

Pulmonary alveolar macrophages

- APCD - Amine
(VIP, substance P)

Alveolar capillary/mucipulatory membrane

Thickness 0.3 - 1 μ m

total area

Alveoli is largest biological membrane

Pleura

parietal (close to chest wall)
visceral (deep)

Neural space - capacity - 2ml

- Adhesive nature
- $\uparrow$ hydraulic trans [2 layer slide each other, but difficult to sep. from apart]
- Lubricant
- Protection
- Facilitate change in size

Applied aspect

- ① Pleural effusion - water in the cavity
- ② Pneumothorax - air in cavity
- ③ Haemothorax - blood in "

Bronchopulmonary segment bronchus (1st lobe)

part of lung supplied by a seg

- segmental bronchi
- Anatomically a functional unit
- Inferior to it lung is common
- Lobe bronchi, $\uparrow$ $\uparrow$ 11 seg

Non-respiratory function

① Filtration function

The hairs @ entrance filter out so that no particle more than 6 μ m enter the lung through the nose

By a process called turbulent precipitation



Air passing through the nasal passages hits nasal turbinates, septum of the pharyngeal wall of each time it hits, it must change the direction of movement

The particles suspended in the air having more mass of momentum than air can't change their direction as air

$\therefore$ they continue forward, striking the surface of obstructing, EI are entrapped in the mucus coating of transported by the cilia to the pharynx & finally swallowed.

of the remaining particles blow

1 of 5 μ m they settle in the small bronchioles as a result of gravitational precipitation

• ciliated mucus - pathology

• particles less than 1 μ m diffuse against the wall of alveoli & adhere to alveolar fluid

particles, less than 0.5 μ m remain suspended in the alveolar layer & are expelled by exhalation

Application

- ciliary immobility
- [Kartagener's syndrome] $\Rightarrow$ Bronchiectasis
- $\hookrightarrow$ it from the birth

② Humidification

Labelled diagram of tracheo-bronchial tree

Zone	Name of divisions	No. of generation	Structure of division	No. of tubes in division
CONDUCTING ZONE	Trachea	0		1
	Primary bronchi (cartilaginous)	1		2
	Secondary bronchi	2		4
	Tertiary bronchi	3		8
	Quaternary bronchi	4		16
	Quinary bronchi	5		32
	Sixth bronchi	6		64
	Seventh bronchi	7		128
	Eighth bronchi	8		256
	Ninth bronchi	9		512
RESPIRATORY ZONE	Ten terminal bronchioles	10		10
	Alveolar ducts	11		10
	Alveolar sacs	12		10
	Alveolar sacs	13		10

② Humidification

humidify inspired air so that cold or hot air becomes body temp. on reaching alveoli

③ Immune function

(i) PAM (Pulmonary Alveolar Macrophage)

- digestible: released into lymph
- not: giant cell capsule (tubercle bacilli, silica, carbon)

no $\uparrow \Rightarrow$ lead to fibrous tissue formation
 $\Rightarrow$ permanent damage

ii, IgA

$\hookrightarrow$ mucus/bronchial secretion

(iii) Protease activated receptors

- $\hookrightarrow$ protect pulmonary epithelial cells
- $\hookrightarrow$ prostaglandin,

④ Pulmonary circulation

(i) Reservoir of LV filling

(ii) Microemboli: blood clots, fat, air bubbles

$\hookrightarrow$ venous blood contains these & are filtered by pulmonary circulation, before reaching systemic circulation.

(iii) Low pulmonary hydrostatic pressure
 $\hookrightarrow$ tends to pull fluid from the alveoli into the pulmonary capillaries.

$\rightarrow$ keeps alveoli dry & favours gas exchange

alveolar capillary net.

(iv) Drug absorption

Anasthetics, bronchodilation can be able to diffuse respiratory membrane & able to enter to systemic circulation.

⑤ Olfaction

Delivers odourants to olfactory epithelium

⑥ Acid-Base Balance

- eliminate CO_2 from body.
- CO_2 - raised - ventilation - $\uparrow$
- rate of respiration - $\uparrow$ - CO_2 released

⑦ Vocalization & Phonation

Vocal cords act as vibrator & vibrating elements are vocal cords.

Resonators - mouth, nose, pharynx, nasal sinuses.

Phonation - vocal cords move together, so that passage of air thru them causes vibration & air movement in resp system helps in voice improvement.

Application: - Nasopharyngitis

⑧ Metabolic & endocrine functions

- Storage - VIP, opioids
- Activation - ACF
- Removal - PGE1, bradykinin, SHT
- Synthesize - surfactant.

Mechanism of Breathing

- Air does not flow into lungs
 $\hookrightarrow$ static lung ppty
- Air flows into the lungs
 $\hookrightarrow$ Dynamic lung ppty

INSPIRATION

① Diaphragm

$\pm 5\%$ change in intrathoracic vol lower 31st ribs & xiphoid process of sternum.

Supplied - phrenic nerve

Parts: - costal - ribs

central - ligament around vertebra

central tendon

contraction pushes rib cage outwards & enlarges thoracic cavity in AP & lateral planes.

Pushes abdominal content downwards & increases vertical diameter.

normal inspi_{dia} - 1-2 cm
deep inspi_{dia} - 7 cm

Applied aspect

- Reduced in obesity, pregnancy
- Transection of spinal cord above C_3 is fatal
 $\hookrightarrow$ phrenic nerve is affected
- below C_3 is ok

Bilateral phrenic nerve palsy

can survive with laboured breathing

Resting position

Ribs slanting downwards allowing sternum to fall backwards towards the vertebral column.

