Toppersnotes
Unleash the topper in you

NEET-PG

PART - C

VOLUME - VII

General Medicine - II

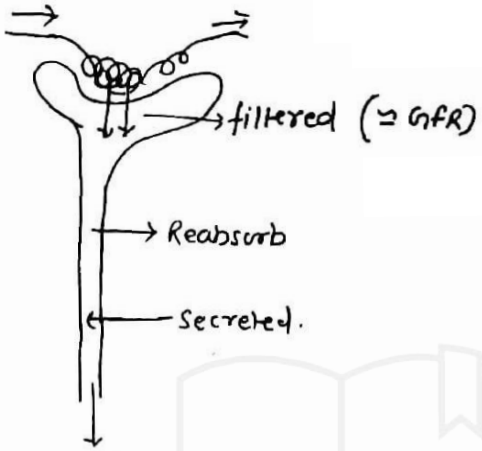
SIERRA INNOVATIONS

NEET PG

CONTENTS

Nephrology

CLEARANCE →



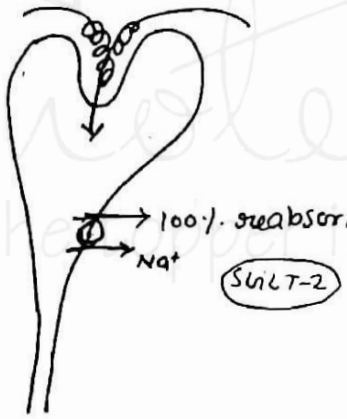
glucose clearance = 0

↓

↓↓ clearance + ve

↓

called Glycosuria.

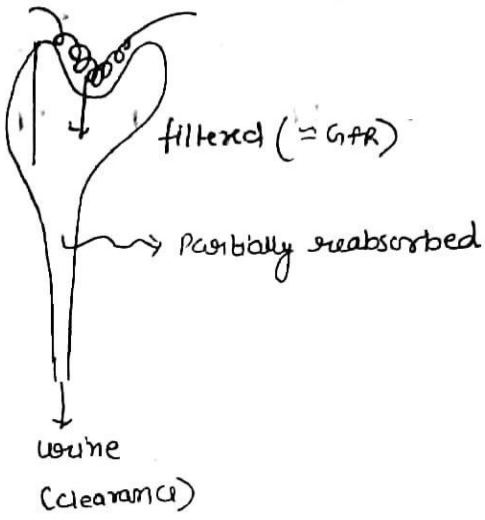


- Hyperglycemic
  - gl → >180mg%
  - ↓
  - ⊕
  - in DM
- Euglycemic
  - ↓
  - Problem in PCT
  - ↓
  - ① Proximal PCT damage.
  - ② Pt is on SGLT-2 ⊖ (Therapeutic glycosuria)
  - ③ Pregnancy (Ⓝ physiological)

[Ⓝ GFR → 90-120 ml/min] (N-clearance → 40-60 ml/min)

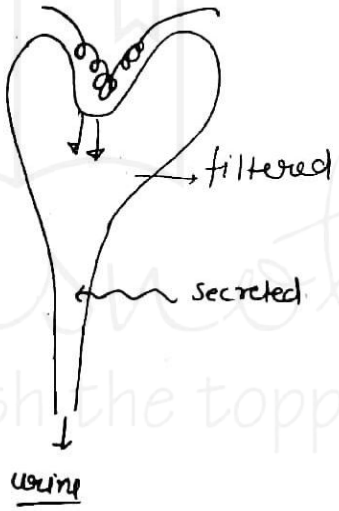
Urea →

Clearance < GFR.



Creatinine → (BIC Partially secreted)

Clearance > GFR



Inulin →

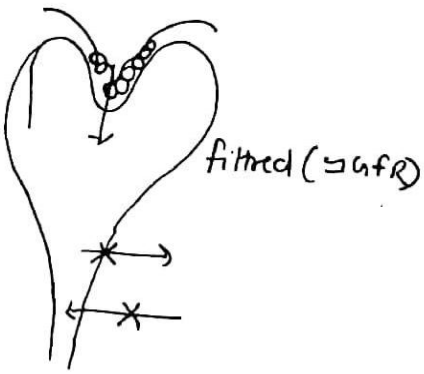
Clearance = GFR

↓

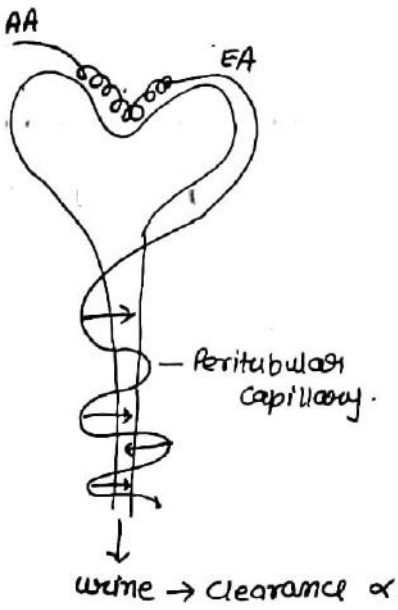
90-120 ml/min.

