

Renal revision

By

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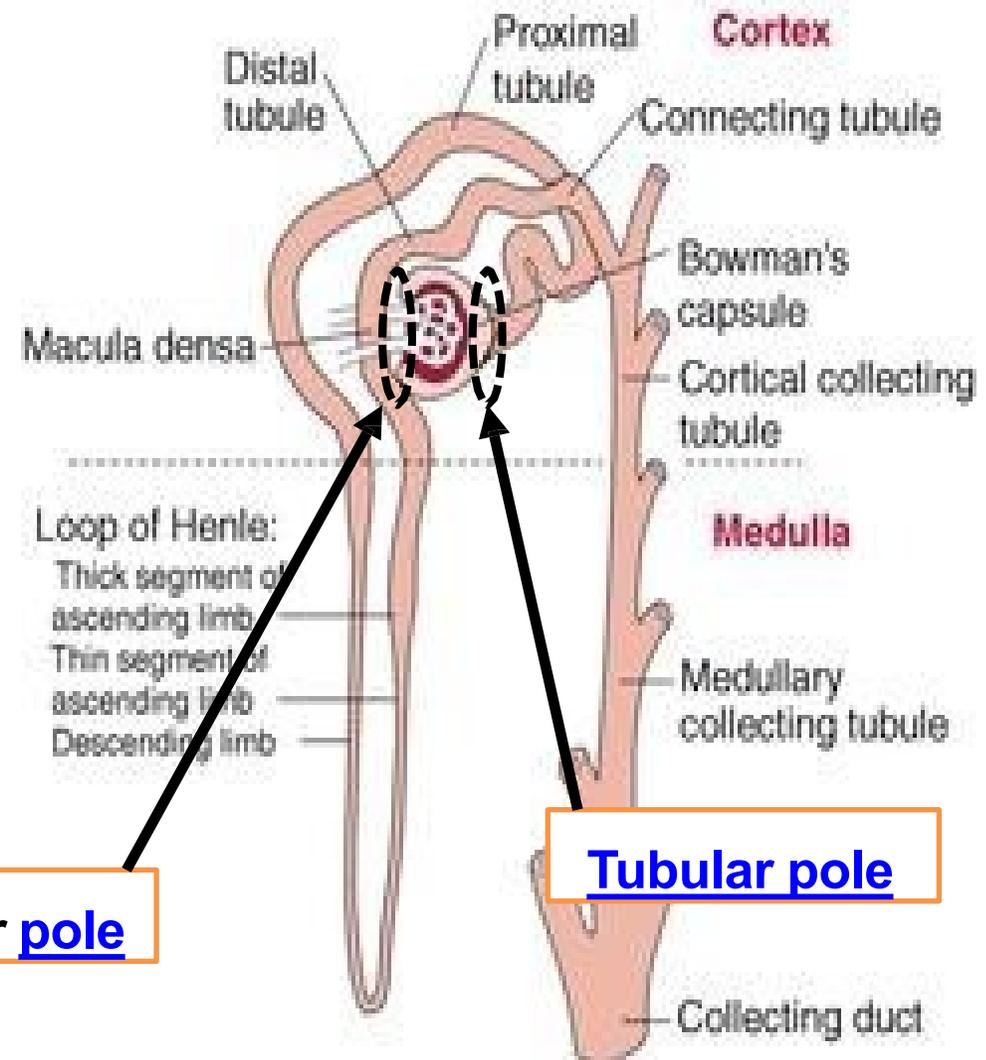
Juxta-glomerular Apparatus

Def.,:

- It is a combination of **vascular, tubular** and **interstitial** cells

Site:

- **Vascular pole** of Bowman's capsule



Juxta-glomerular Apparatus

Components:

Macula Densa cells

In transitional zone between thick ALH and DCT

Densely crowded tubular cells

JG cells

In media of afferent arterioles

Modified smooth ms cells with epitheloid appearance

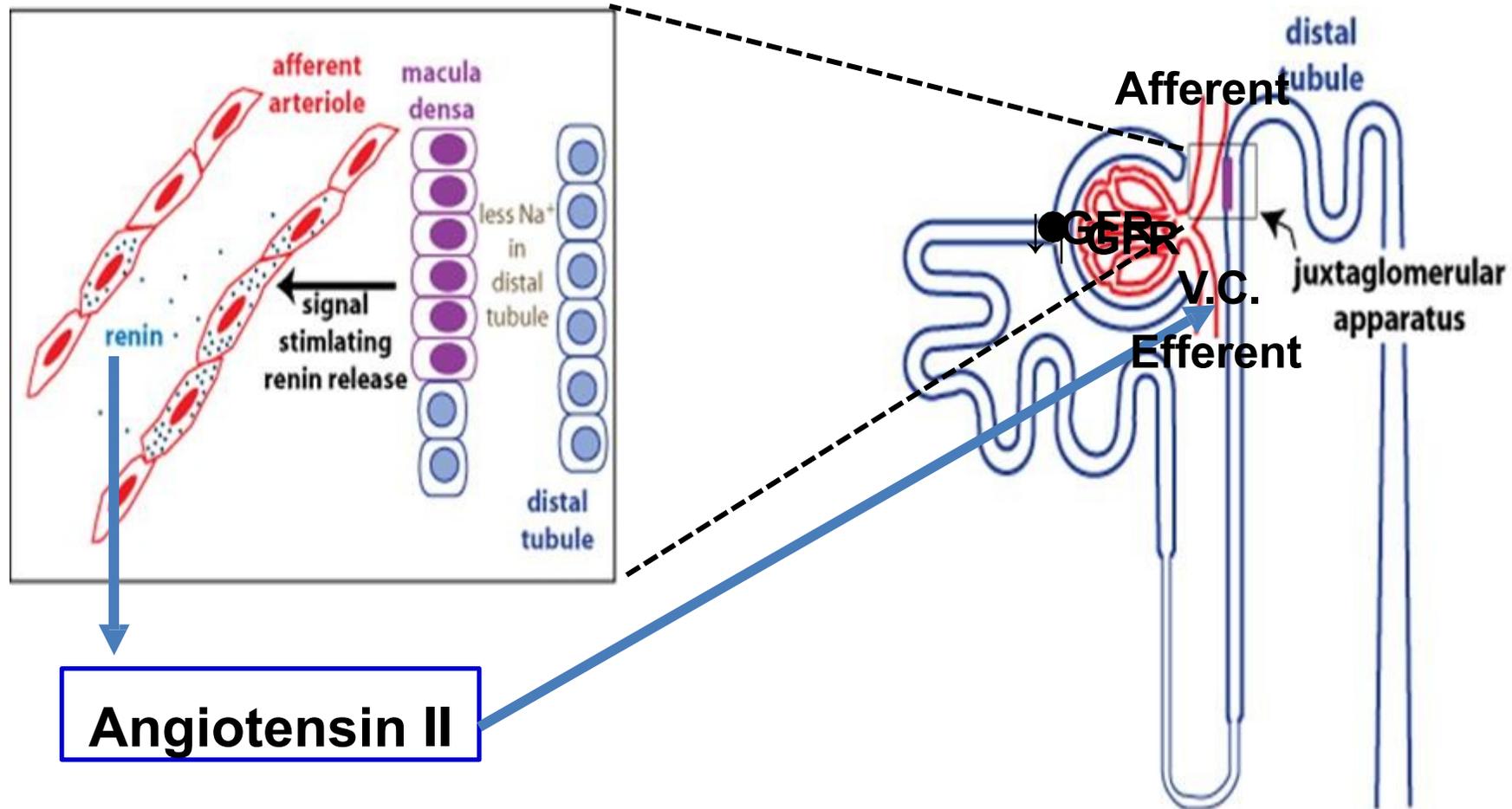
(E.G. mesangial) or Lacis cells

Continuous with intraglomerular cells

Interstitial cells between JG and MD cells

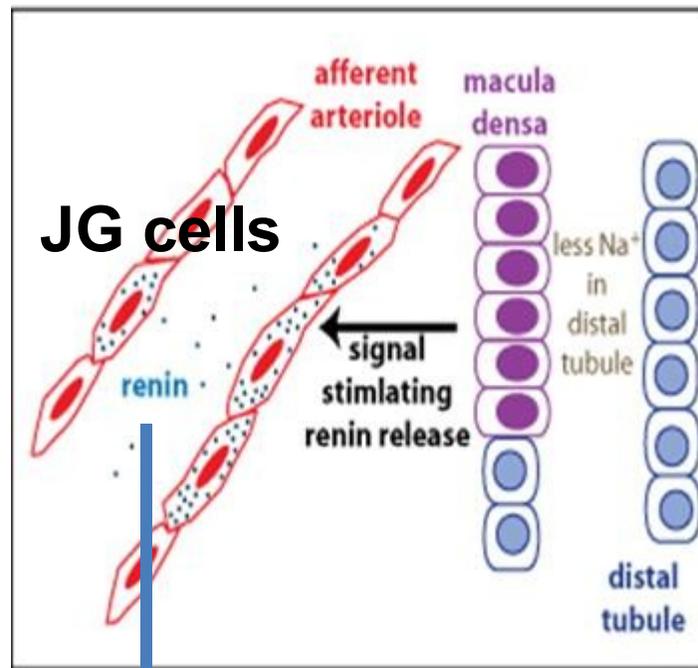
Macula Densa Function

- Monitor NaCl concentration in DCT (stimulated by low NaCl)

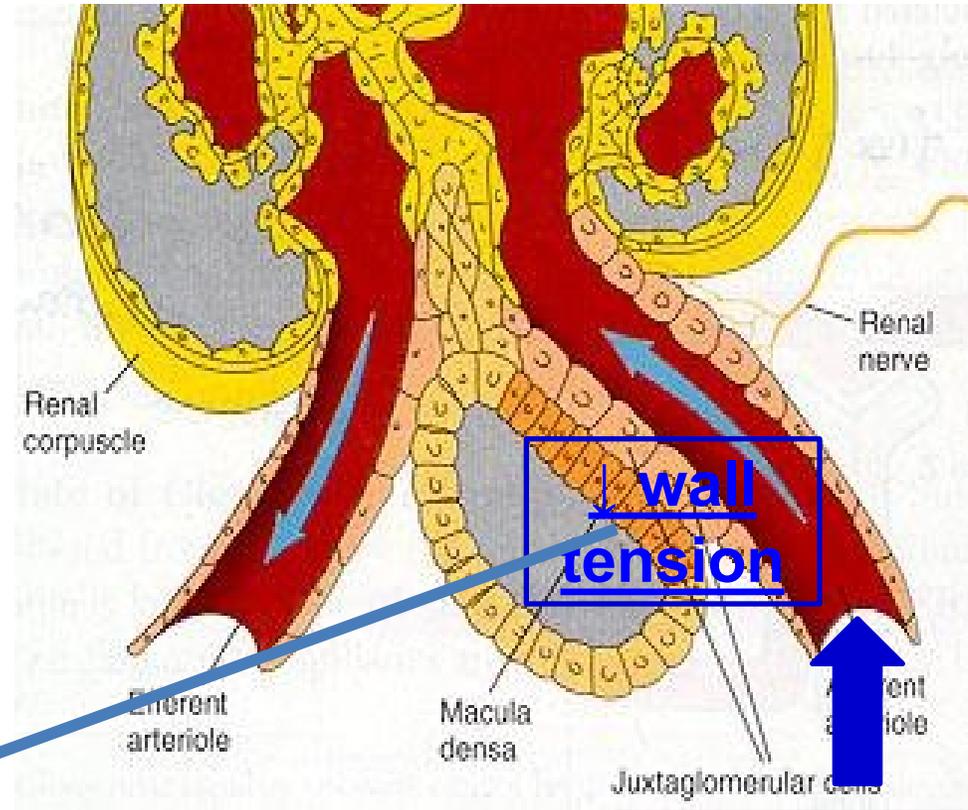


Juxta-glomerular cell Function

- A) Synthesis, store and release of renin
- B) acts as Baroreceptors (detect tension in wall of afferent arterioles)



