
↓
Local vasodilat of cerebral vts
↓
Fluid leak into cerebral tissue
↓
Cerebral edema
⊕

ACUTE PULM EDEMA
Severe hypoxia
↓
Pulm arterioles constricted (pulm hypertension)
↓
Capillary p is high and hence there is exudation of fluid into interstitial space and alveoli

• Symptoms

- headache, irritability, breathlessness, convulsions

• Treatment

- 1) Glucocorticoids
- 2) Acetazolamide
- CA inhibitor
- ↓ COF produ

• Prevention

- 1) Slow ascent to facilitate acclimatization
- 2) Breathing O_2

• Treatment

- 1) Rest
- 2) Hyperbaric O_2 therapy
- 3) Nifedipine → Ca^{++} channel blocker, lower pulm artery p.

3) Chronic mountain sickness

- If a person stays at high alt for a long time, initially he may get acclimatized, later fails.

- FEATURES:-

- 1) Polycythemia → ↑ red viscosity - ↓ tissue blood flow
- 2) Pulmonary hypertension } → hypoxia (as there is pulm vasocent)

3) Hypertrophy of Rt side of heart

4) COF

5) Fall in peripheral arterial p

6) Death, if person not shifted to lower altitude

4) Acclimatization

- physiological adaptation to a new altitude
- changes in response to long term exposure to hypoxia

→ ↑ red barometric p.

→ ↓ red v

Changes	Physiological basis
1) ↑ Pulmonary ventilation	• Stimuli of arterial chemoreceptors • 5 times the normal (within 2-3 days)
2) ↑ Diffusing capacity and TLC	• 3 fold ↑ - - ↑ in volume and pulm blood flow - ↑ in SA and ↓ in thickness of resp membrane
3) Cellular acclimatization	• ↑ in no. of mitochondria • ↑ in cytochrome oxidase • ↑ capillarity of tissues • ↑ in myoglobin
4) ↑ in RBC count and Hb	- Hypoxia stimulates erythropoietin ↓ polycythemia - Hb - 22g%, PCV - (60-65%) - BV ↑ ed by 20-30%
5) ↑ in 2,3-DPG	• ↓ es affinity of Hb for O_2 • ∴ ↑ ed unloading of O_2 to tissues
6) Cardiovascular changes	• CO ↑ ed by 20-30%

Features seen

1) Polycythemia

2) Shift of ODC to right

3) Barrel-shaped chest

4) Enlargement of rt side of heart

5) Weak capacity of cardiac and skeletal m ↑ ed

DEEP SEA PHYSIOLOGY (↑ ed barometric p.)

- deep sea divers, mine workers etc are exposed to high barometric p.
- As depth ↑ es, barometric p ↑ es, vol of gases inc (Boyle's law)

~~1) Nitrogen narcosis~~ / Rapture of depth

- At high p., N_2 readily dissolves in fat of body, esp membranes of neurons
- This alters ionic conductance through membrane of neurons and finally ↓ es neuronal excitability.

Characteristics (Similar to alcohol intoxication)

- 1) At 4-5 atm - Euphoria, joviality followed by ^{impaired} mental ^{activation} for
- 2) At 6-7 atm - Drowsiness, strength of muscle ↓ es
- 3) > 8 atm - Pain insensitivity, convulsions and coma

Prevention → Breathing O_2 -He mixture

→ Advantages is

- 1) Low density of He (1/7 that of N_2)
↳ ↓ airway resistance and work of breathing
- 2) Less narcotic effect
- 3) Less soluble

Complications (due to prolonged use of He)

High Pressure Nerve Syndrome (HPNS)

- Inert gases (like He) act as anaesthetic at high p. due to lipid solubility and action on nerve cell membrane
- Tremor and drowsiness

2) CAISSON'S DISEASE / decompression sickness / dybarism / Compressed air sickness / Bends / Diver's palsy

- disorder which occurs when a person returns rapidly to atm pressure after exposure to high barometric p as in deep sea.
- When diver descends into deep sea, large amt of N_2 dissolved in body fluids
- As diver ascends rapidly, N_2 is decompressed and escapes from tissue at a faster rate.
- As it is a gas, N_2 forms bubbles while escaping hence occluding vessels.