↓

[gold standard for GFR]



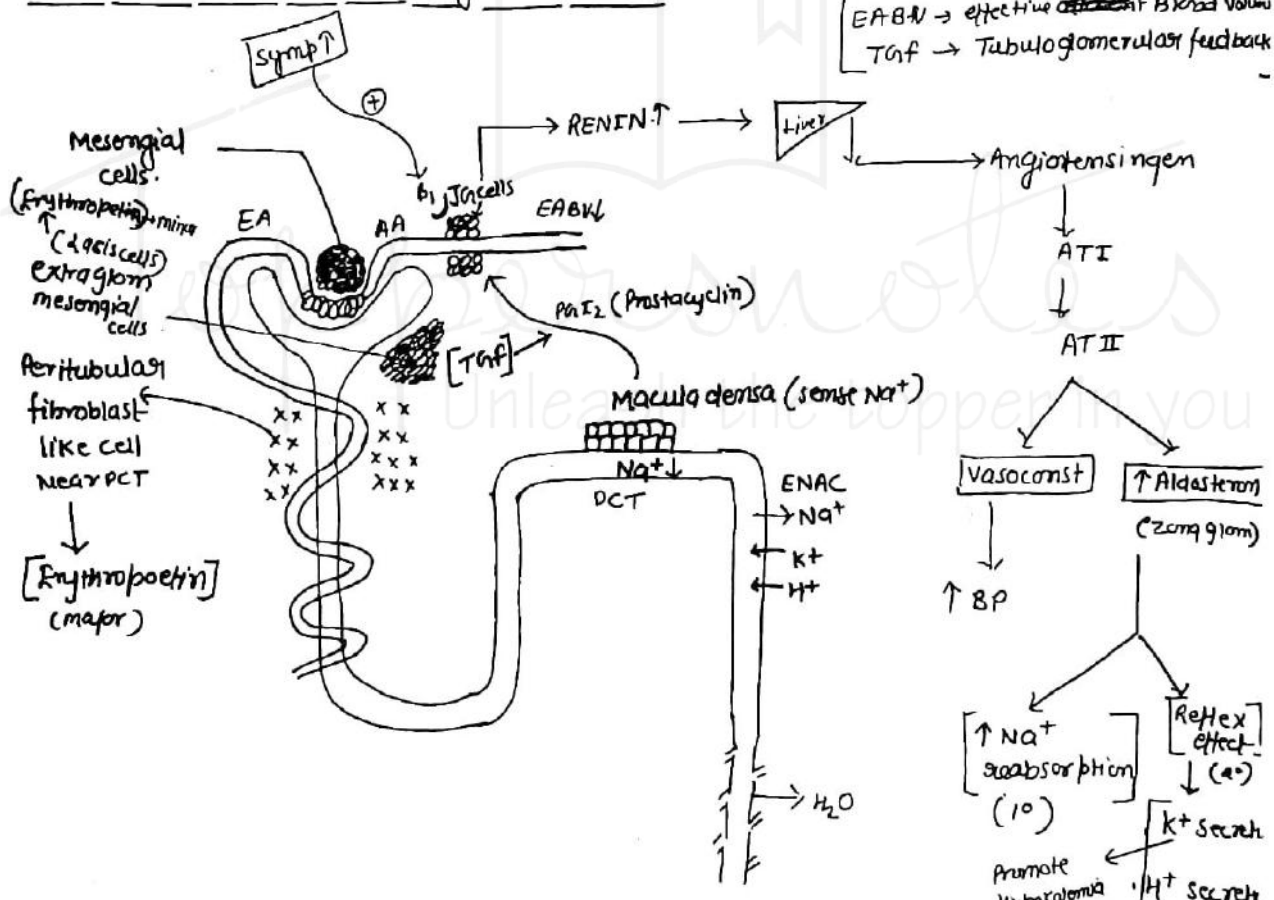
PAH → (only secreted)
↓
clearance ∝ RBf
↓
625 ml/min.



urine → clearance ∝ RBf

Renal Mechanism to Regulate GfR :-

arterial
EABN → effective Blood Volume
TGF → Tubuloglomerular feedback



Amount of Na+ In Body ↓ → PGI2 Secreted by mD cell → ⊕ JG cells
↓
↑Renin
↓
↑ Na cubs

Secondary effect of AT II → ⊕ ↑ ADH ($\uparrow H_2O$)

→ ⊕ Thirst center ($\uparrow H_2O$)

→ act on hypothalamus → ↑ symp flow (in CNS)

⇒ assume filtration ↑↑ → conc of filtrate in Gl. capsule High

→ conc of subs ↓ in Peritubular cap.