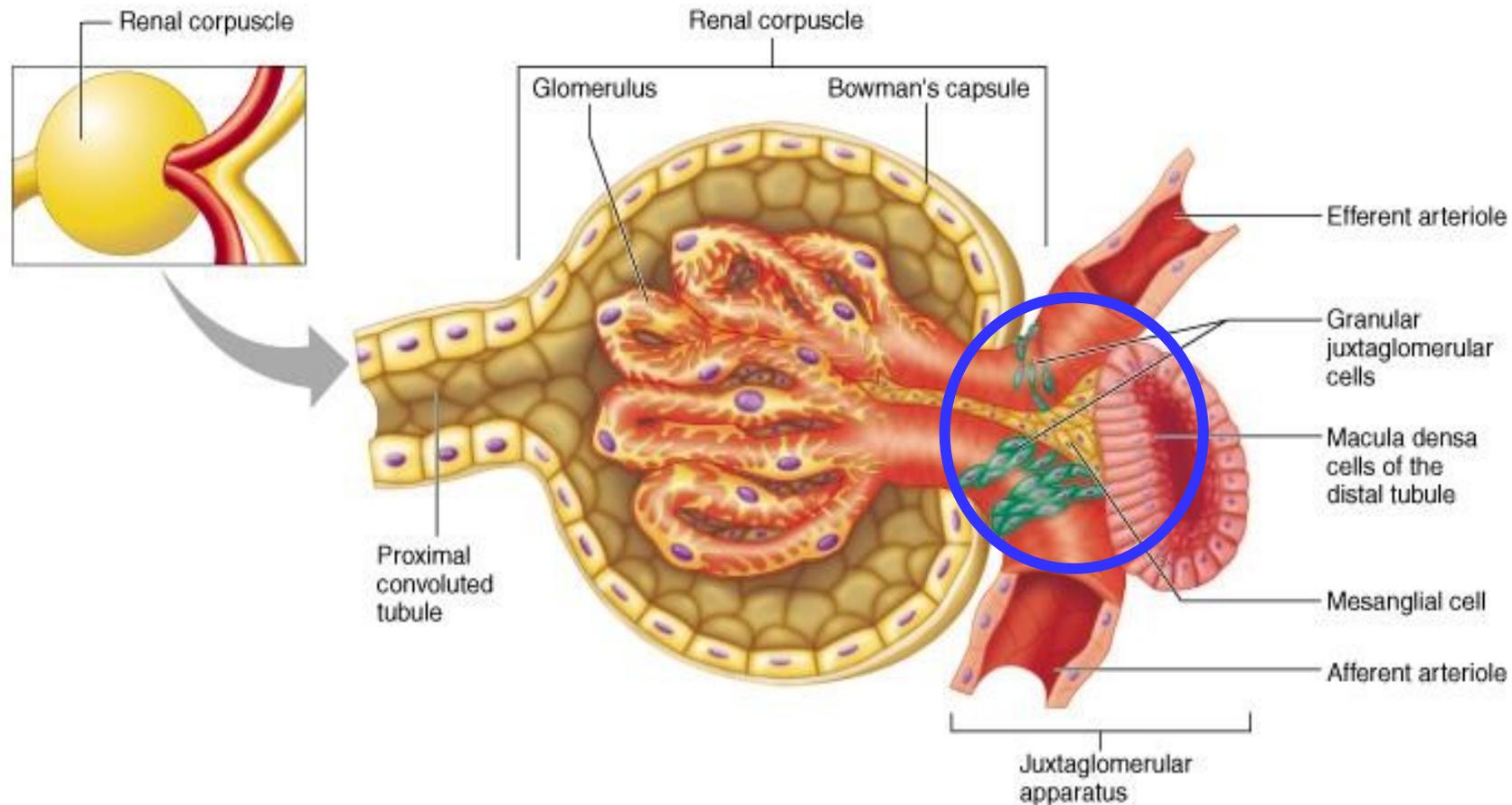
Renin



↓ Renal Blood Flow

Extra-glomerular Mesangial cell or Lacis (Polkisson) Function

- Form functional syncytium with macula densa and JG cells



MCQ

Which part of the nephron monitors sodium chloride concentration in the distal convoluted tubule?

A) Juxtaglomerular cells

B) Podocytes

C) Macula densa

D) Mesangial cells

E) Bowman's capsule

Answer: c



Renal blood flow

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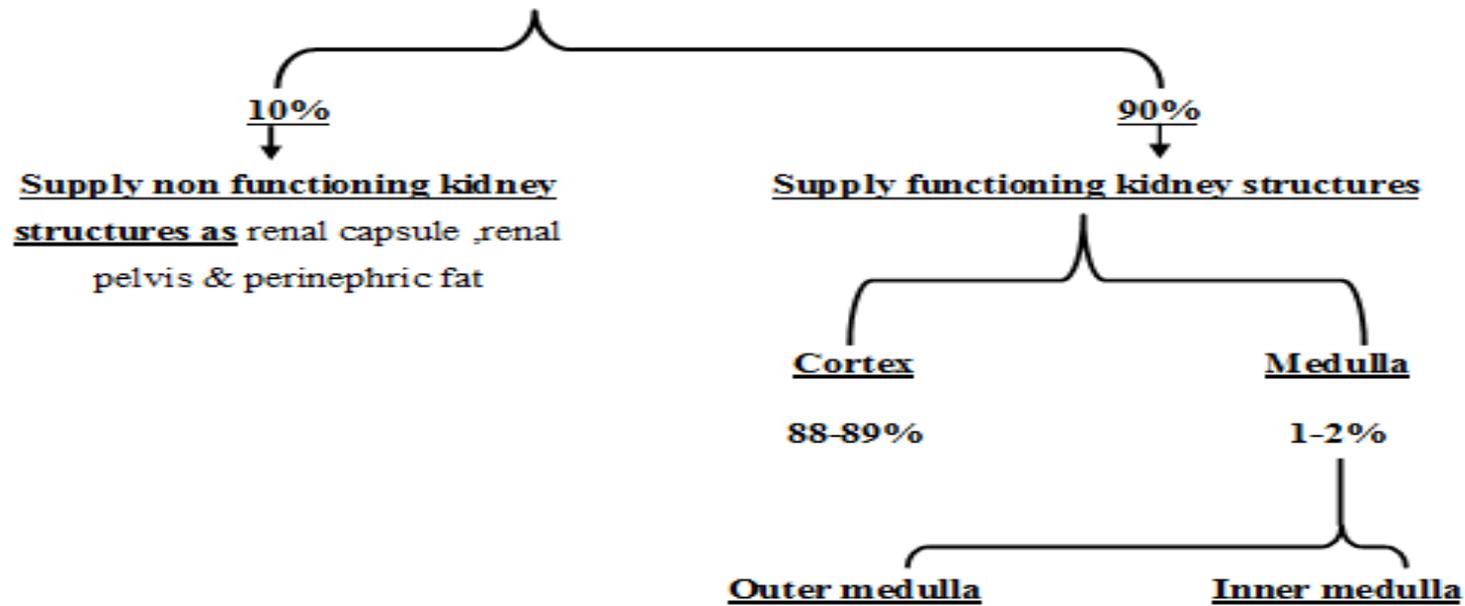
Renal Blood Flow (RBF)



- Renal fraction:

- It is the fraction of COP that supplies both kidneys.
- It is about: $\frac{1}{4}$ COP, or 1200 ml/min, or 4ml/min/gm kidney tissue.
- This high RBF is to ensure high GFR.

- Distribution of RBF:



- Relative low BF in medulla is due to:**
- 1- Small number of bV_s.
 - 2- High length of vasa recta.
 - 3- High viscosity of medullary blood due to high medullary osmolarity.

Distribution of RBF



- **Regulation of RBF:**

- (I) Intrinsic regulation (Auto-regulation) of RBF:**

- **Def:** It is the ability of the kidney to maintain nearly **constant RBF & GFR** in spite of changes in ABP in the range of **80-180 mmHg**.

- **Significance of auto-regulation:**

- **Minimize the effects of changes in ABP** on Na & H₂O excretion.

➤ Mechanisms of auto-regulation:

1-Myogenic theory:

- ↑ ABP → stretch of the blood vessels → ↑ Ca^{2+} influx of smooth muscle fibers of afferent arteriole → Vasoconstriction (VC) → ↓ RBF to its normal level.
- ↓ ABP → opposite mechanism

2- Tubuloglomerular –ve feed back mechanisms:

- ↑ ABP → ↑ RBF → ↑ GFR → ↑ NaCl delivery to macula densa → release of adenosine → VC of the afferent arteriole → ↓ RBF to normal.

▪ ↓ ABP → ↓ RBF → ↓ GFR → ↓ Na⁺ delivery to macula densa →
release of PGI₂ →

□ VD of afferent arteriole.

□ Stimulation of JG cells → release of renin → formation of
angiotensin II (A II) → VC of efferent arteriole → ↑ glomerular
hydrostatic pressure → ↑ GFR to normal.

(II) Extrinsic regulation of RBF:

1- Neural regulation of RBF:

2-Chemical regulation of RBF:

Renal vasoconstrictors	Renal vasodilators
1- Endothelin. 2- ADH & serotonin. 3- Thromboxane A ₂ 4- Angiotensin II: <ul style="list-style-type: none"> • <u>Small dose</u> → preferential VC of <u>eff arteriole</u> → protect GFR • <u>Large dose</u> → VC of <u>aff & eff arterioles</u> → ↓ RBF & GFR. 	1- Nitric oxide. 2- Dopamine. 3- Bradykinin. 4- ANP. 5- Prostaglandins (PGI ₂ , PGE ₂)

MCQ

What is the renal fraction of cardiac output (COP)?

- a) 10%
- b) 15%
- c) 20%
- d) 25%
- e) 30%

Answer: d



Glomerular filtration

By

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Glomerular Filtration



- **Def:** It is the **bulk flow of a solvent** through a filter, carrying with it solutes that are small enough to pass through the filter.