Elevation of rib cage causes the sternum to move forward away from spine & during the AP diameter to 20%.

② External Intercostal m. [Ext ICM]

Run obliquely downwards & forwards from upper to lower ribs.

Ribs pivot as if they were hinged at the back & hence contraction of external ICM causes elevation of lower ribs.

Pushes the sternum outwards & $\uparrow$ AP diameter (lessen degree of transverse diameter)

Mechanism

① Pump handle movements

Greatest effect of movt of sternum & upper ribs is in the anterior diameter of thorax.

Combined effect of upper ribs & sternum occurring in sagittal plane is called pump handle movt.

2. Bucket handle movements

- Movements of lower ribs is at lateral aspects of have more angle shape with indirect attachment with sternum anteriorly.

• Munt causes changes in transverse diameter at frontal plane

③ Accessory m.

- Sternocleidomastoid - lift sternum upwards.
- Anterior serratus - lift 1st 2 ribs.
- Alae nasi - flaring of nostrils

EXPIRATION

At the end of inspiration, inspiratory m. relax & thoracic vol ↓

Elastic recoil of lung is unopposed causing lung to deflate

produce passive expira

Forced expiration

① Abdominal m.

contraction ↑ intraabdominal pr & pushes diaphragm upward.

② Internal IIR

Pass obliquely downwards & posteriorly & pull the ribcage downwards when they contract.

Applied

- Bronchoconstriction (Asthma)
 - use exp muscles even in resting phase.
- Endurance training

Strengthening of exp'm.

Pressure changes during deep

- Intra pleural (thoracic) pressure - pr. of fluid in thin space b/w lungs & chest wall pleura.

Lungs have tendency to collapse & chest wall to expand equal & oppo. forces acting in a closed chamber → produce -ve intra pleural pr - start of inspiration, -

Base = -2.5 mmHg

Subatm @ peak of insp'm = -6 mmHg

After exp return back to -2.5 mmHg

* Lungs pulled into more expanded pos'n & pr. in airways become -ve & air flows into lungs

* End of insp → sucking of lung brings chest wall back to expira, pos'n,

Recoil pr. of lung & cw balance

Strong insp. pressure to -30 mmHg

Bracking effect

some part of insp muscles contract in early phase of exp. & sucking the exp.

IPP: Factors

1. Deep inspiration
2. Valsalva manoeuvre - sudden forceful expiratory mouth ↑ pr upto 60 to 70 mmHg
- ↓
- Lung vol. causing defla.

• Beats of cough can straining is dangerous in old patients (repeated coughing)

• Large ↑ in IPP produced by pressing over great vessels within thoracic cavity

↓ venous return & COP

③ Gravity

• More negative at apex (-7 mmHg) compared to bases (-2 mmHg)

Transmural pr: mea sure of the elastic forces in the lungs that tend to elastic forces in collapse the lungs at each instant of expiration, called the

equal pressure

- * Less @ base & lungs are less expanded @ base
- End of expira
- ↳ structures on becomes -ve causing airways to close at the base

• Most of inspired air goes to the apex than the base of lung

Resting lung :- Apex expanded base less expanded

Significance: IPP

1. Facilitates inspiration
2. Maintains shape of chest & size of lungs

- easiest stretching of lung & chest wall

- loss of normal IPP results in collapse of lung & barrel shaped chest

Measurement

- needle insert
- intraoesophageal pr. by inserting balloon catheter through mouth.

Applied

① Emphysema

- Loss of lung elasticity.

② Pneumothorax

common, made by air in pleural space, air moves into pleural space & IPP becomes atmospheric. ↳ lung collapse

- loss of IPP, lung collapse

due to inward recoiling of
subcase expands outwards
due to outward recoiling.

- T_{PP} is 0 as there is no pr. diff.

↳ from pleural pr.

- gunshot
- knife injury
- rupture lung abscess
- pleural fluid aspiration
- TB therapy.

② Transpulmonary P_r / Distending P_r

- P_r - diff across chest wall
- IAP_r - IPP
- keeps lungs inflated & prevents lung collapse

③ Intra-alveolar / Intrapulmonary pr

Quiet breathing:- at end of expiration &
end of inspiration (IAP_r = atmos pr)

↓
as no air is going in & coming out of
lungs,

with the beginning of inspiration, intra-
alveolar pr ↓ to 1 mmHg below the atm.
pr, but reaches to atm. pr @ the end of
inspiration.

when expiration follows passively, the elastic
recoil of lungs causes the intraalveolar pr
to move slightly to the tre side, i.e. 1 mmHg
& again becomes atm. pr @ the end of

expiration.

Factors - IAP_r.

Valsalva manoeuvre

↳ forced expiration against a closed
glottis (P_r > 100 at P_r)

Muller's manoeuvre

↳ forced inspiration against a closed
glottis (P_r to < 80 mmHg)

Sequence of events during inspiration

contraction of diaphragm

(& other insp. muscles)



Expansion of thoracic cage



IPP becomes more -ve



↑ in transpulmonary pr



Infln of lungs



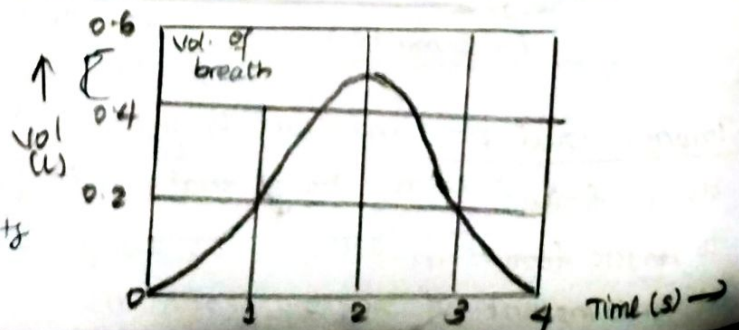
↓ in alveolar pr. which becomes
subatmospheric



Air flows into airways & lungs



Cessation of contraction of inspiratory
m. ends inspiration



Relaxation of insp. m.