↓

[Promotes reabsorption]

↓

(Preserve substance)

⇒ assume filtration ↓↓ → Promotes secretion

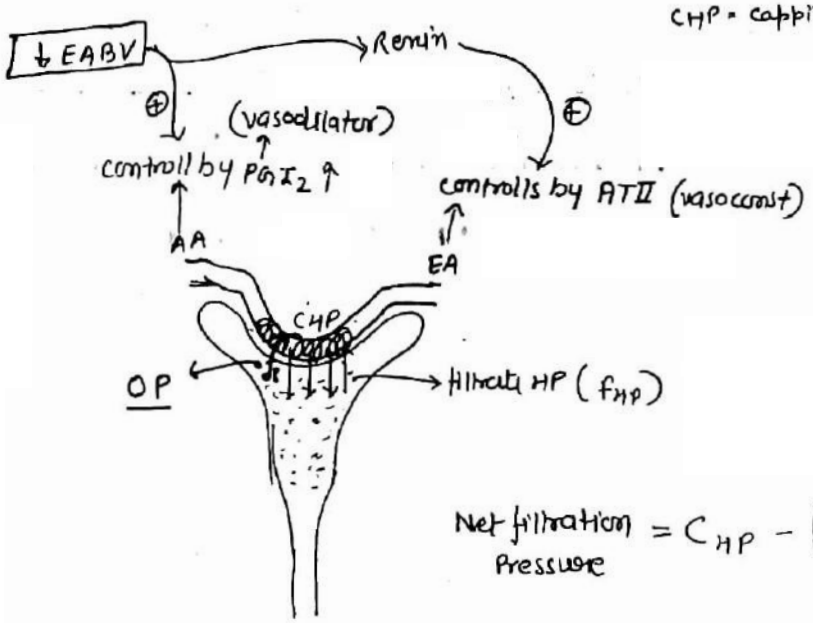
↓

(wasting substance)

called glomerulo tubular Reflex.

- { Reab / Secr } tubular.
- { GfR } → filtrate
- urine → final

CHP = capillary HP



Net filtration Pressure $= C_{HP} - [f_{HP} + OP]$

$= 55 - [10 + 35]$

$NfP = 10$ mmHg.

$GfR \propto K_f \times NfP$

↓

filtration coefficient

$\simeq GfR = \downarrow K_f \times NfP \uparrow$ ⇒ In early Stages of CKD

↓ (SA ↓↓)

↓ Surface area of Nephron (kidney)

↑ filtration load in Normal remaining Nephrons.

AA diam	EA diam	NfP / GfR
N	↓	↑
↑	N	↑
N	↑	↓
↓	N	↓
MI ↑	↓	↑↑
↓	↑	↓↓

AAdiam ↑ EA ↑ GFR/NFP (N)

↓ ↓ (N)/↓

Diabetic Nephropathy →

⇒ Thickening of BM of EA in diabetic Nephropathy (1st in D. Nephropathy)

⇒ ↓ EA diameter → ↓ Peritubular Blood flow ⇒ Ischemia

Ischemia → distal Renal papillary Necrosis

Infection → vasoconst of EA

↓ dit glycosylation of Bm Protein → cover -ve charge of BM

[↑ GfR] → ↓↑ Physiological change in D Nephropathy.

→ dit hyperfiltration Injury

↓ damage filtration slit. (No BM repulsion of albumin)

→ Proteinuria (Microalbuminuria)

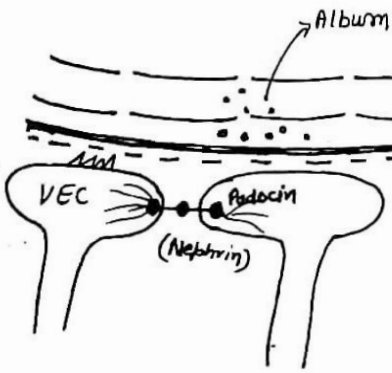
[HbA1c → best target to know disease]

↓ first detectable change in D. Nephropathy

↓ convert

change in overt-proteinuria.

-ve charge of BM ↑ Phospholipase A_2 R ↑ foot Process of Podocytes (Visceral epithelial cell)



made up of type IV collagen

N end → Collagenous domain → Produce Anti GBM antibody

C end → Non collagen domain → [can become Antigen]

CKD → hyperfiltration injury to Remaining Nephron → damage to filtration slit

↓ Proteinuria

CKD ↓ overall GFR ('d/t ↓↓↓ Kf') [B/c total No of Nephron ↓]

↓ eGFR (estimated GFR)

↓ GRR of entire kidney.

→ ACE ⊖ → dilate the EA → ↓ filtration press → glycosylation ↓

↓ Protein.