- **Composition of glomerular filtrate:**

It is an **ultra filtration** because it is a **plasma minus plasma proteins.**

• Filtering membrane:

	1- Capillary endothelium	2- Basement membrane	3- Bowman's capsular epith.
<u>Functions</u>	Prevent passage of <u>blood cells and platelets.</u>	Prevent passage of <u>plasma proteins.</u>	(1) Lay down & maintain <u>basement membrane.</u> (2) <u>Phagocytose</u> escaped macromolecules.

Glomerular Filtration Rate (GFR)



- **Def:** Volume of plasma filtered **by both kidneys** per unit time.
- **Value:**
 - **125**ml/min in human adult.
 - **180** L/day.
- **Filtration fraction:**
 - It is the fraction of RPF that is filtered in the glomeruli.
 - Filt. Fraction = $GFR/RPF = \underline{\underline{20\%}}$.

• Dynamic of glomerular filtration

(I) Forces Increasing filtration

1- Hydrostatic pressure of glomerular capillaries (G_p): (60 mmHg)

- The only force that promotes filtration.
- Normally, this is the main factor that determine GFR.
- Causes of high pressure: see before

□ ***This pressure can be altered by:***

a) Changes in ABP.

b) Balance between aff & eff arteriolar resistance

2- Oncotic pressure of protein in Bowman's space ($B\pi$):

- **Very little if any** protein is present

- This factor can be **considered zero (neglected).**

(II) Forces decreasing filtration

1-Oncotic pressure of plasma protein ($G\pi$): (32 mmHg)

- If \uparrow e.g. hyperproteinemia, dehydration \rightarrow
 \downarrow GFR.
- Leakage of plasma albumin from
glomerular membrane $\rightarrow \downarrow G\pi$ and $\uparrow B\pi$
 $\rightarrow \uparrow$ net filtration force $\rightarrow \uparrow$ GFR.

2-Hydrostatic pressure in Bowman's space (Bp): (18 mmHg)

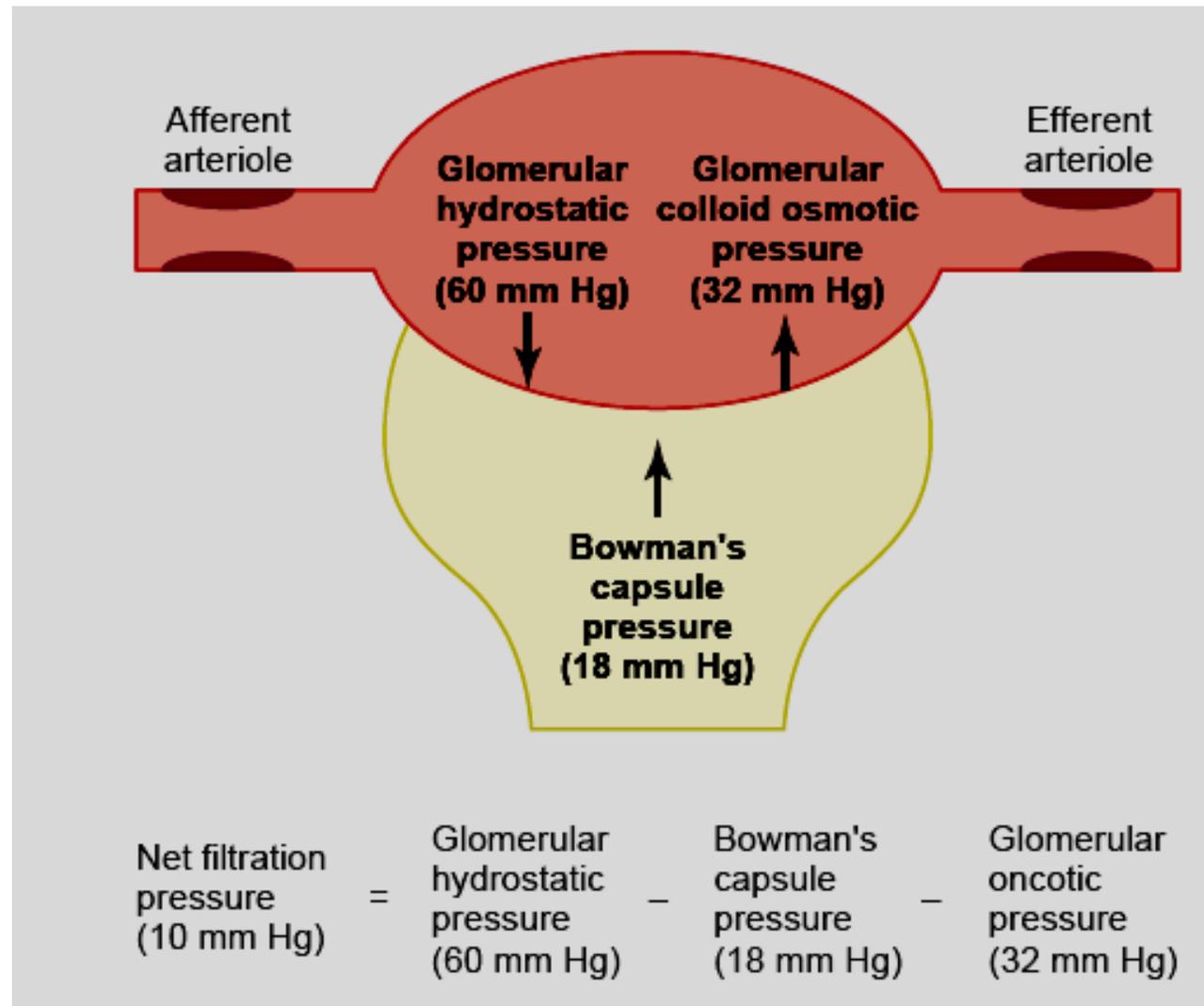
□ Importance:

a) Maintain the tubule patent.

b) Acts as a driving force to propel GF along the whole length of the renal tubule.

c) Detect the NFF (net filtration force)

□ If ↑ e.g. ureteric obstruction → ↓ GFR.



Dynamic of glomerular filtration or forces of filtration

• Factors affecting GFR:

- 1-Hydrostatic pressure of glomerular capillaries (Gp)**
- 2-Bowman's capsular hydrostatic pressure (BP)**
- 3-Oncotic pressure of plasma proteins (G π)**
- 4-Effect of renal plasma flow (RPF)**
- 5-Filtration coefficient (KF)**

MCQ

What is the normal GFR in a healthy adult human?

- a) 50 mL/min
- b) 100 mL/min
- c) 125 mL/min
- d) 180 mL/min
- e) 200 mL/min

Answer: c



Renal Tubular Functions

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Renal Tubular Functions



- **Urine is formed by two major processes:**

1. Glomerular filtration.
2. Tubular transport: reabsorption and secretion.

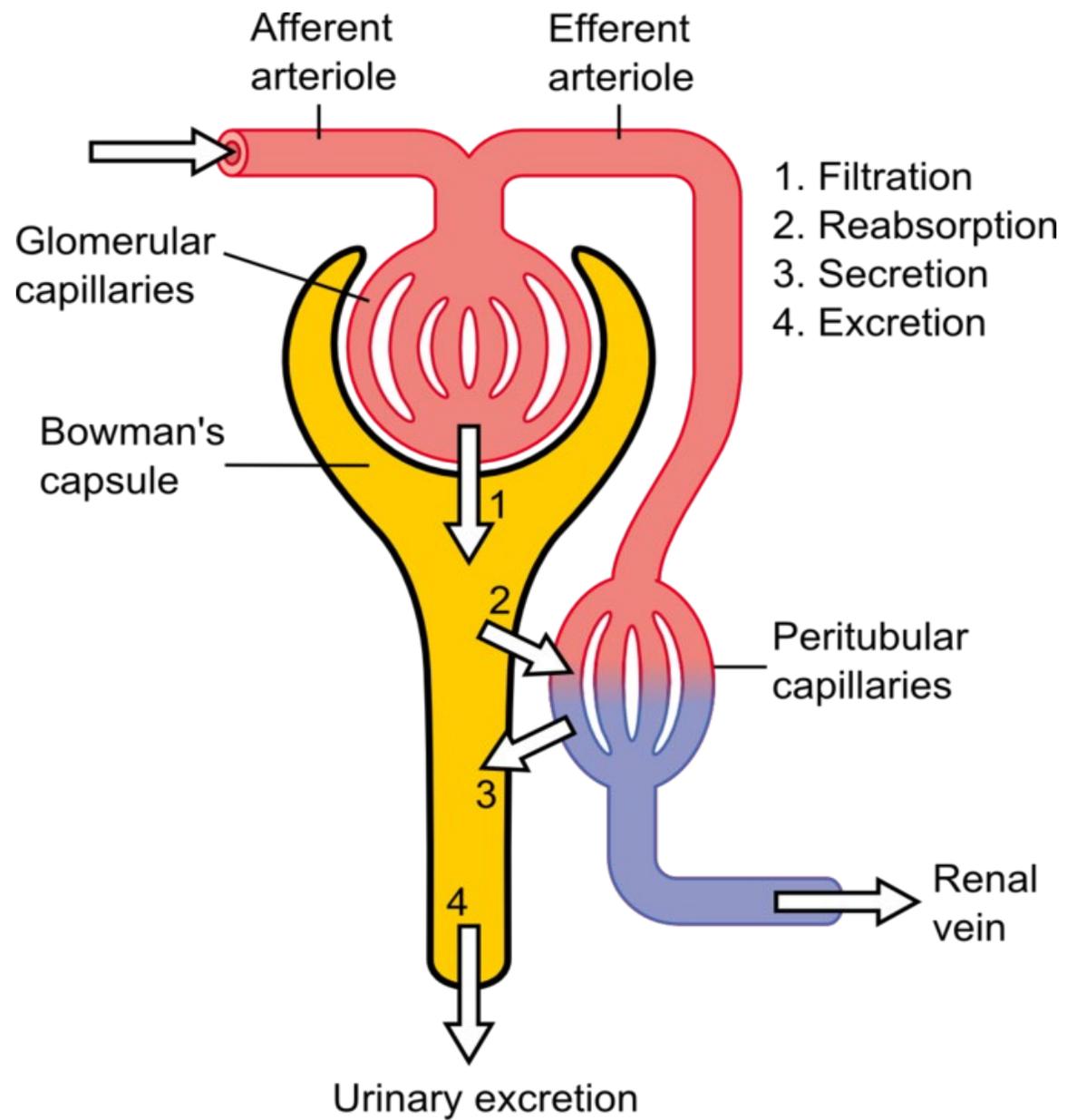
- **Tubular Transport:**

- 1- Reabsorption:**

Transport of materials from tubular lumen (filtrate) to interstitial fluid to peritubular capillaries (PTC).

- 2- Secretion:**

Transport of materials from blood in PTC to interstitial fluid to tubular lumen.



