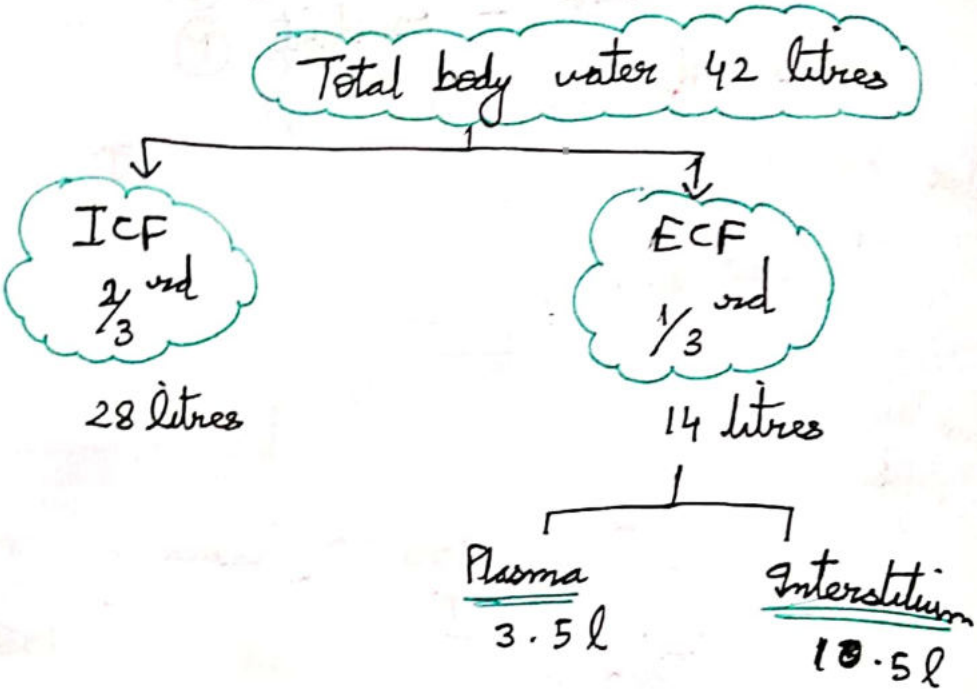
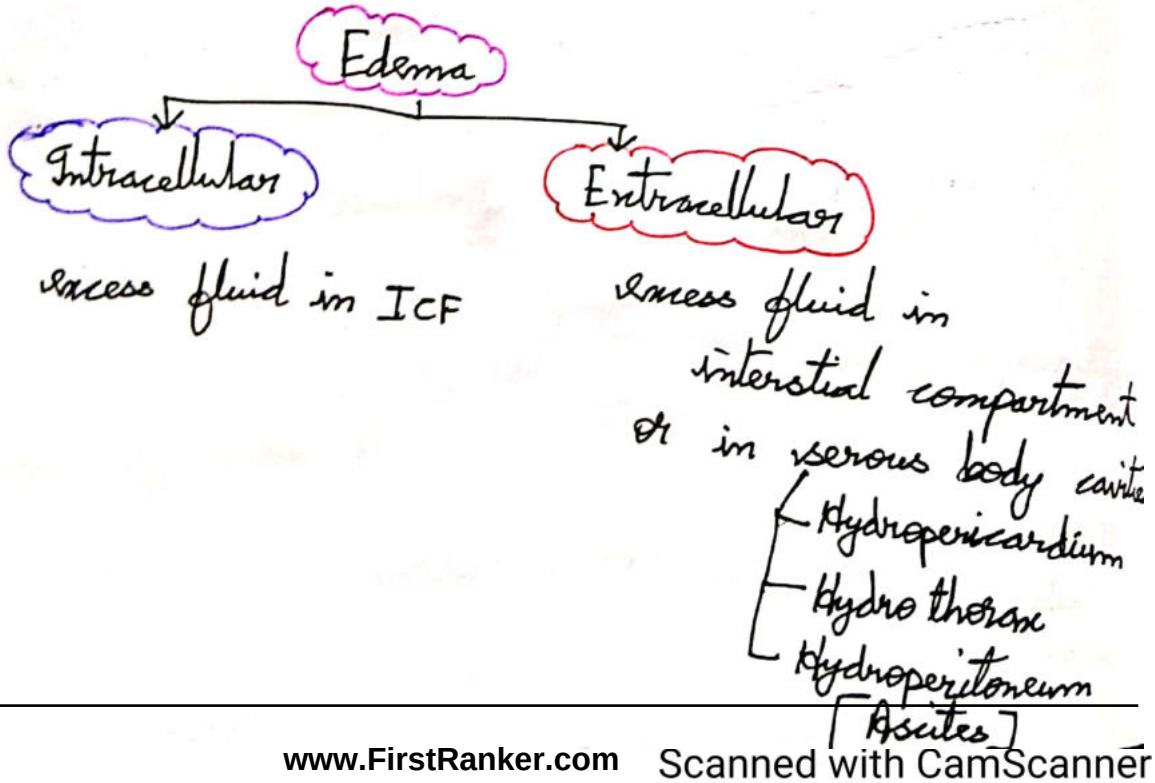
Mastering Edema :-