ACE ⊖ ↓ [DOC for Diabetes Nephropathy]

⇒ CKD Pt c̄ Proteinuria →
- Less salt
- Less fluid

~~already ↓GFR~~

~~150 ... ~~

↓ give ACE⊖ (DOC) → ↑ Nephron perfusion of other area of kidney.

↓ ↓ filtration load of kidney → [fall in GFR acceptable]

↓ ↓ Proteinuria

[fall in GFR acceptable] ↓ {maxm → 25% ↓GFR; → 35% ↑ Cr}

→ ACE ⊖ → Hyperkalemia.

↓ Limit $K^+ > 5$ ↓ C/I to ACE⊖

during Rx of ACE⊖ ↓ cutoff ≤ 5.5 ↓ ≥ 5.5 ↓ C/I to continue ACE⊖

ACE⊖ ⟶ ↑ Bradykinin → dry cough
→ Nasal stuffiness

- Avoid ACE ⊖ ⟶ ① $K^+ > 5$ (absolute C/I)
② Acute kidney injury (Relative C/I)
↓
If Prerenal failure.

Creatine is Never C/I to ACE⊖

Analgesic Nephropathy ⟶

NSAIDS ⟶ Blocks PGI_2 ⟶ ↓ GFR → damage to filtration → Proteinuria

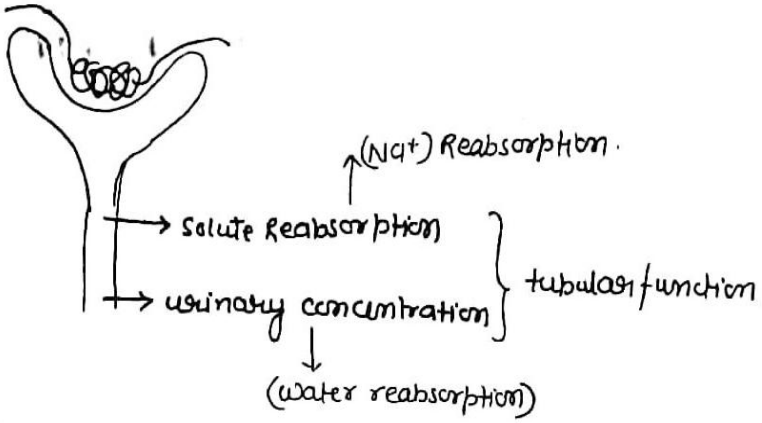
↓ GFR
- ↓ filtrate ⟶ ↑ Na^+ in urine
↓
Polyurea
(Nocturnal Polyurea)
(Nocturia)

↓ Peritubular capillary blood flow
↓
- damage proximal Nephron
↓
Renal papillary Necrosis

Tubular functions of Kidney →

→ ① Solute Reabsorption

→ ② Urinary Concentration.

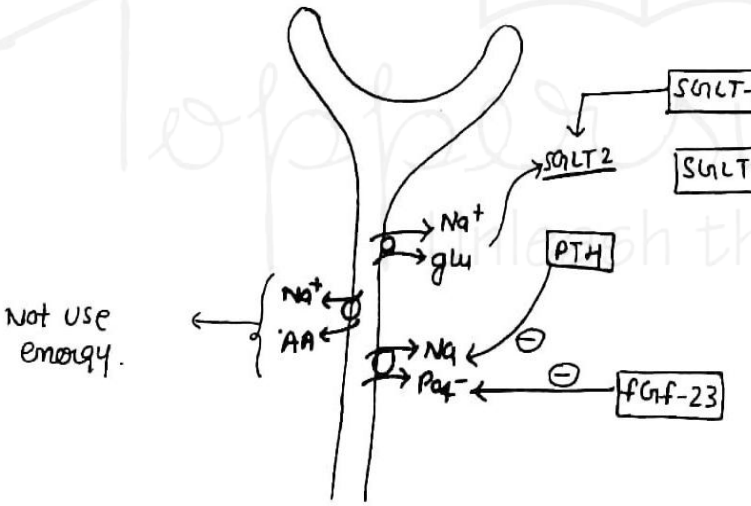


PCT functions →

① Solute Reabsorption. (>65% of all solute absorb by PCT)

[100% glucose]

Mg^{++} → TAL ⊕ > DCT



⇓

MA → causes therapeutic glycosuria

↓

S/E ⊖ dehydration

↓

DKA (euglycemic)