Basic kidney processes

Proximal Tubule (PT)



- **Overall Function of Proximal Tubules**

- a) Reabsorption of:**

1. All filtered glucose, amino acids, & vitamins.
2. About 2/3 (67%) of filtered load of Na^+ & water.
3. About 90% of the filtered load of HCO_3^- .
4. About 80% of the filtered inorganic phosphate.
5. Variable amount of K^+ , Ca^{2+} , Mg^{2+} & urea.

- b) Secretion of:** Organic solutes as PAH, drugs, various amines and ammonia.

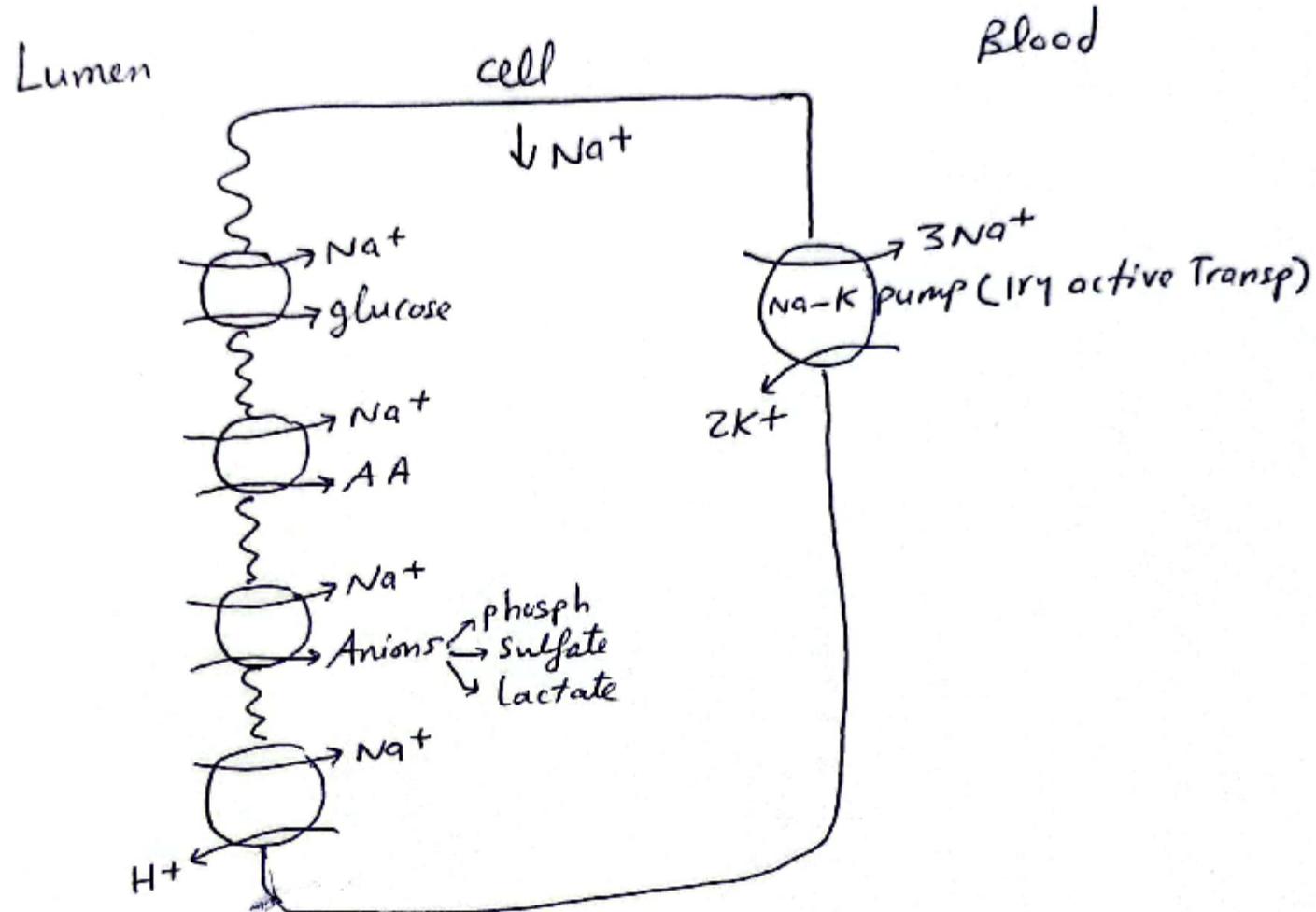
Na⁺ Reabsorption by Proximal Tubules

(I) Early proximal tubule:

- Transcellular transport .
- Water, glucose, HCO₃, amino acids & organic anions as lactate, pyruvate, & phosphate → all are absorbed secondary to Na⁺.
- At the basolateral membrane of PT: Na⁺ is actively pumped out of cell by the electrogenic Na⁺-K⁺ ATPase pump (primary active transport) → ↓Na conc inside cells.
- At the apical border (2ry active transport): Na⁺ is transported through:
 - 1- Na⁺- glucose co-transport or symport
 - 2- Na⁺- amino acid co-transport or symport.
 - 3- Symport with anions other than HCO₃⁻ as phosphate, sulphate, lactate.
 - 4- Na⁺- H⁺ antiport.

Na⁺ Reabsorption by PT

(I) Early PT [Transcellular Transport]



(II) Late proximal tubule:

- Paracellular transport .
- It includes:

1- Cl⁻ - derived Na⁺ reabsorption:

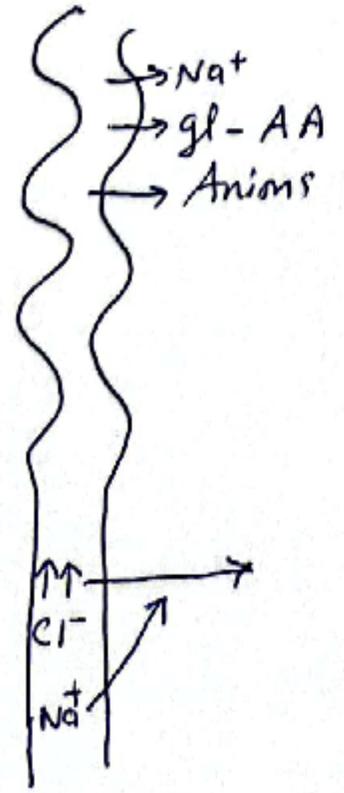
- The preferential reabsorption of HCO₃⁻, solutes and anions in early PT than Cl⁻ → ↑ Cl⁻ concentration at late PT.
- This facilitates passive diffusion of Cl⁻ & the Na⁺ ions follow it.

2- Solvent drag:

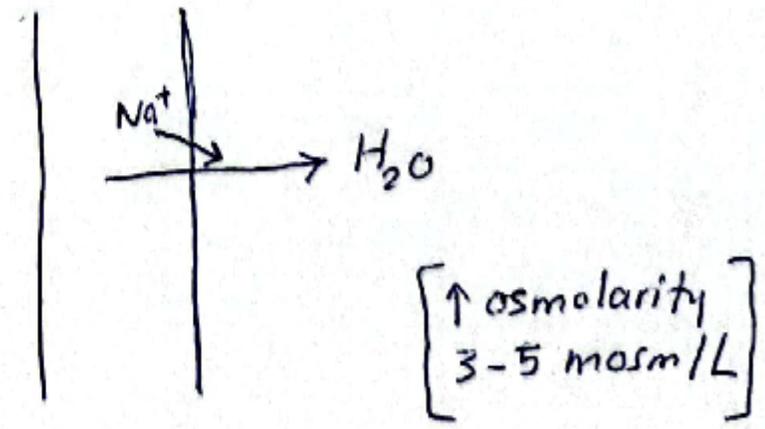
The slight ↑ in peritubular osmolarity (by 3-5 mosmol/L) in late PT causes dragging of water from lumen to paracellular space taking with it NaCl.

(II) Late PT [Paracellular Transport]

1- Cl⁻-derived Na⁺ reabsorption:



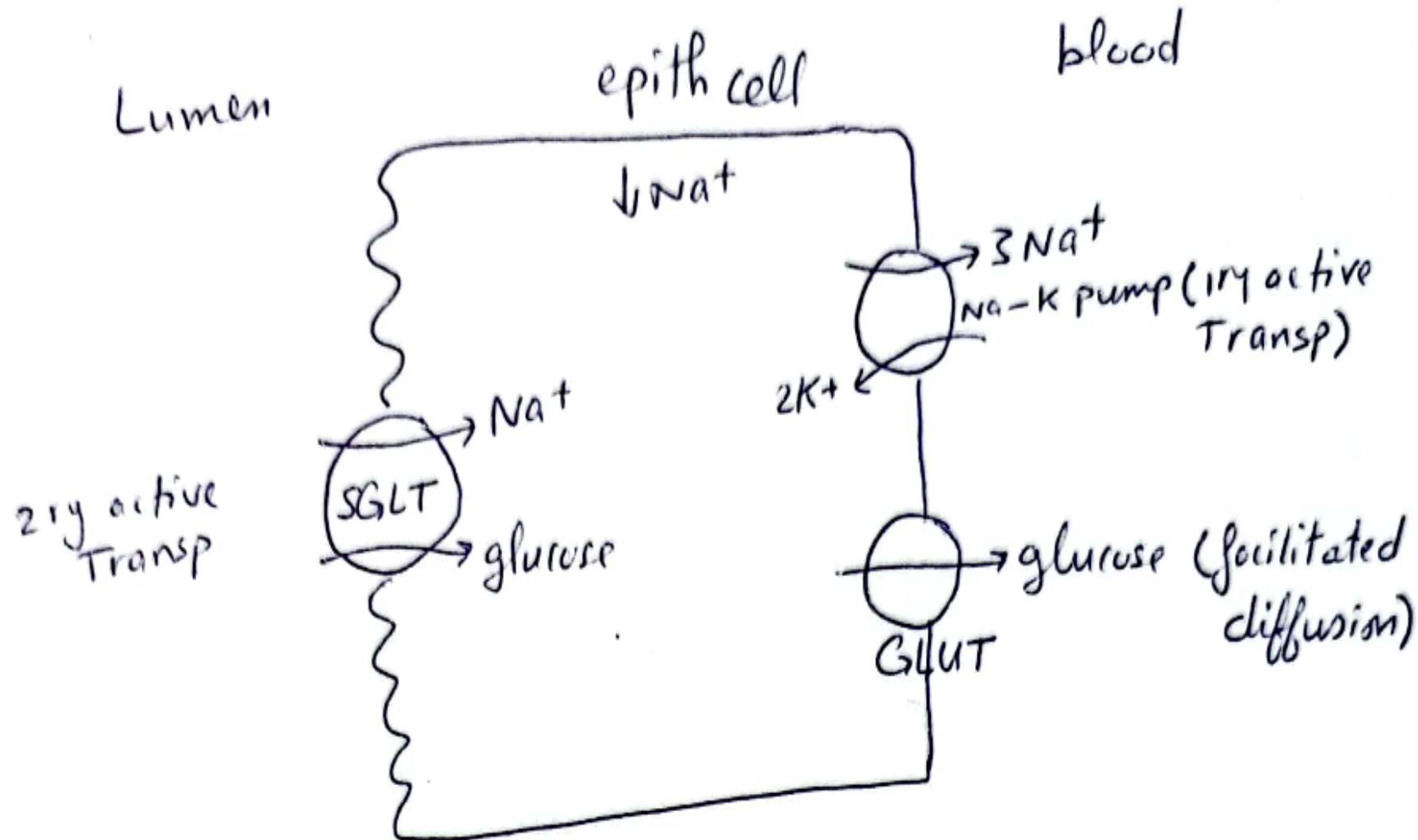
2- Solvent drag:



Glucose Reabsorption by PT

- At the basolateral border, Na^+ is actively pumped outside the cell by $\text{Na}^+ - \text{K}^+$ pump $\rightarrow \downarrow$ Na^+ concentration inside the cells \rightarrow activation of symport (**Na^+ - glucose co-transporter**) (**SGLT**), that transport **Na^+ , & glucose** at apical border (*2ry active transport*).
- Glucose diffuses to paracellular space by GLUT (glucose transporter) (*facilitated diffusion*).