Dropping of rib cage



Intrapleural pressure becoming less -ve



↓ in transpulmonary pr.
(back toward pre-inspir. level)



Deflation (recoiling of stretched lungs)



Decrease in alveolar diameter



Alveolar pr. becoming > greater than atm. pr.



Air is pushed out of lungs

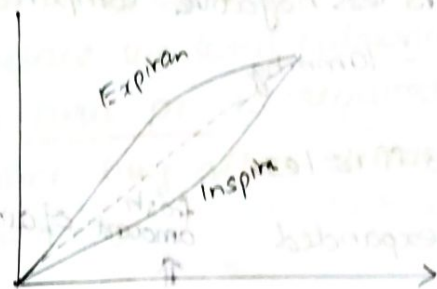


Air flow (exp) continues until alveolar pressure equals atm. pr.

compliance of lungs

• The extent to which the lungs will expand for each unit ↑ in transpulmonary p.r.

↑
Lung vol. change



pleural pr.

- Total compliance of both lungs together

= 200 ml of air / cm.

H₂O trans. pul. pr.

Caused by

(1) elastic forces of lung tissue (1/3rd)

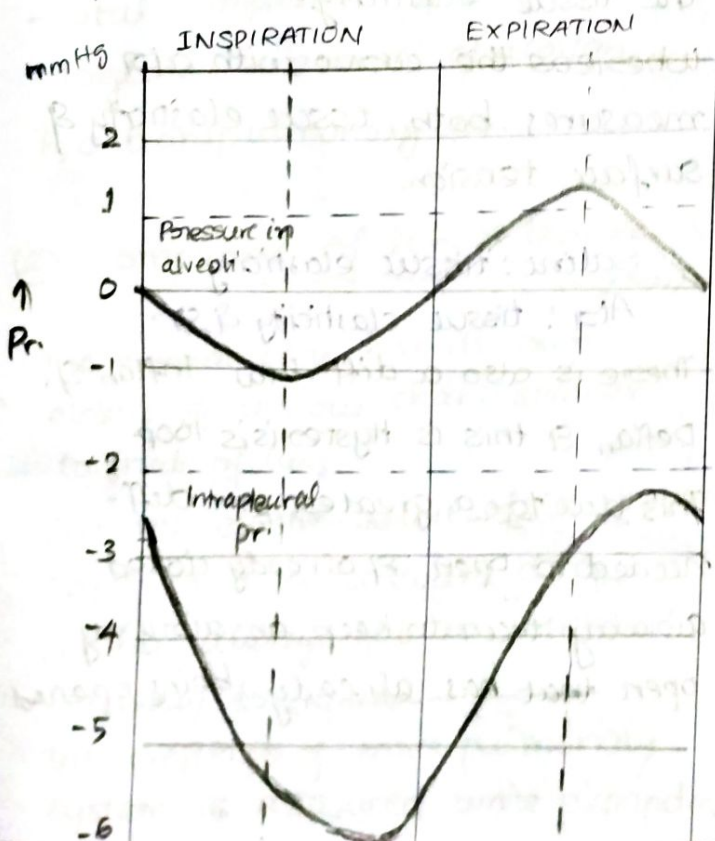
- elastin & collagen fibers interwoven among the lung parenchyma

Deflated lung - fibres are in an elastically contracted & kinked state

Expanded lung - fibres to fibres become stretched & unkinked elongating & exerting even ↑ elastic force

(2) elastic forces caused by surface tension of the fluid that lines the inside of walls of alveoli - 2/3rd.

Regional variation of compliance



23/10/23

Regional variation of compliance:

Base.

- IPP during this - 2.5 to - 6 mmHg, which is less negative compared with apex - 10 mmHg
- TPP diff is less
- Less expanded $\uparrow$ fresh amount of air
- Upright posn: ventila, per unit lung vol. is greater
- Compliance is more

Apex

- More neg TPP holds lung in expanded position
- $\uparrow$ in vol./unit $\uparrow$ in IPP is smaller as expanded lung is stiffer.
- Compliance is lesser.

Compliance

1. Elastic tissue (1/3rd contribn)

- Stretchability / distensibility: ability of lung to inflate.
- Distensibility & elastic recoil: opposite.
- Overstretching: loss of elastic recoil.

Presence of elastin & collagen

Fibres: contribute to lung elasticity.

* Arrangement of elastin fibre network in lung parenchyma is similar to arrangements of fibres of nylon stocking.

It allows to easily stretch, but if it is overstretched, it loses its elasticity & becomes loose. (breathes) Hence it cannot be deflated easily due to the loss of elastic recoil.

compliance: 2. Surface Tension
of fluid lining alveoli (2/3rd)

The mag. of each component can be measured by measuring the lungs from body of an exp. animal & distending them alternatively with saline & air while measuring intrapulmonary pr.