SYMPTOMS

Location of bubbles	Symptoms
1) In and around joint	Bends
2) In muscle	Muscle cramps and severe pain
3) Myelin sheath and sensory fibre	Sensory impairment, numbness, itching, paraesthesia
4) Motor fibre	Temp paralysis, resp failure
5) Brain and SC	Sensory and motor impairment
6) Pulmonary capillaries	Ulcers $\rightarrow$ pulmonary edema and dyspnea
7) Coronary arteries	Myocardial damage and MI

PREVENTION

- 1) Slow decompression
- 2) Decompression tables

TREATMENT

- 1) Recompression chamber
- 2) O_2 - He mixture
- 3) Hyperbaric O_2 therapy
- 4) SCUBA

3) Effect of O_2 at high barometric p $\rightarrow$ O_2 toxicity

- due to prodn of superoxide anion which is a free radical of H_2O_2 and occurs if 100% O_2 is inspired at high barometric p.
- Prevented by $\downarrow$ conc of O_2 in gas mixture

4) Effect of $\uparrow$ CO_2 - CO_2 narcosis

- Hypocapnia initially stimulates resp, but ^{retention} ~~depression~~ of large amts of CO_2 produces depression of CNS
 $\downarrow$
 CO_2 narcosis

Characteristics

- Confusion, headache, coma, death
- Resp acidosis and plasma HCO_3^- may exceed

SCUBA (BA) - Self Contained Underwater Breathing Apparatus

- Contains air cylinders, valve system and a mask
- Possible to breathe air/gas mixture without high p
- Valve system $\rightarrow$ ensures only req amt of air enters during insp ^{through} ~~mask~~ mask and exp air is expelled out of mask

DISADVANTAGE

- person can remain in sea only for short period

BA / Respiratory Adjustments in exercise

Effect

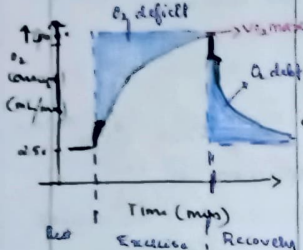
Physiological basis

Activation of
higher centres
↑

1) ↑ in ventilation

1) Before starting exercise (prophetic phenomenon)

- anticipation of exercise itself ↑ rate and force of resp

 O_2 consumption fell

from 250ml/min

↓
400ml/min

(achieved) ↑

2) Changes in diffusion

- Pulm blood flow ↑

from 5L/min

↓
2.5L/min

3) Changes in metabolism

2) During exercise

- ↑ p_{CO_2} (central chemo stim)
- ↓ p_{O_2} (peripheral chemo)
- ↑ in H^+ (lactic acid)
- ↑ in body temp
- ↑ in K^+
- ↑ symp stimuli (catecholamines cause vasoconstriction)

3) Recovery phase

- there is an abrupt ↓ in ventilation, followed by a pause and then gradual decline to pre-exercise state

- O_2 debt1) ↑ p_{CO_2} pulm blood flow → due to excitement of capillaries

2) Stretching of resp memb → ↓ thickness and ↑ SA

3) Exercising muscle utilises more O_2

4) Shift of ODC to rt (due to hypoxemia, hypercapnia, acidosis, temp)

→ Aerobic glycolysis (→ glucose)

→ Anaerobic (→ form of lactic acid)

E2 O_2 debt (PA)- It is the extra amt of O_2 consumed during recovery phase of exercise- The ↑ in ventilation continues for as long as 90 mins after exercise i.e., until O_2 debt is repaid.- This ↑ in ventilation is due to lactic acidosis (not ↑ p_{CO_2} / hypoxia, bcoz p_{CO_2} and p_{O_2} are normal after exercise)- O_2 debt is req for :-

- 1) Regenerate ATP, creatine phosphate etc
- 2) Remove off the metabolites (like lactic acid)
- 3) Replace O_2 that has come from myoglobin

E2 Resp changes in trained athletes (BA) A FI1) Alveolar PO_2 falls
ventilation ↑

- due to stronger resp muscles and improved lung compliance

2) ↑ Functional capacity ($V_{O_2 \max}$)- $V_{O_2} \rightarrow$ maximal O_2 consumption
- Athletes can use more O_2 (better utilization by tissues)

3) Improved diffusion capacity

- due to ↑ capillary volume

4) Tidal volume ↑

- due to strong resp muscles, compliance

5) ↓ lactic acid accumulation

-

6) O_2 debt falls

7) ↓ Number of breaths (RR)

8) ↑ Gas exchange efficiency