Glucose Reabsorption by P.T



Loop of Henle (LH)



- Overall Functions of LH

1. Reabsorption of 15% of filtered load of water.
2. Reabsorption of 25% of the filtered load of Na^+ .
3. Secretion of urea.
4. Dilution of fluid delivered to DT (to 150-200 mOsm/L).
5. Has an important role in counter-current multiplier system.

Distal Segment of the Nephron



- **It includes:**

- a) Distal convoluted tubule (early distal tubule)
- b) Connecting tubules (late distal tubule)
- c) Collecting ducts

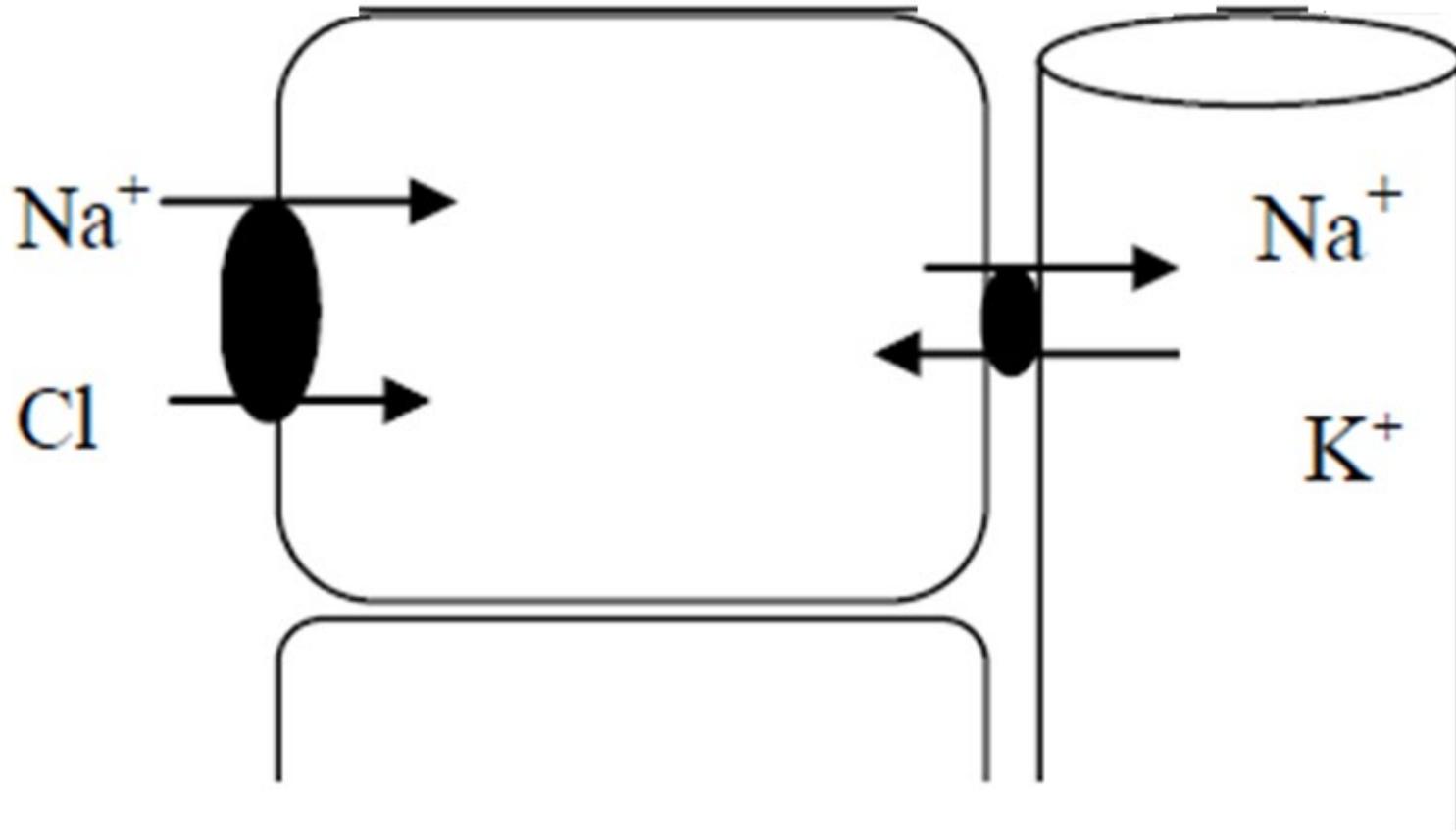
- **Overall functions of the distal segment of nephron:**

- 1) **Final adjustment of urine** formation.
- 2) **Reabsorption of:**
 - a) 7% of filtered load of Na^+ .
 - b) 17% of filtered load of water
- 3) **Secretion of** variable amount of H^+ & K^+ .
- 4) **Major control site** for Na^+ , K^+ , Ca^{2+} & acid-base balance of body.



(A) Distal Convoluted Tubules (DCT)

- At the basolateral border, Na^+ is actively pumped outside the cell by Na^+-K^+ pump $\rightarrow \downarrow \text{Na}^+$ conc inside the cells \rightarrow activation of symport ($\text{Na}^+ - \text{Cl}^-$ transporter) that transport Na & Cl at apical border.



Na reabsorption in distal convoluted tubule

b) Connecting Tubule

- It is the **late 1/3** of distal tubules.
- **Cell lining:** principal and intercalated cells.

c) Collecting Ducts (CD)

- **Include:** cortical (CCD), outer medullary (MCD) & inner medullary (papillary) (PCD).
- **Cell lining:**
 1. Principal cells.
 2. Intercalated cells.

Functions of principal cells

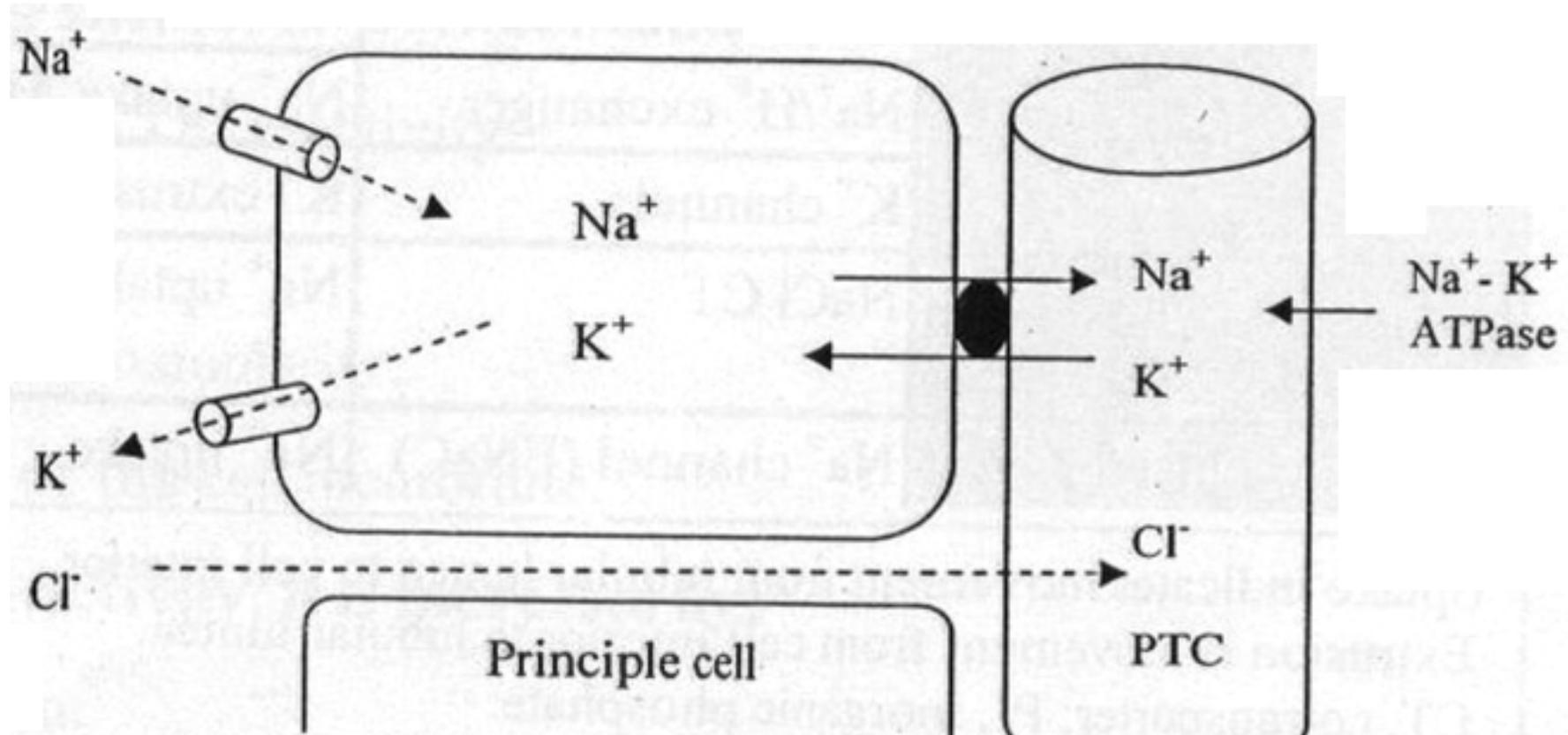
1. Reabsorbs $[\text{Na}^+]$ (3%) & Secrete K^+ under the influence of aldosterone hormone.

2. Reabsorb water (under control of ADH).

• Mechanism of Na^+ reabsorption & K^+ secretion :

□ At the basolateral border, Na^+ is actively pumped outside the cell by $\text{Na}^+ - \text{K}^+$ pump $\rightarrow \downarrow$ Na^+ conc inside the cells $\rightarrow [\text{Na}^+]$ diffuses passively from tubular fluid into the principal cell via apical epithelial $[\text{Na}^+]$ channel.