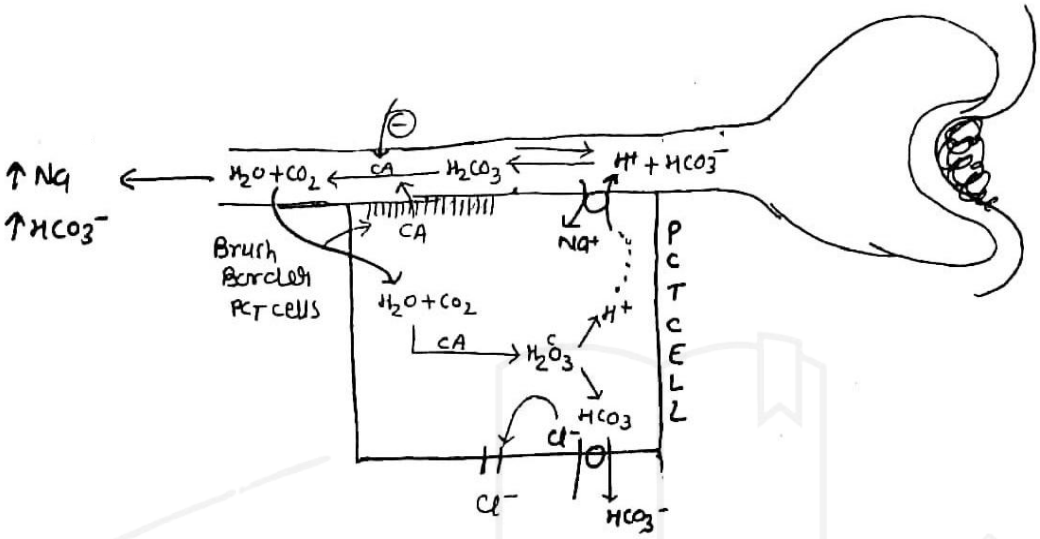
② Recurrent UT infection

↓

[Candida]

(Balanoposthitis)

② Bicarbonate (HCO_3^-) reclamation →



CA ⊖ (Acetazolamide) → H_2CO_3 → $H^+ + HCO_3^-$

→ ↑ Na^+ in urine (Natriuresis) → diuretic action (weak)

→ Bicarbonaturia → metabolic acidosis

DOC

Acute cong glaucoma

↓ aqueous production (H_2CO_3 —CA⊖→ $H_2O + CO_2$ ↓↓)