As the saline structures surface T. to 0, the pr-vol. curve obtained with the saline measures only the tissue elasticity.

whereas the curve with air measures both tissue elasticity & surface tension.

Saline: tissue elasticity
Air: tissue elasticity & ST.

There is also a diff. b/w Inflation & Deflation & this is hysteresis's loop. This due to a greater pr. diff. needed to open & already closed airway than to keep an airway open that has already being opened

Hysteresis loop

greater pr. diff. needed to open closed airway



Measurement of compliance.

1. Static lung compliance.
2. Dynamic lung compliance.

Static: pr-vol curve.

① compliance of lungs (alone)

- The extent to which the lungs will expand for each unit $\uparrow$ in TPP.
- Total compliance of both lungs together - 200 ml of air/cm H₂O transpulmonary pr.

② compliance of lung & thorax (together)

The thoracic cage has its own elastic & viscous char. similar to that of lungs.

But the elastic recoil of (w)/thoracic cage is in a outward direction.

It has always got an intrinsic tendency to expand.

The compliance of entire pulmonary system is measured while expanding

lungs of totally extubated or paralyzed subject. The compliance is measured by forcing air into the lungs in small quantities while recording the lung pr & vol.

To inflate the total pulmonary sys. almost twice pr. is required to inflate the same lung after removal from the chest cage

• Value: 110 cm/cm of water

* Compliance of lung & thoracic cage together

Compliance is determined from the slope of pr-vol. curve.

Factors affecting compliance.

- Distensibility & lung recoil
- Lung vol. [comp. $\uparrow$ at $\downarrow$ lung vol]
- Lung size & comp
- ST $\uparrow$ comp $\downarrow$

Vasoconstrictions.

INCREASE: Aging
Bronchial asthma
emphysema
DECREASE: Fibrosis
pulmonary HT
Atelectasis
Low surfactant.

Airway resistance

Under normal respiratory cond:
A pr. gradient of less than 1cm
water is only needed to pass the
air from alveoli to atmos.

$$< 1 \text{ cm H}_2\text{O}$$

Greatest resistance: large bronchioles

2 bronchi near the trachea

Smallest airways are aligned parallel

SA is $\uparrow$ R so $\downarrow$ airway resistance

Applied

Airway dls are diagnosed very late
as these dls affect mainly
which contribute only very less to airway
resistance

Determinants of resistance.

- Tone of SM (smooth m.)
- Lung vol.
- Gas density & viscosity
- Edema of airway lining.
- In forced exp. the resistance will be $\uparrow$
due to compression of airway.
- Phases of resp - forced exp.
- Mucus collecting in lumen.
- Diameter of airways.

Work of breathing

1. Elastic work: (65%)
is req. to expand the lung &
cw elastic force.

2. Viscous resistance (7%)
to overcome viscosity of lung & cw
structure.

3. Airway resistance (28%)

Normal values: 0.3 to 0.8 lbf/cm²
increased: exercise, emphysema,
asthma

Alveolar ST

At air-water interface, the water
mole. on surface has a strong attrac.
with each other.

As a result, the water molecules
always try to contract. This is
happening on the inner side of
alveoli also. This tends to force air
out of alveoli through the bronchi
which causes alveoli to collapse.

So the net effect is to cause
an elastic contractile force of the
active lung called **surface tension**
elastic force.

Role of surfactant

It is a surface active agent in
water which reduces the ST
produced by Type II alveolar cells
and these cells are granular, that
form 10% of SA of alveoli. These cells
contain lipid inclusions that are secreted

into the surfactant & into the
alveoli

1. Type I cell $\rightarrow$ flat cell.
2. Type II cell $\rightarrow$ cuboidal
cell

composition
Dipalmitoyl phosphatidyl choline

Surfactant : regulation of feedback
protein A uptake by type II
(SP A)

SP B, C : monomolecular film.

SP D : organisation of B, C into
surfactant layer

A & D : innate immunity

Regulation of Sur. Syn.

Hormonal : cortisol, thyroxine,
insulin.

Stretching of lungs:

Yawning, yawning, sighing,
physical exercise

Sur. Syn.

Time : 34 weeks
completed : 38 - 40 weeks

ST of pure water : 72 dyne/cm
ST of alveolar fluid : 50 (with surfactant)
ST of alveolar fluid : 5-30 (with surfactant)

Functions

1. Promotes alveolar stability

Acc. to the law of Laplace, in a
spherical str. like an alveolus,
the distending pr. P equals

a time, the tension $\div$ radius

$$P = \frac{2 \times \text{tension}}{\text{radius}} = \frac{2T}{R}$$

In the lung parenchyma, alveoli are
interconnected by each other &
all alveoli don't have the same
diameter, which changes during
respiration.

If surface tension was same in small &
large alveoli, acc. to Laplace law,
pr. would be greater in small
alveoli than in the bigger & so
small alveoli can drain into the
larger one & collapse.

However this doesn't happen as the
S.T. is not pen unit S.A.

With the presence of surfactant
S.T. of the alveoli $\downarrow$ with $\downarrow$ in S.A
& so, S.T. is $\downarrow$ in smaller alveoli
& $\uparrow$ in bigger alveoli.

Thus the distending pr. remain same
& the smallest alv. don't collapse.

Hence the alv. are stabilised & the
tendency for the smaller alv. to
empty into the larger one is reduced.

2. This prevents lung collapse

During deflation compn. of surfactant
mole. $\downarrow$, S.T. $\uparrow$ causing infln. new
surfactant is released onto the alv.
surface forming a new film on the
alv. epithelium.