□ K^+ enters the cell by the basolateral $\text{Na}^+ - \text{K}^+$ ATPase \rightarrow high intracellular K^+ \rightarrow secreted via apical K^+ channels to tubular fluid.



Na reabsorption & K secretion by principal cells

MCQ

What is the primary function of the proximal tubule?

- a) Filtration of plasma
- b) Concentration of urine
- c) Reabsorption of most of the filtered nutrients
- d) Secretion of renin
- e) Secretion of vasopressin

Answer: c



Renal handling of electrolytes

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Renal handling of Na



- About 99 % of the filtered load of Na is reabsorbed:
 - 67 % from PCT.
 - 25 % from ascending limb of Loop of Henle.
 - 4 % from DCT.
 - 3 % from principal cells of CD.
- About 1% of the filtered load of Na is excreted in urine.

Factors affecting renal Na^+ Excretion



1) Angiotensin II (All): ↓ Na^+ excretion by:

- ↓ GFR: by *VC of both aff & eff arterioles.*
- ↑ Na^+ reabsorption from PT (direct effect) & CD (indirect effect through stimulating aldosterone secretion).

2) Renal sympathetic nerve: ↓ Na^+ excretion by:

- ↓ GFR: by VC of renal bVs.
- ↑ renin release.

3) Aldosterone: ↑ Na^+ reabsorption by increasing synthesis of Na^+ channels into the apical membrane of CD principal cells.



4) Atrial natriuretic peptide: (ANP)

- **Source:** wall of right atrium.
- **Stimulus:** stretch of atrial wall due to ↑ plasma volume.
- **Effect:** ↑ Na^+ excretion rate through:
 1. ↑ **GFR:** by *VD of aff arteriole*.
 2. Inhibition of Na^+ reabsorption from **CD** by inhibiting the action of aldosterone.
 3. Inhibition of renin release.

5) Antidiuretic hormone (ADH):

- It stimulates Na^+ reabsorption from thick AHL

Renal handling of K⁺

- The fraction of K⁺ reabsorption from PT and thick ALH is constant:

* 67% in PT.

* 20% in thick ALH.

- Further handling of K⁺ in the following tubular segments depends on K⁺ intake as follows:

	Distal tubules	Collecting ducts
K ⁺ depletion	3% of filtered load of K ⁺ is reabsorbed	9% of filtered load of K ⁺ is reabsorbed
Normal K ⁺ intake	10% of filtered load of K ⁺ is secreted	5% of filtered load of K ⁺ is secreted
Increase K ⁺ intake	50% of filtered load of K ⁺ is secreted	30% of filtered load of K ⁺ is secreted

• Factors affecting K^+ secretion by the principal cells

1) Plasma K^+

↑ K^+ concentration → ↑ K^+ secretion via:

- Activation of Na^+ - K^+ ATPase
- Stimulation of aldosterone hormones.

2) Tubular flow rate:

↑ tubular flow rate → ↑ K^+ secretion via:

- Delivery of Na^+ to the principal cell.
- Dilution of the secreted K^+

3) Acid - base status:

- Acidosis → ↓ K^+ secretion &
- Alkalosis → ↑ K^+ secretion.

4) Aldosterone hormone:

- Represents the only hormonal control of K^+ output.

- Actions:

a) ↑ activity of $Na^+ - K^+$ ATPase.

b) ↑ synthesis of Na^+ channels → Na^+ reabsorption.

c) ↑ formation of K^+ channels → K^+ secretion.

Renal handling of Ca^{2+}



- 99% of filtered load of Ca^{2+} is reabsorbed by renal tubules:
 - 60-70% at PT.
 - 10-15% at thick ALH.
 - 5-10% at distal tubules.
 - Less than 5% at CD.
- 1% filtered load of Ca^{2+} is excreted.



- **Mechanism:**

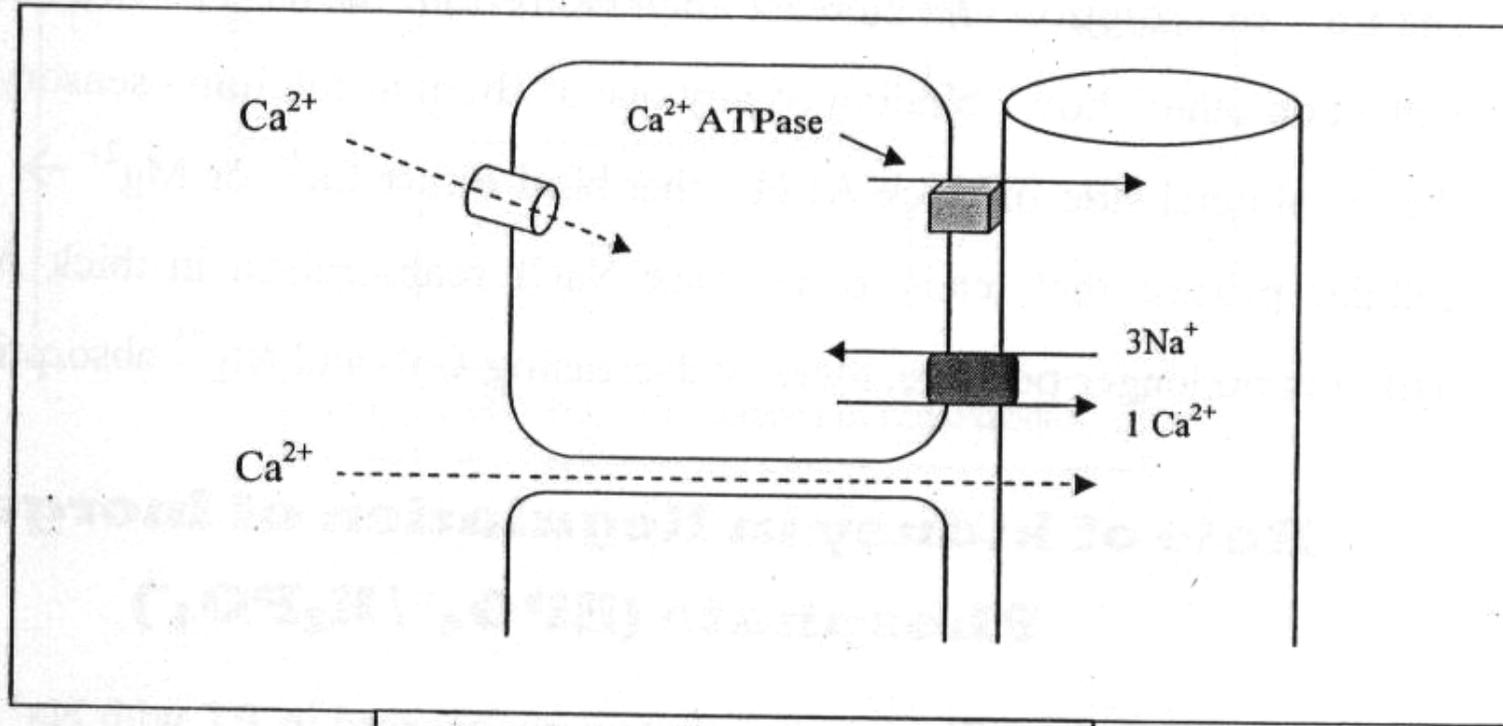
- **(1) In PT:**

- **Transcellular transport:**

- **Account for** $1/3$ amount reabsorbed at PT.
- **At the basolateral border**, Ca^{2+} is actively extruded by **Ca^{2+} -ATPase** or **3Na^{+} - Ca^{2+} anti porter** \rightarrow \downarrow Ca^{2+} conc inside PT cells \rightarrow \uparrow Ca^{2+} entry through special channels at **apical border** by conc. gradient.

- **Paracellular transport:**

- **Account for** $2/3$ amount reabsorbed at PT.
- **Occurs by:**
 - 1- Solvent drag.
 - 2- +ve lumen in 2nd half of PT



Ca reabsorption in proximal tubule



(2) In thick ALH:

- Transcellular transport: as PT.
- Paracellular transport:
 - Only by +ve lumen.
 - No solvent drag as thick ALH is *impermeable to H₂O*.

(3) In DT and CD:

- Transcellular transport: as PT.
- Paracellular transport:
 - *Lumen is -ve*, so Ca²⁺ can not be absorbed passively through *paracellular space*.



• Factors Affecting Ca^{2+} Excretion:

1-Parathyroid hormone: stimulates Ca^{2+} reabsorption.

2- Calcitonin: inhibits Ca^{2+} reabsorption.

3- ECF volume: \uparrow body fluids volumes $\rightarrow \uparrow$
 Ca^{2+} excretion, while its $\downarrow \rightarrow \downarrow$
 Ca^{2+} excretion

MCQ

What percentage of filtered sodium is reabsorbed in the proximal tubule?

- a) 50%
- b) 67%
- c) 75%
- d) 85%
- e) 90%

Answer: b



Water balance

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Renal handling of water

Obligatory reabsorption

- 67 % of the filtered load of water is reabsorbed from proximal tubule (by osmosis 2ry to reabsorption of solutes).
- 15 % of the filtered load of water is reabsorbed from DLH (by osmosis 2ry to high osmolarity of medullary interstitium).
- Not under hormonal control.

Facultative reabsorption

- 17 % of the filtered load of water is reabsorbed by the collecting duct, under the effect of ADH.
 - 11% from cortical CD.
 - 6 % from outer medullary CD.
- Only 1ml of urine is formed /min → urine flow rate.

Regulation of water balance



Role of kidney (water output)

- The kidney can make diluted urine up to 25-50 mosmol/L or concentrated urine up to 1200-1400 mosmol/L.

Role of thirst (water input)

- Stimuli for thirst:
 - 1- Hyperosmolarity: 2-3% \uparrow in plasma osmolarity.
 - 2- Blood volume: 10-15% \downarrow in blood volume.
 - 3- Angiotensin II: by direct action on thirst center.
 - 4- Dryness of the mouth.

- **Requirement for the kidney to make diluted or concentrated urine:**

1) Formation of medullary gradient.

2) Maintenance of this medullary gradient.

3) Role of ADH.

A) Formation of Medullary Gradient

- Def of medullary gradient: It is gradually increasing medullary osmolarity from **300 mosmol/L** at the **cortico-medullary junction** up to **1200-1400 mosmol/L** at the tip of **renal papillae**.
- Causes of medullary gradient:
 - 1) Counter-current multiplier system.
 - 2) Urea recycling.

(1) Counter-Current Multiplier System

- **Def:** It is the system in which the inflow runs *parallel to, close to* and in *counter direction* to the outflow.

- **Mechanism:**

1- Active NaCl reabsorption at thick ALH (single effect).

2- Different water & solute permeability of loop of Henle.