↓ CSF Production → use in ICH

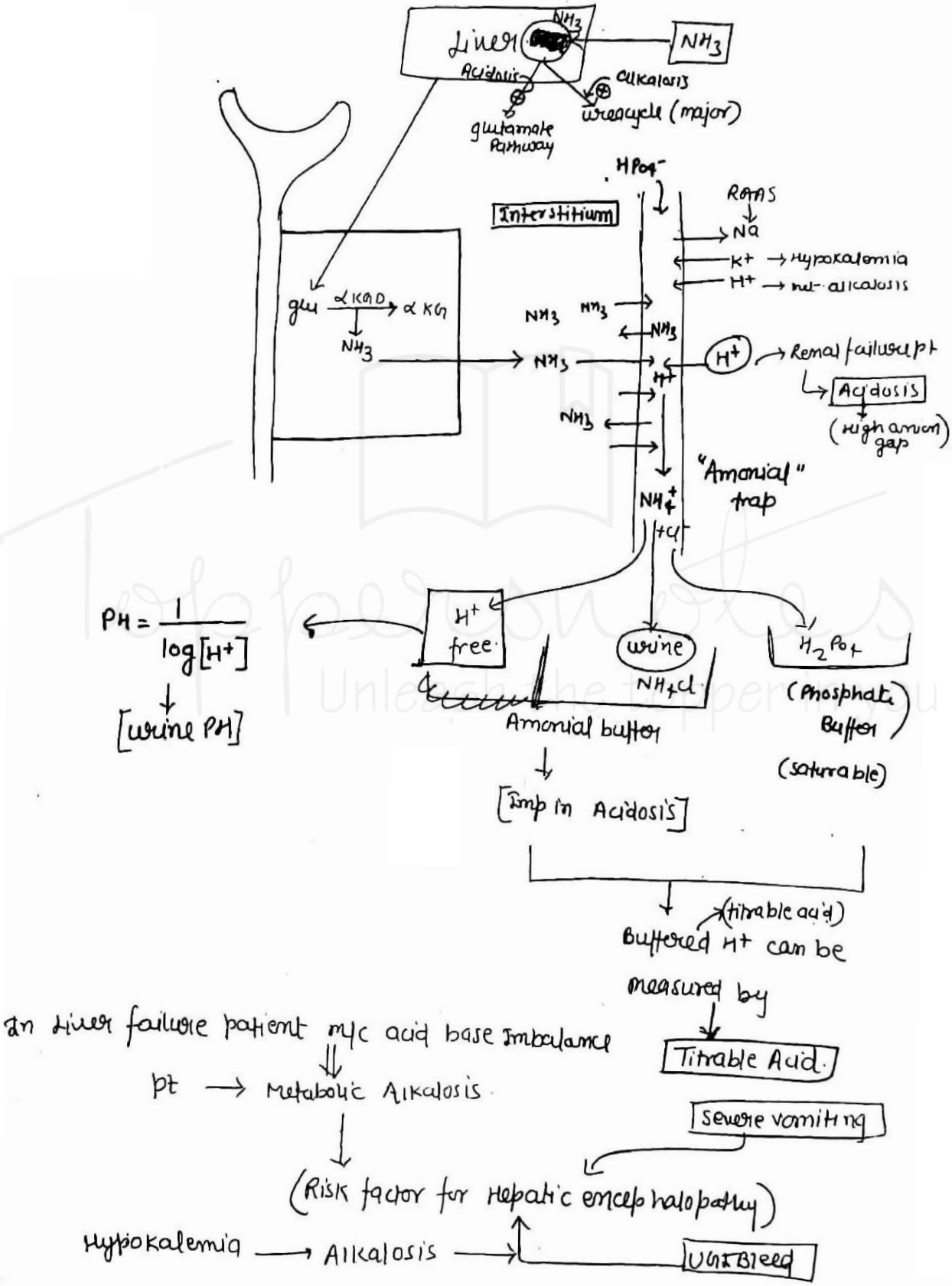
Dox → High altitude Pulmonary edema

↓

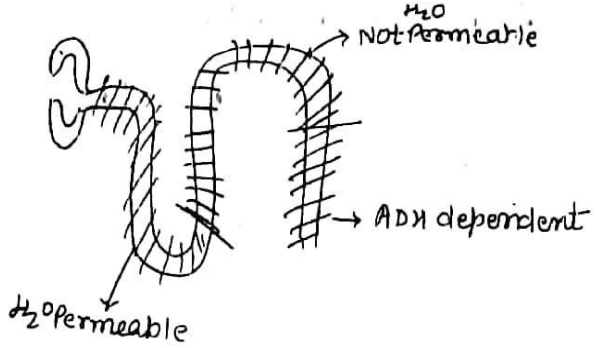
TOC → Rapid descend

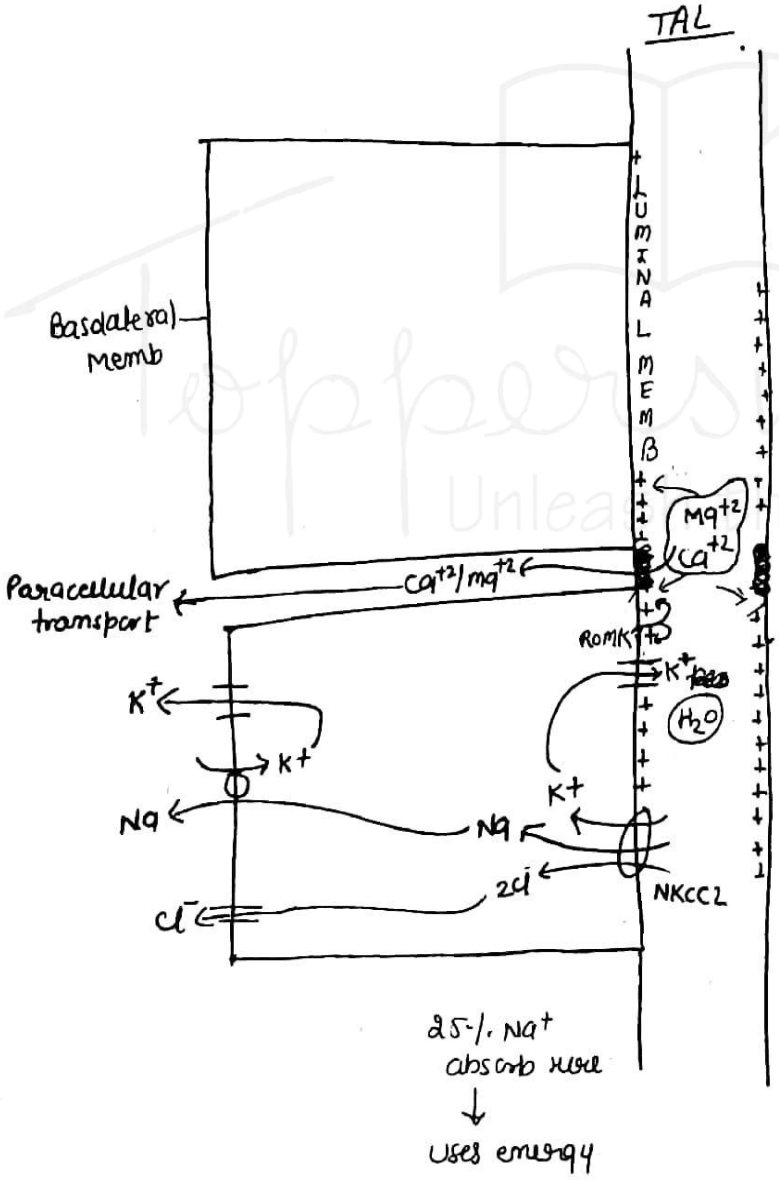
③ Vit D Production (1α Hydroxylase)