3. It prevents pulmonary edema

If surfactant was not present, the
unopposed S.T. in the alveoli would

produce 20 mmHg force favouring the translocation of fluid from the blood into the alveoli.

④ Work of breathing ↓

⑤ Immunity [VAD]

Applied aspects

- Before birth
- foetus makes resp. efforts in utero but lung remains collapsed
- After birth
- lung expands due to strong ins. effort.

• Surfactant: prevents collapsing again

① IRDS [Infant Resp. Distress syndrome]

- Pulmonary dist. that develops in infants born before the surfactant is functional.
- ST maybe ↑: alv. collapsed in many areas

↓
pulmonary atelectasis

- Hyaline-membrane d/s (conium namu)
- ↓
- glass-like
- surfactant system is not functional
- atelectasis
- retention of fluid
- diabetes

Rx

- surfactant replacement therapy
- Glucocorticoid supply therapy

② Pulmonary alv. proteinosis

over production of alv. prot.

③ Atelectasis following surgery

Deep breaths → surfactant syn ↑
otherwise collapse

④ Pneumothorax

Short work **Alveolar stability**

these are 2 main factors affecting alv. stability

1. Surfactant
2. Interdependence

there is a mutual support among the adjacent alveoli as they are interconnected. This is a major factor in the adult lung contributing to prevent collapse of adj. alv.

when alv. tend to collapse, the surrounding alv. develop greater expanding forces preventing their collapse.

This interdependence not only prevents atelectasis but also opens up alv. that are already collapsed

• major factor that maintain alv. stability in adults

Spirometry L-VOL R-capacity

Pulmonary ventilation can be studied by measuring the volume moved by alv. in to ...

Expiratory reserve vol

Lung volumes

① Tidal vol.: vol. of air inspired or expired with each normal breath (500 ml)

(Draw the lung vol.)
Sphygmomanometer (measuring blood pressure)

② Ins. reserve vol.: Extra volume of air that can be inspired over & above the normal tidal vol. when the person inspires with full force (3000 ml)

③ Ex. reserve vol.: Max extra vol. of air that can be expired by forcing exp. after the end of a normal tidal expira. (1100 ml in men)

④ Residual volume

Vol. of air remaining in the lungs after the most forceful exp.

Vol.: 1200 ml

Lung capacities

• Inspiratory cap: TV + IRV.

→ Max. vol. of air that can be insp. after tidal expira.

→ 3500 ml.

⑤ Functional residual cap.

ERV + RV

• Amount of air that remains in the lungs at the end of normal expira.

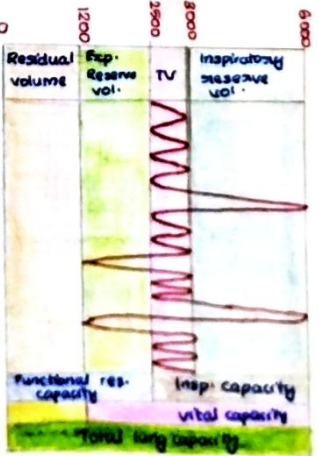
→ 2300 ml.

⑥ Vital capacity

ERV + TV + IRV

• Max vol. of air that can be exp. after a max ins. effort

Men - 4 L
Women - 3 L



Significance of FRC

- maintaining stable gas tension for alveolar gas exchange

- Buffering & prevent sudden interruption of respiration

- increased: air trapping in asthma, emphysema

- Decreased: hypoxia

Forced vital capacity/FVC

• Amount of air that can be expired with greatest force & speed, after a maximal inspiration.

It is measured after a person first inspires to TLC & then exhales to (rest) spontaneous with a maximal exp. effort as rapidly & completely as possible.

Significance

- Index of pulmonary fn.
- Access strength of exp. m.

④ Total Lung Capacity

• Max. vol. to which lung can be expanded with greatest possible effort.

- Men is about 5.8L
- Women 4.2L

pul. vol. & Capacities		
	Men	Women
TV	500	400
IRV	3000	1900
EV	1100	700
RV	1200	1100
Capacities (ml)		
IC	3500	2400
FRC	2300	1800
VC	4600	3100
TLC	5800	4200

* It is difficult to be completely emptied following forced expiration

RV, FRC, TLC - Helium dilution method.

20-25% lesser in females.

Dynamic LVQ/LC

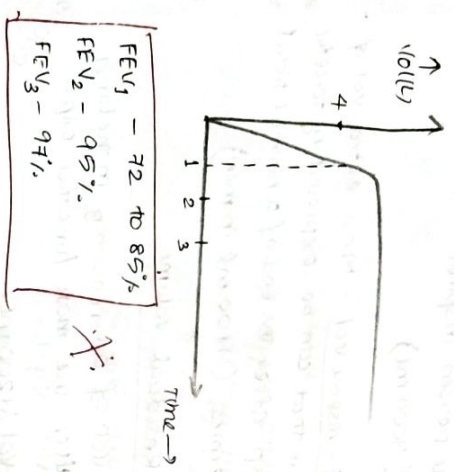
① Timed VC/Forced exp. vol.

- Measures FVC in 2 sec, with time.
- Indicates airflow rates

• Procedure
After sec. a few normal TV, the subject inhales to the TLC and hold his breath. Immediately the speed of breathing is ↑ & the subject exhales forcefully as maximum as possible. This graph represents exp. flow over time in 2 sec.