3- Counter-current flow in the loop of Henle

4- Role of distal tubule and CCD

5- Osmotic equilibrating device of medullary CD

B) Maintenance of Medullary Gradient

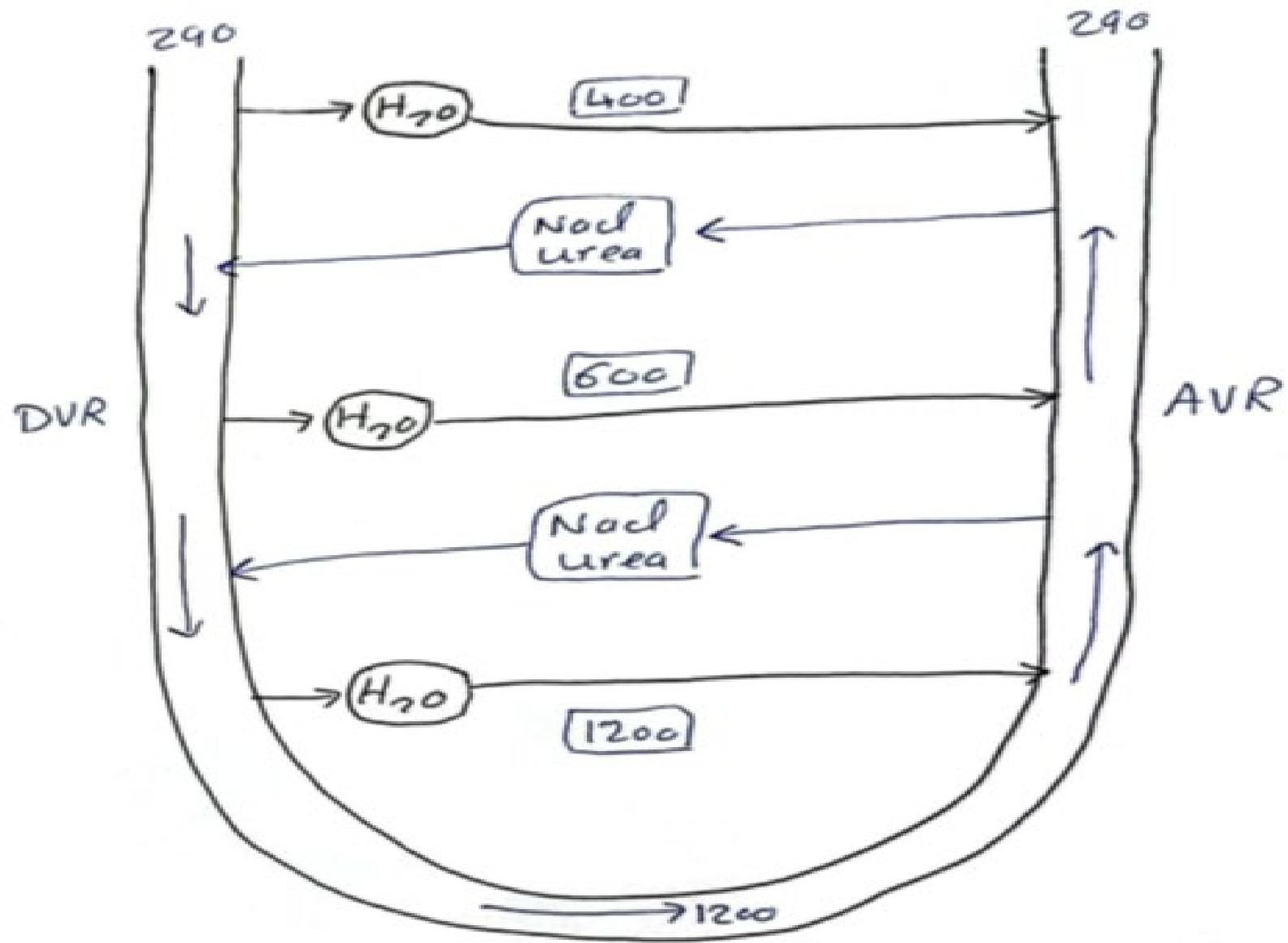


'Role of Vasa Recta'

- **Means** the preservation of the medullary osmolarity constant by keeping the concentration of solutes constant in the medulla.
- **Maintenance of the medullary gradient** is produced by the vasa recta which is formed of descending (DVR) & ascending (AVR) limbs.
- **DVR** removes excess solutes & **AVR** removes excess water.

□ Vasa recta (VR) is characterized by:

- Counter-current exchanger system.
 - Capillary wall is permeable to solutes & water. So, solutes enter DVR & water leaves it while in AVR, solutes leave & water enters it.
 - Long capillaries
 - High viscosity of blood
- } slow blood flow in vasa recta
- To keep the medullary constant, solutes input must equal the solutes output & water input must equal the water output.



Vasa recta

Vasa Recta

C) Role of ADH:

- 1) Stimulation of co-transport of Na^+ , K^+ & Cl^- at thick ALH \rightarrow \uparrow amount of solute reabsorption.
- 2) It \downarrow medullary blood flow, so it helps maintenance of medullary gradient, not washed out by high blood flow.

3) It ↑ water permeability of connecting tubule, CCD & medullary CD → ↑ urea concentration
→ ↑ urea reabsorption from inner medullary CD.

4) It ↑ urea permeability in inner medullary CD
→ so, it will diffuse to the medulla increasing its osmolarity. Urea re-circulate between LH & CD → entrapping of urea in the medulla → share by 50% of medullary osmolarity (600 mosmol NaCl and 600 mosmol urea).

MCQ

What percentage of water is reabsorbed in the proximal tubule?

- a) 55%
- b) 67%
- c) 47%
- d) 85%
- e) 50%

Answer: b



Role of kidney in acid-base balance

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Role of kidney in acid-base balance



- The Kidney plays an important role in keeping the constancy of $[H^+]$ concentration through two main mechanisms:
 - I) Reabsorption of the filtered HCO_3^- .
 - II) Excretion of fixed acids and formation of new HCO_3^- .

(I) Reabsorption of filtered HCO_3^- :

1- In proximal tubules:

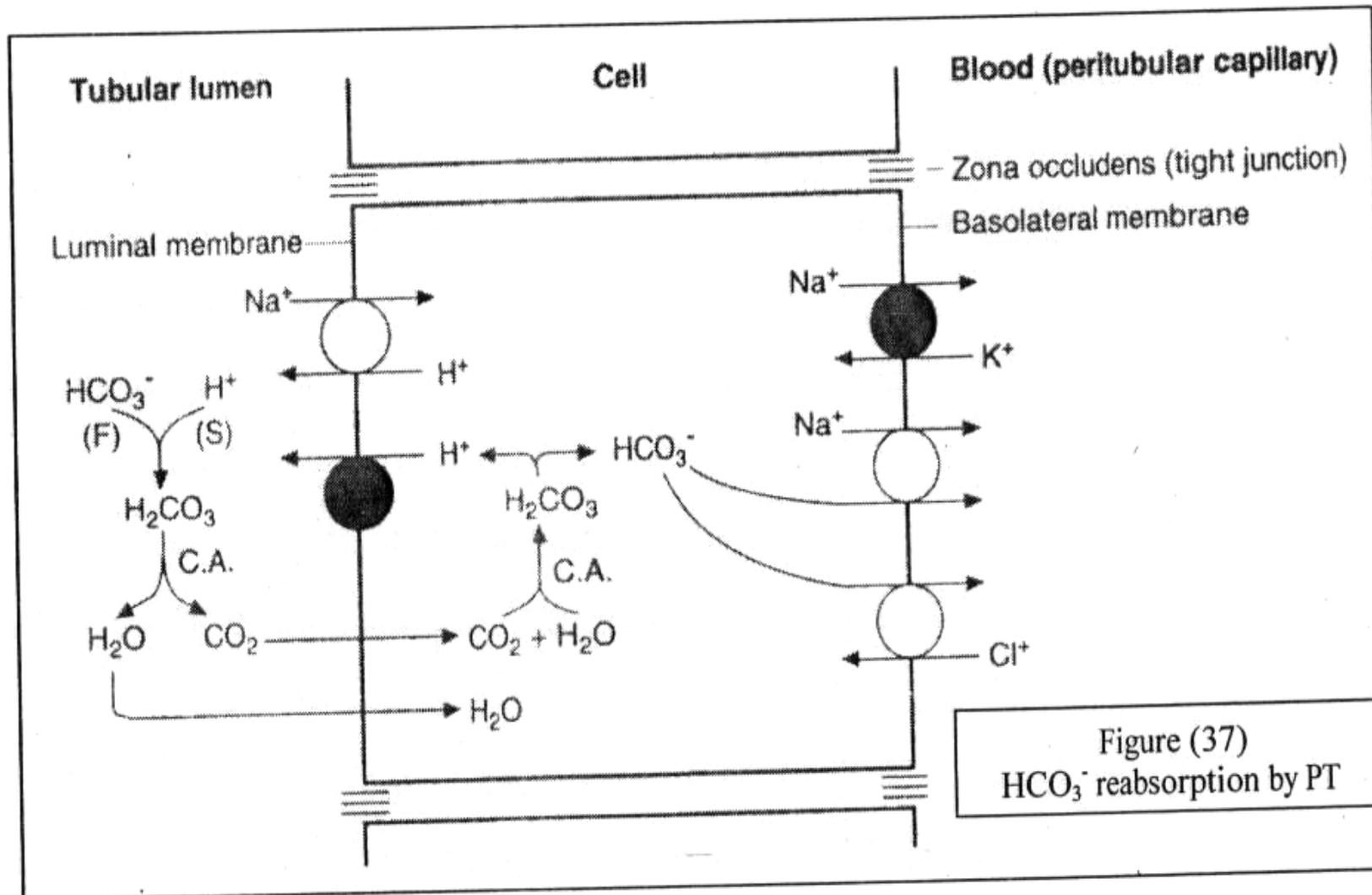
- *Fraction reabsorbed:* about **85%** of filtered load of HCO_3^- .

- *Mechanism:*

□ Carbonic anhydrase enzyme (**CAE**) facilitates reaction of H_2O & CO_2 to form H_2CO_3 which is **dissociated** into H^+ & HCO_3^- . H^+ is **secreted** in **exchange** with Na^+ or by **H^+ ATPase**.

□ H^+ (secreted by the **PT**) reacts with **filtered HCO_3^-** forming H_2CO_3 which is dissociated into **CO_2 & H_2O** by the **brush border CAE**.

- ❑ The intracellular HCO_3^- is reabsorbed from the basolateral border into the peritubular capillaries with Na^+ or in exchange with Cl^- .
- ❑ Thus, for every HCO_3^- molecule loss from the filtrate, there is gain of one molecule of HCO_3^- , so the absorption occurs by indirect mechanism.



HCO₃ reabsorption in proximal tubule

2- In thick ALH:

- **Fraction reabsorbed:** about 10% of filtered load of HCO_3^- .
- **Mechanism:** similar to that in PT.

3- In DT and CD:

- **Fraction reabsorbed:** the remaining fraction (less than 5% of filtered load of HCO_3^-).
- **Mechanism:** similar to that in PT.