④ Amoniagenesis. →



THICK ASCENDING LIMB :-





ROMK → Renal outer medullary K^+ → Conc on side of cell

Loop diuretic → ⊖ NKCC2 → Natriuresis → ~~↓~~ Hypervolumia ✓

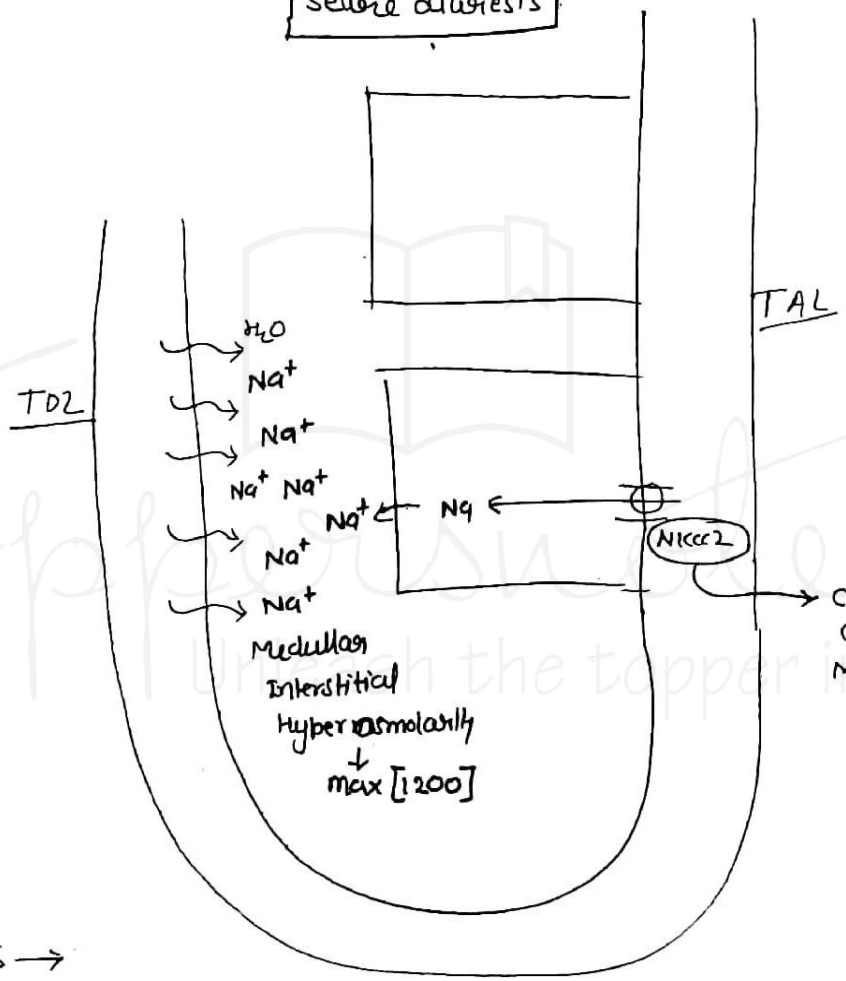
- furosemide
- Ethacrynic acid
  - ↓ CA activity also

↓ Medullary hyperosmolarity

↓ less water reabs from PCT

↓ dilute the urine.

↓ Severe diuresis



Loop diuretics →

Natreuresis
Diuresis
} Mild Hypo Natremia (125-135) (B/C Both Na + H_2O loosing)