FEV₁

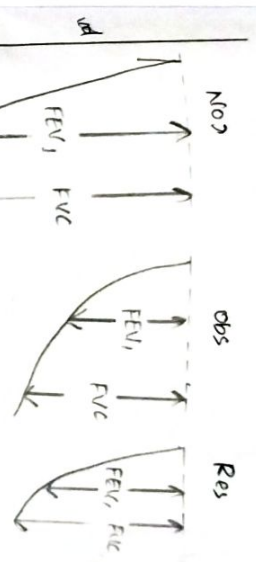
The fraction of VC which is exp. during the 1st sec of a forced exp.



* FEV₁ ratio is a useful measurement

• FVC to recognize airway d/s, it helps to differentiate b/w obstructive & restrictive lung d/s.

FEV₁ / FVC ratio - obstructive



	No	Obs	Res
FEV ₁	3.3	1.0	1.8
FVC	4.0	2.0	2.0
FEV ₁ /FVC	83%	50%	90%

In obs. d/s the curve shows a slow steady slope to the FVC.

FEV₁ as % of FVC is grossly ↓ as there is obs. to airflow in the RT so there will be a marked ↓ in both

FVC & FEV₁.

* bronchial asthma

Restrictive lung d/s

These patients airflow tends to be fast at first, but quickly tapers out to reach FVC. Hence the pattern of FEV₁ curve is same as that of normal & also FEV₁ as % of FVC is also normal, but FVC is grossly reduced.

∴ In restrictive disorder, there is less of FVC without loss of FEV₁/FVC.

Obs.	Res.
↓ in both FVC & FEV ₁ /FVC	loss of FVC without loss of FEV ₁ /FVC

② MMEFR

(Max. mid exp. flow rate)

* Max. flow during middle third of total expired volume.

* FEF 25-75%.

* Patency of small airways

③ PEFR

(Peak exp. flow rate)

* Max. flow rate attained during a forceful exp. effort

* 400-600 L/min

* Index of patency of

④ MVV (Max. voluntary ventilation)

* Largest vol. of air that an individual can breathe into & out of lungs in 1 min with max voluntary effort

* 140-180 L/min.

* Breathing reserve - MVV

- max. ventila. avail. during exp. exercise

* 20-50% of MVV

⑤ RMV (Resp. minute vol) (Sporadic)

Total amount of new air moved into the resp. passages in each min.

TV x RP = 500 ml x 12 (Resp rate) = 6L/min

Dead space vol. (DSV)

① Anatomical DSV ✕

- Air in conducting areas which do not take place in gas exchange
- resp. system vol. exclusive of alveoli
- 150 ml (500 - 350 ml)
TV -

② Physiological DSV

- It includes anatomical DS & alveolar DS.
- Alveolar DS (under perfused & over ventilated alveoli which do not take place in gas exchange) non equilibrating gas vol.

- anatomical DSV + alveolar DSV
= Phy DSV

- alveolar DS - negligible & so.
- Anatomical DS = Physiological DS
- Diseased state : - Phy DS > Anat DS
- Measured - single breath N₂ curve

Alveolar ventilation spotting ✕

- Rate @ which new air reaches alveoli, sacs, ducts & resp. bronchioles
- Vol. of air that actually reaches alveoli per min.

$$(TV - RDS) \times RR$$

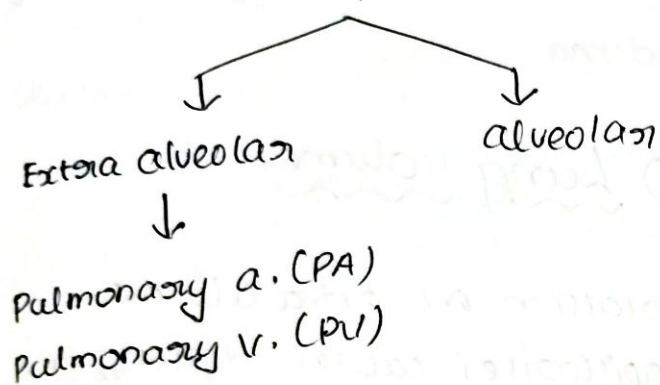
$$= 350 \times 12 = 4200 \text{ ml/min}$$

06/02/2023
Monday

Respiratory System (cont...)

Pulmonary circulation

Types of vessels



40% of total wt. of lungs.

Pressures in pulmonary system

Right ventricle : Systolic BP - 25 mmHg
DBP :- 0 to 1 mmHg.

PA :- SBP - 25
DBP - 8
MAP - 15
(mean arterial pr.)

P. cap - 7

LA & PV - 1 to 5

pulmonary circulation = mean = 14

Systemic circulation = mean = 93

* Pulmonary circulatory system is a low pr system

Spcl features of pulmonary circulation.

① Low pr. system.

② PA

- branches are very short, large diameters, thin walls
- lesser smooth m. & more distensible
- large compliance (7 ml/mmHg)
- accommodate SV from RV.

This large compliance which is similar to that of entire systemic arterial tree.

Applied aspect. (PA)

• Lung act as reservoir.

- Haemorrhage compensated by automatic shift from lungs to systemic circulation. (5L - stroke vol.)

• Failure of Lt side of heart:

- damping of blood into pulm. circulation.