- Excessive fluid in body tissue - interstitial or [extravascularly] intracellular.
- Excess fluid is vessels - not edema.
- Adult:- 72 kg ⟶ 60% is Tot Body water - 42 l





... vascular compartment of a tissue

Hyperemia

Due to more arrival of blood into the tissue

① Blood flow

Congestion

Obstruction to the outflow of blood.

⇒ Intracellular Edema

- When cells become hyperosmolar → they absorb water from ECF.
- Cells are hyperosmolar when Na^+/K^+ ATPases do not function → Na^+ accumulation
  - When the cells become ischaemic → No O_2 → No ATP → No pump.
- When the cell membrane is damage → Na^+ & Ca^{2+} enter the cell [hyperosmotic] → Cell absorbs water & swells
- Concentration of Na^+ in blood ⇒ 135 – 145 mEq/L

Hyponatremia [low Na^+ conc]

↓

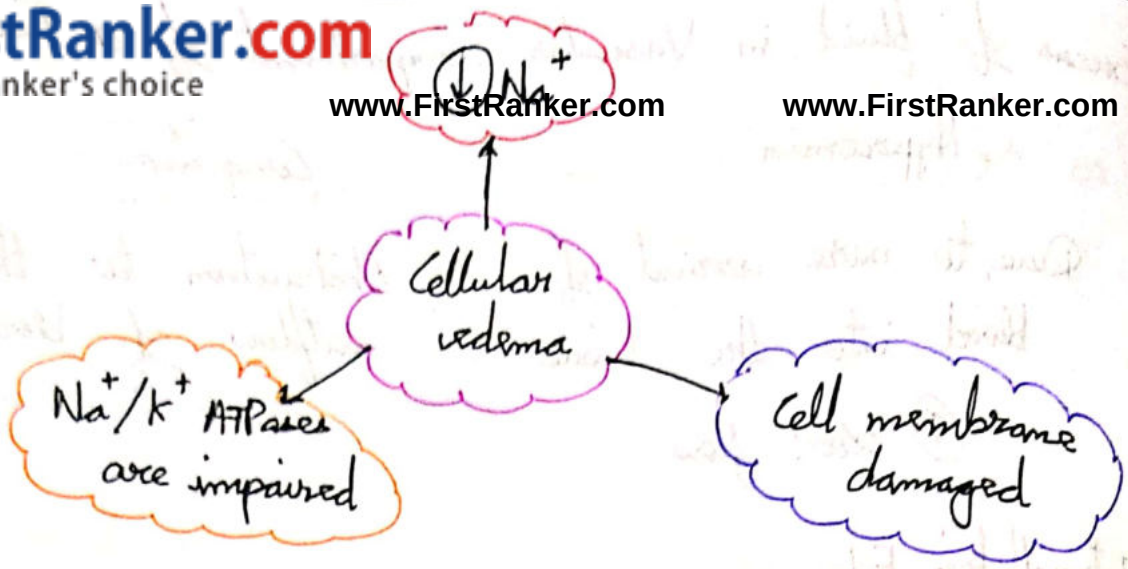
ECF becomes hyposmolar

↓

fluid moves from ECF → ICF

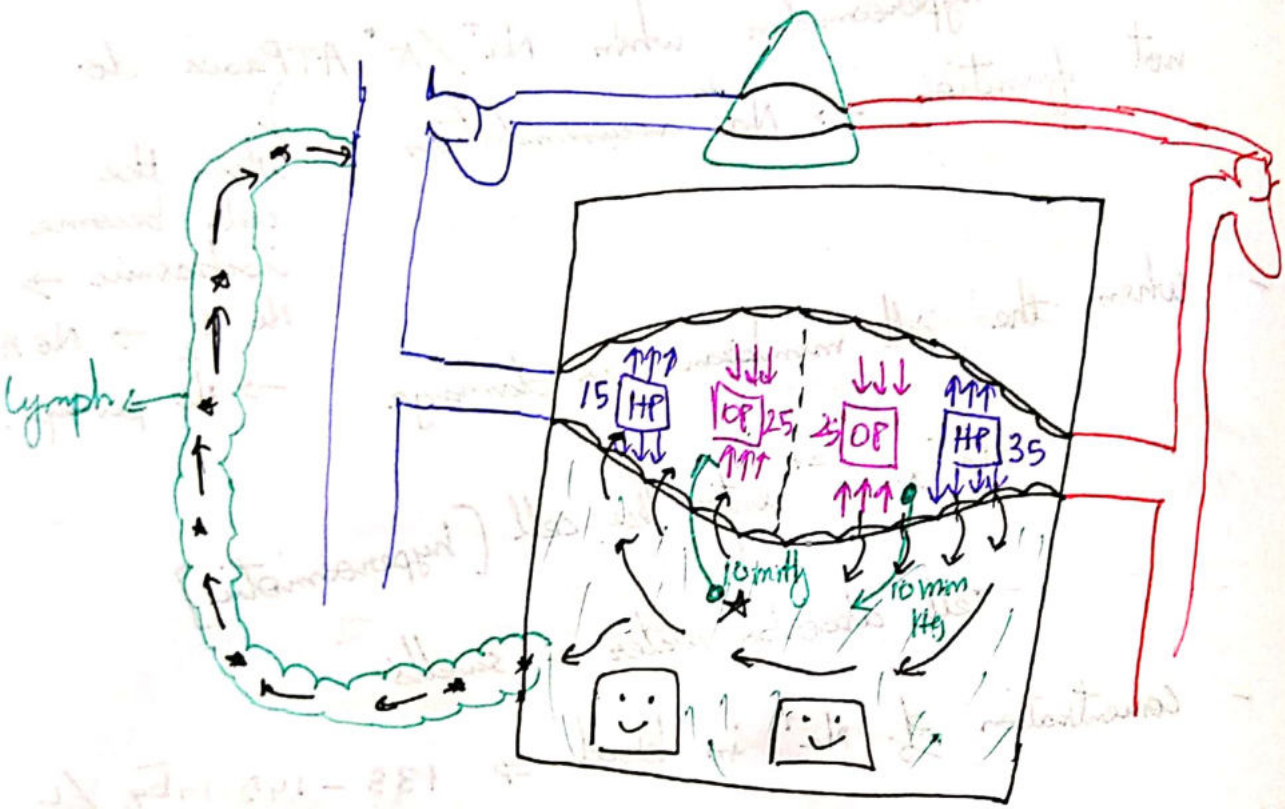
↓

Cellular edema



⇒ Extracellular edema :-

- Interstitial fluid keeps on changing ie dynamic.



- In a typical tissue fluid input is thru arterial side & fluid output is by venous side. There is also an additional lymph pathway

- Interstitial edema → Salt & water in body is too much
→ Too much fluid accumulating in interstitium
→ fluid not going back from interstitium

There is ↑ permeability of microcirculation
Lymphatics are not working — ↓ reabsorption