↓ more volume loose

+++ → RAAS

RAAS → Hypokalemia (severe) } d/t aldosteron
RAAS → Met. alkalosis }

RAAS → ENAC (cap 3-5%) → Load dilute → ~~NAC~~ NKCC-2 → 25% capacity

↓

Can't compensate

↓ Ca^{++} & Mg^{+} Reabsorption ⟶ ↑ urinary Ca^{++}

↓

Hypocalcemia

Hypomagnesia

↓

STONES

Loop diuretics works only in Hypervolumic state

↓

Not Produce Hypovolumia

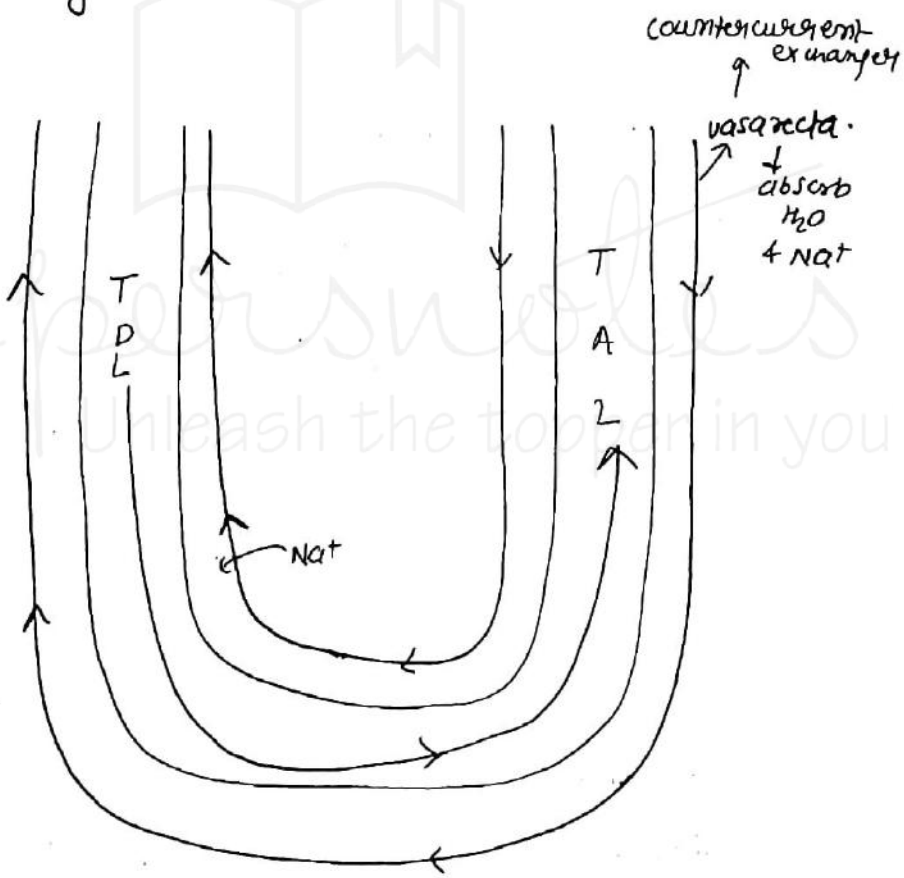
→ Ototoxicity (SNHL) → (Reversible)

↓

High frequency

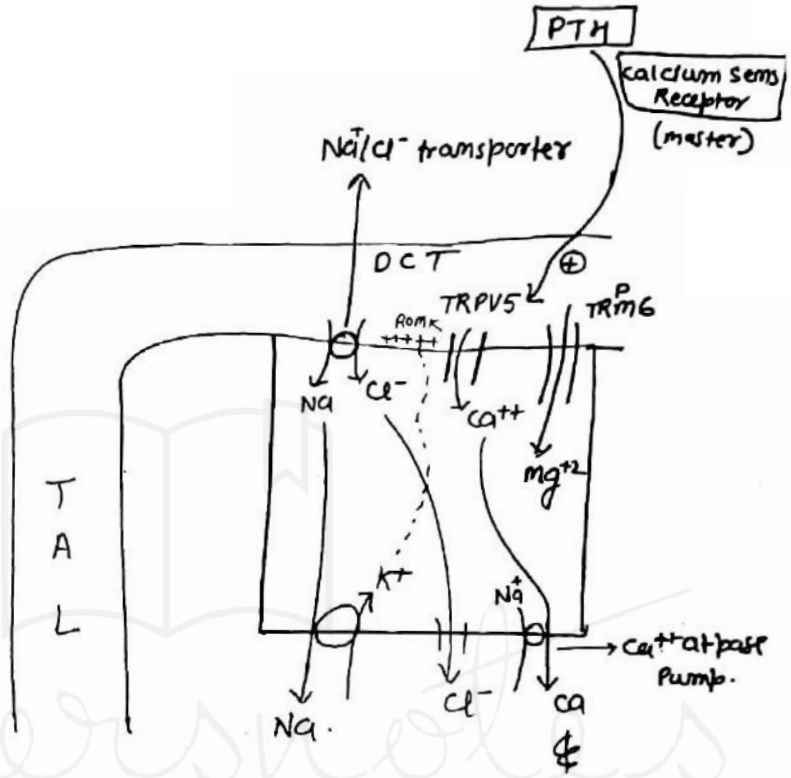
Loop diuretic → abused to Excessive water loss

counter current exchanger →



TAL → counter current multiplier

DCT →



→ CASR → regulate Ca^{++} abs

→ PTH → ↑ Ca^{++} Regulati

→ ROMK → ⊗ mg absorption through → TRPM6 Receptor Chanel
(K^+)

Ca^{++} / Mg^{++}
- In DCT → absorb throug trancellular Roul-
- TAL → Intercellular Roul-

Pass conc urine

Thiazide diuretic → ⊖ Na/Cl^- transporter → Natriuresis (High) → capacity 7-8%
→ Diuresis (little)

(Hydrochlorothiazide) → Not affect the medullary Int Hyperosmolarity (O/C It is cortex)