③ Pulmonary Arterioles

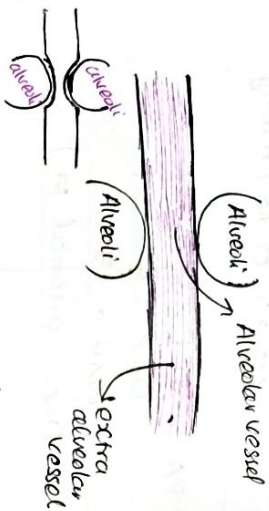
- thin walled & lesser smooth m.
- lesser ability to contract rather than the thick walled systemic arterioles

Applied aspect

- In heavy exercise: - the lung has ability to accommodate $\uparrow$ COP without $\uparrow$ ising the PA pr
- Prevents dipt of Pulmonary edema.

② Lung volumes

- Traction on extra alveolar capillaries causes distens, $\uparrow$ resistance reduce
- Alveolar vessels are compressed by enlarging alveoli $\uparrow$ $\uparrow$ resistance increase.



③ Chemical & humoral factors.

- Vasoconstrictors:
 - thromboxane
 - norepinephrin
 - angiotensin
- Vasodilators:
 - bradykinin
 - prostacyclin
 - nitric oxide

④ P. Vein

- thin walled & highly compliant

⑤ P. capillaries

- form lattice in alveolar wall & blood flows as a thin sheet.

- don't form capillary network.
- thin walled with no structural support

collapse

- alveolar pr $>$ pul. capillary pr.
- ⑥ Changes in PV & LA pr also aff. gas exchange.

⑦ Pulmonary vascular resistance

Imp (PVR) $\times$

- vascular system has thin wall, less smooth m, less resistance, more compressible & more compliant.

- even lowered if pr. within pulm. system $\uparrow$, due to.

1. Recruitment
2. Distension

Factors affecting PVR

- ① Blood flow increased.

Hypoxia - VC in pulmonary arterial system

- * Hypoxia inhibits O_2 sensitive K chns pul vascular smooth m. (VSM) contract in pulmonary arterial system

$\downarrow$

Depolarisation of actives, Ca chns

$\downarrow$

Ca influx causes vasoconstrict.

Significance of hypoxic VC

- causes blood to flow through open aerated areas providing an automatic control for redistribution.

Factors affecting Pulmonary blood flow (PBF)

1. Pulmonary vascular resistance
2. Res Gravity
 - standing position: BP is 90 mm Hg greater @ foot than @ heart level (hydrostatic pr)
 - lungs: 23 mmHg exists b/w apex & base

15 mmHg $\swarrow$ 8 mmHg

Pul Arterial pr:

Apex lungs: 15 mmHg (es) than the pul. ar. pr. @ level of heart.

Base: 8 mmHg greater than the heart.

* causes regional difference in Pul. Blood flow (PBF)

- capillaries in the alveolar wall are distended by the blood pr. inside them.

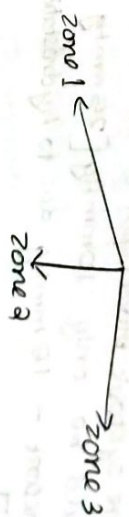
- compressed by the alveolar air pr. from outside.

• Lung alv. air pr $>$ capillary pr:

capillaries close

$\downarrow$

No blood flow depending upon regional blood flow, lungs $\rightarrow$ divided into 3



Zone 1: 1. No blood flow

* Local alveolar capillary pr. never rises higher than the alveolar air pr.

• very small or non-existent in healthy individual.

• But in condn (like ~~He~~ Haemorrhagic shock, we can see zone 1.

Gravity causes capillary bed in apices to be under perfused!

08/02/2023
Wednesday
Gravity...

capillaries in the alveolar wall are distended by the blood pressure inside them.

compressed by the alveolar air pressure

Zone 2: Intermittent BF (blood flow)

when a person is in the upright position, the pulmonary pressure at the apex is 15 mmHg less than the pressure at the level of heart.

∴ the systolic blood pressure at the apex would be only 10 mmHg [25 mmHg @ heart - 15 mmHg due to hydrostatic pressure]

This 10 mmHg is greater than the alveolar air pressure. ∴ so blood flows through the apical capillaries during systole

whereas in diastole, the 8 mm diastolic blood pressure at the level of heart is not sufficient to push the blood up to the 15 mmHg hydrostatic pressure gradient, resulting in no blood flow during diastole.

∴ blood flow through the apical part of lung is intermittent with the flow only during systole & cessation of blood flow during diastole & this is known as the zone 2 blood flow

Arterial pressure > Alveolar pressure > Venous pressure in zone 2.

Blood flow varies with diff. blood arterial & alveolar pressure & greater at bottom.

Zone 3 - Continuous flow

here the pulmonary pressure during both systole & diastole remains greater than the alveolar air pressure, resulting in a continuous flow in the alveolar capillaries in the lower areas of lung. here the blood flow depends upon arterial-venous pressure diff.

* Normal lungs have only zone 2 & zone 3.

zone 2 → intermittent flow @ apex

zone 3 → cont. flow @ base

Applied

lying down: only zone 3 including lung apices.

exercise: pulmonary vascular pressure rises to convert lung apices from zone 2 to zone 3.

Assessment of pulmonary blood pressure

↳ Swan-Gang catheters

↳ indirect measurement by measuring wedge pressure

Pulmonary capillary mechanics (dynamics)

forces tending to cause movement of fluid outward from the capillaries and into the pulmonary interstitium:

• Capillary pressure - 7
• Interstitial fluid colloid osmotic pressure - 14

• Negative interstitial fluid pressure - 8

TOTAL OUTWARD FORCE - 29

Force tending to cause absorption of fluid into the capillaries:

• Plasma colloid osmotic pressure = 28

TOTAL INWARD FORCE: 28

Net filtration force = 29 - 28

(NFF) = 1

causes the flow of fluid from the capillaries into interstitial spaces & from there into circulation via lymphatics.