At arterial end

- Outward pressure from capillaries ⇒ Hydrostatic pressure on arterial side of capillaries → 35 mm Hg at arterial end

Fluid holders ⇒ Plasma proteins [Albumin] ⇒ Colloid Osmotic pressure
↳ trying to retain fluid inward
↓
25 mm Hg

Net pressure by which fluid moves = 35 − 25 = 10 mm Hg

Note:- osmotic pressure is the colloid osmotic pressure exerted by plasma prts & not interstitial prts

- At venous end of capillaries the hydrostatic pressure drops − 15 mm Hg
But osmotic pressure remains almost same as plasma prt did not leak thru capillaries

Abnormal conditions :-

① ↑ HP :-

If the ↑ HP ↑ due to any reasons → Arterial dilatation / Venous outflow obstruction

↓

Pressure for outward filtration will be more

↓

Excess amnt of fluid is coming into interstitium

&

Reabsorption at venous end is reduced

↓

The extra fluid does not produce edema immediately as lymphatics ↑ their capacity to transport fluid.

↓

When fluid coming out become so much that the lymphatics are overwhelmed

↓

Edema is produced

② [Alumin] ↓ → osmotic pressure ↓

de Hypoalbuminemia

TP = 6-8 g/dL
Albumin 3-5 g/dL
Globulin 1.5-2.5 g/dL
Fibrinogen 0.25-0.8 g/dL

Main prot to determine colloid osmotic pressure

Colloid osmotic pressure ↓

↓

More fluid leaks out at arterial end ~~less~~ fluid is reabsorbed at venous end.