Mechanisms of keeping alveoli dry

• Quest Permeability of lymphatics maintain negative pressure in the interstitial spaces.

Extra fluid is appears in alveoli

small openings flow alveoli & epithelial cells

Mechanically sucked into lung interstitium

Applied

Pulmonary edema

① Increases filtration
② Impedes lymphatics

↓

• Pulmonary interstitial fluid pressure to the side
• Results in fluid collection

Causes

• Left sided heart failure - pulmonary pressure

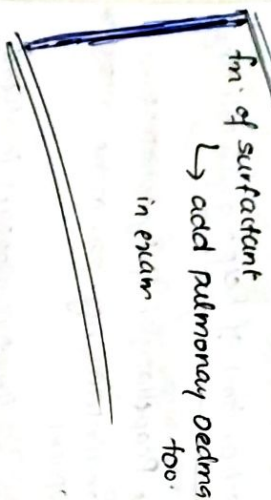
• Damage to pulmonary membrane

- pneumonia, toxic substance

• Pulmonary capillary pressure > 50 mmHg - Acute pulmonary edema

Safety factor = 21 mmHg

[28-7]



Ventilation-perfusion ratio: V_A/Q .

Ratio of alveolar ventilation to pulmonary BF

$$V_A/Q = 4/5 = 0.8$$

ventilation ↑ from apex to base

P. ventilation ↑ @ base

perfusion ↑ @ base

Ratio ↑ @ apex.

↓ in ventilation ↑ @ apex hence ratio ↑

$$\textcircled{1} V/Q = 0$$

Alveoli in alveolar equilibrium with O_2 & CO_2 in the venous blood.

$$P_{O_2} = 40 \text{ mmHg}$$

$$P_{CO_2} = 45 \text{ mmHg}$$

$$\textcircled{2} V/Q = \text{infinity}$$

- no perfusion

- no capillary blood to carry O_2 away & to bring CO_2 to alveoli

Alveolar air is equilibrated with inspired air.

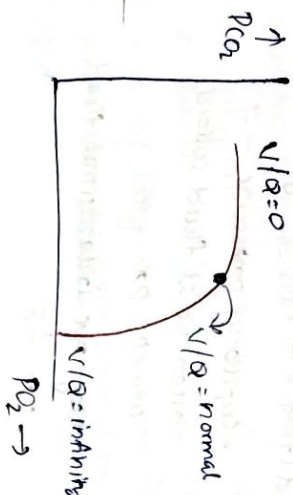
$$P_{O_2} = 149 \text{ mmHg}$$

$$P_{CO_2} = 0 \text{ mmHg}$$

$$\textcircled{3} V/Q = \text{Normal}$$

$$P_{O_2} = 104 \text{ mmHg} (149 \& 40)$$

$$P_{CO_2} = 40 \text{ mmHg} (0 \& 45)$$



$$\textcircled{4} V/Q < \text{Normal (Physiological shunt)}$$

These three are inadequate ventilation to provide O_2 needed to fully oxygenate the blood flowing through the alveolar capillaries. ∴ a certain fraction of venous blood

passing through the P. capillaries don't become oxygenated.

This fraction is called shunted blood

Also 2% of blood through the bronchial vessels which is also un氧enated shunted blood & this total amount of shunted blood per min. is called physiological shunt

Disorders

- emphysema, pneumonia, atelectasis, Pulmonary

$$\textcircled{5} V/Q > \text{Normal : Physiological DS (Dead space)}$$

more O_2 available in the alveoli that can be transported away by the flowing blood.

- ventilation is wasted (Alveolar DS)
- ventilation in conducting zone is also wasted (Anatomical DS)
- ventilation will be wasted (Alveolar DS)

$$\textcircled{6} V/Q : \text{Upper \& lower lung}$$

Upright position: Air & PBF are less in upper part.

↓ in BF is considerably greater than the ↓ in ventilation

$$V/Q = 3.6 \text{ (Phys DS)}$$

Base: Too little ventilation, relative to BF & value 0.6 (Phys shunt)

Applied aspect

- ① To affect the opening of lung
- higher O_2 facilitates growth of mycobacterium tuberculosis
- low perfusion

Exercise

② COPD

Assessment of V/Q

N_2 washout method

Questions

- ① Tracheo bronchovascular
- ② Non-respiratory fraction of lung
- ③ Mechanism of inspiration & expiration (flow & pressure changes during resp. cycle)
- ④ Factors changing during resp. cycle [Draw a label - 8M]
- ⑤ Compliance (def, lung & chest factors, variations)
- ⑥ Factors maintaining alveolar stability - 8M (Surfactant, interdependence)

✓ Spirogram (Draw & label)

- Significance of FRC, TLC

- Vital capacity

- RMR, Alveolar ventilation.

(any vol. can be asked)

- Dead space vol. (150ml)

✓ Spd features of p. circulation (8M)

- Factors keeping alveoli dry

(pul. cap. dynamics)

✓ V/p ratio

- 1. Give reason

- To affect open of lung

- Alveoli kept dry

1 mark

- Normal resp. rate

- Intral pleural pr / alveolar pr.

- Pleural fluid

- Value compliance

- Surfactant synthesized by which cell.

- LV/LC

- V/p ratio