↓

Edema

Note :- generalised edema => Anasarca

③ If lymphatics cannot drain well → Cancerous cells lodge lymphatics
→ Radiotherapy
→ Filariasis

- Here forces are absolutely normal
- Small amts of prt accumulate over time
  in interstitial fluid → ↑ colloid osmotic press
  in interstitium → This will hold water

↓

Edema
[lymphaedema]

Cases ① ② => Pitting edema → water is displacable
↳ Managed by diuretics

Case ③ => Non pitting edema → water is trapped with prts.

- dependent edema - distribution of edema is affected by gravity ie. more in lower parts

- Interstitium has many glycoproteins which act as water holders → absorb water → do not allow much movt of water in interstitium.
↳ has brush piles

But when excess water is filtered from capillaries a water channel is created B/w glycoprt molecules → movt of this water in the water channels is affected by gravity.

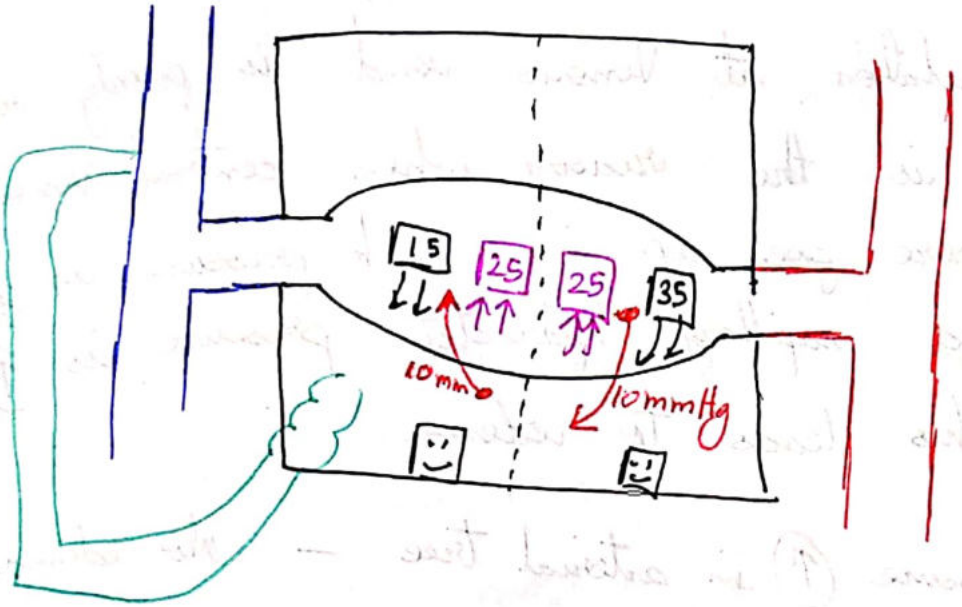
1/4 ① permeability of microcirculation - classically in inflammation.

⑤ Salt & water retention :- eg :- excessive aldosterone

Salt → Interstitial fluid → Edema
Salt & water → Blood → hypertension

Hydrostatic Pressure

- BP determines the pressure in the microcirculation ie it produces hydrostatic pressure
[CO × TPR]



- If BP goes up what will happen to capillary hydrostatic pressure ?

A.) It will also go up

But { filtration ↑ > reabsorption ↓ does not occur
no edema occurs, Why ?

A.) Bcoz of precapillary sphincter
↳ Regulator of microcirculatory bed

when BP ↑ → they constrict → don't allow excess blood in capillaries

Hence with transient fluctuations of BP - no edema occurs

No edema ← as HP does not ↑

high pressure comes → sphincter smooth muscle is stretched → Ca channels are stretched → This leads to constriction

- Microcirculation at Venous end is poorly regulated. This is the reason when central Venous pressure goes up → Back pressure is generated and capillary hydrostatic pressure is ↑. This leads to edema.

∴ Pressure ↑ in arterial tree – No edema
" " " venous system – Edema

- Cardiogenic shock – No edema as there is no pressure

⇒ Causes of ↑ hydrostatic pressure :-

- ↑ hydrostatic pressure shud be so high such that the extra fluid which is filtered cannot be drained by the lymphatics.

① Arteriodilatation :- If the pre capillary sphincters are failed.

If a part of body is heated – pre capillary sphincters fail → dilated. → Not burned

Neurohumoral dysfunction → pre capillary sphincters fail & dilate.

(2) Venous outflow is reduced :-

causes :- a) external pressure on veins
b) Thrombosis eg :- deep venous thrombosis of legs.
c) prolonged sitting.

(3) Causes of generalised ↑ HP
cardiac failure, portal hypertension
eg RV failure → ↑ central venous pressure
↓
All the capillary beds HP ↑