(II) Excretion of Fixed Acids & Formation of New HCO_3^-

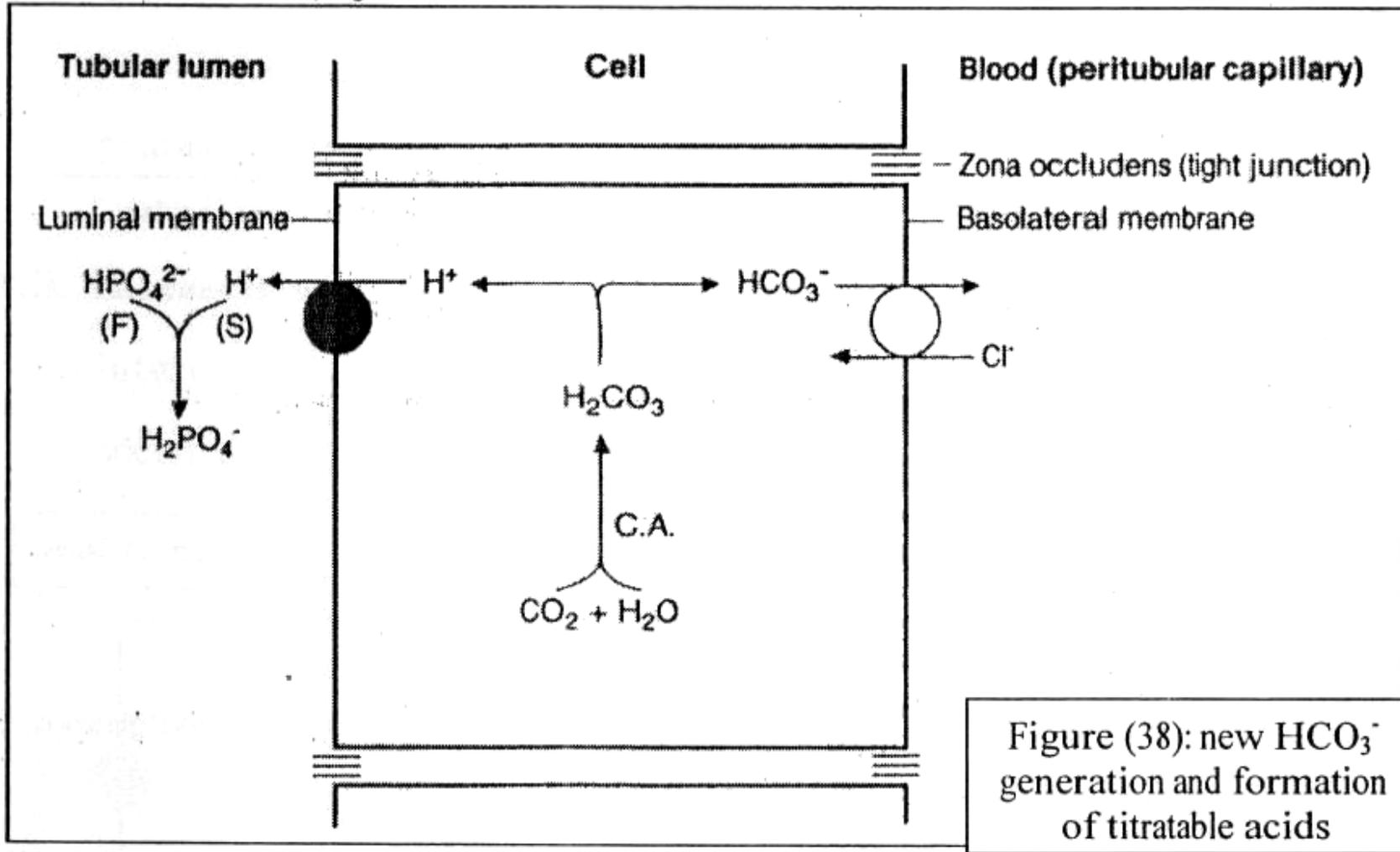
- The mechanisms of new HCO_3^- formation and H^+ secretion includes:

- 1- Secretion of H^+ in the form of titrable acids.
- 2- Secretion of H^+ in the form of NH_4Cl .

1) Formation new HCO_3^- and excretion of titratable acidity:

- **Def:** Titratable acidity is the portion of H^+ bound to filtered & excreted buffers.
- **The filtered buffers** are mainly phosphate & to less extent creatinine, urate & β -hydroxy butyrate.

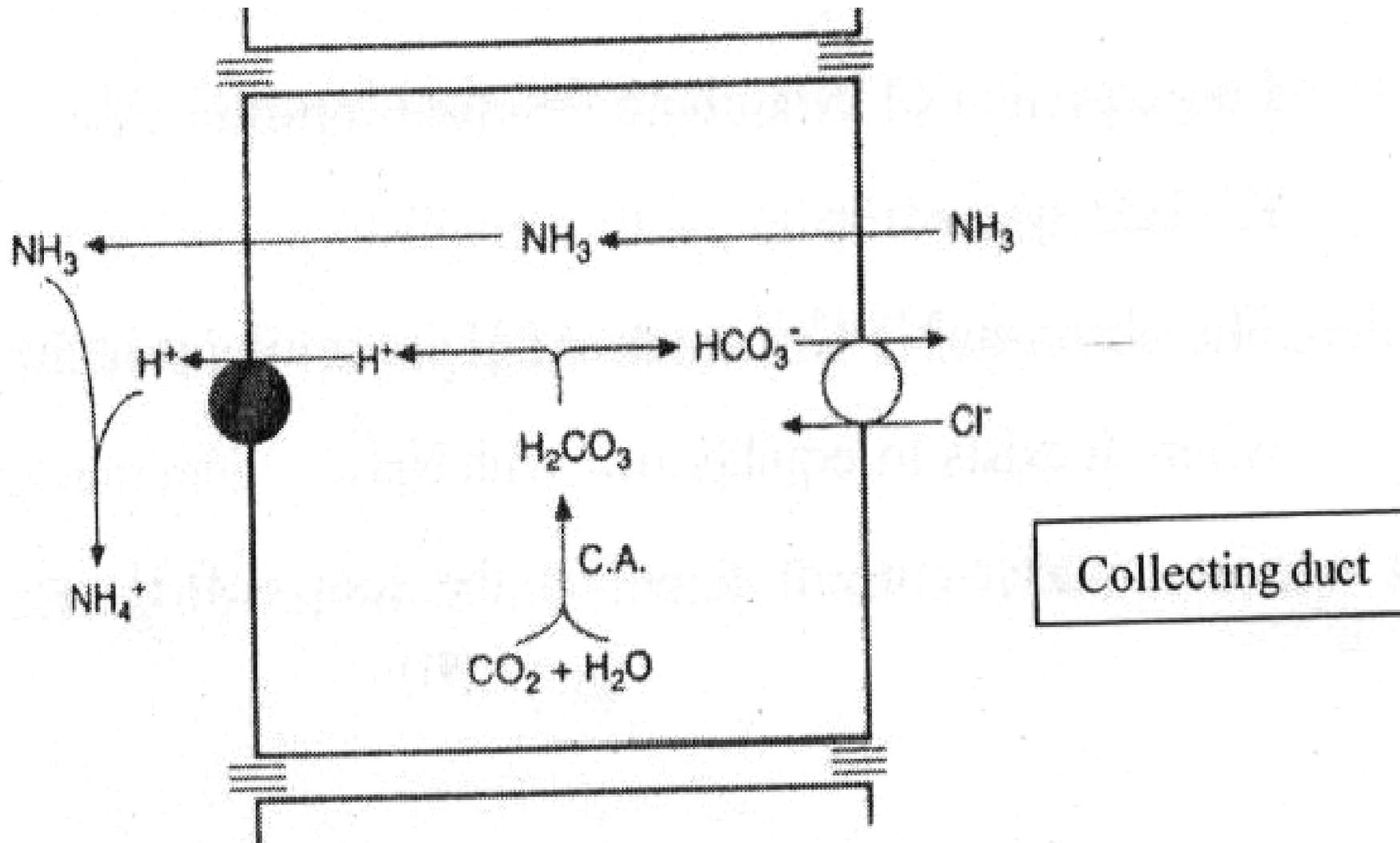
- **In the collecting ducts**, the secreted H^+ (by H^+ -ATPase) in ***α -intercalated cells*** combine with Na_2HPO_4 (di-basic phosphate) forming NaH_2PO_4 (monobasic phosphate).



Formation new HCO_3^- and excretion of titratable acidity

2) Formation of new HCO_3^- & excretion of ammonia:

- Ammonia is the most important urinary buffer; it is formed in the kidney by metabolism of glutamine.
- NH_3 diffuses from medullary interstitium to the lumen of the collecting ducts to buffer the secreted H^+ forming NH_4^+ which is excreted in the form of NH_4Cl .
- Thus, for each one molecule formed of NH_4^+ , there is again of one molecule of HCO_3^- .



Formation of new HCO_3^- and excretion of ammonia

• Factors affecting renal secretion & excretion of H^+ :

1) Acidosis: especially intracellular acidosis $\rightarrow \uparrow H^+$ secretion.

2) Arterial PCO_2 :

\uparrow arterial $PCO_2 \rightarrow \uparrow$ formation of intracellular H_2CO_3 which is dissociated into H^+ & HCO_3^- . H^+ is secreted by tubules.

3) Carbonic anhydrase enzyme (CAE):

→ ↑ secretion of H^+ .

4) Plasma K^+ :

➤ **Hyperkalemia** → acidosis (↓ H^+ secretion)

➤ **Hypokalemia** → alkalosis (↑ H^+ secretion)

5) Aldosterone: stimulates H^+ secretion.

MCQ

What percentage of filtered bicarbonate (HCO_3^-) is reabsorbed in the proximal tubule?

- a) 5%
- b) 50%
- c) 85%
- d) 90%
- e) 100%

Answer: c



Micturition

By

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Micturition

- Innervation of urinary bladder:

	1-Parasympathetic	2-Sympathetic
Via	Pelvic nerve (S2, 3, 4)	Hypogastric nerves (LHCs of L1, 2, 3)
Actions	Contraction of UB wall & relaxation of internal urethral sphincter → micturition	Relaxation of UB wall & contraction of internal urethral sphincter → urine retention

3- Pudendal nerve (somatic nerve): (AHCs of S2,3 and 4):
 Innervate external urethral sphincter (skeletal voluntary muscle).

- **Micturition reflex:**

- **Receptors:** stretch receptor in the wall of bladder.
- **Afferent :** pelvic nerve.
- **Center:** sacral segments 2, 3 & 4.
- **Efferent and response:**
 - **Pelvic nerve** → Contraction of UB wall & relaxation of internal urethral sphincter
 - **Pudendal nerve** → relaxation of external urethral sphincter.



- **Supraspinal centers affecting micturition:**

1) **Cerebral cortex:** Motor cortex exerts a voluntary control of micturition either **stimulation or inhibition.**

2) **Hypothalamus:** There is **facilitatory** area in the hypothalamus.

3) **Midbrain:** Inhibition.

4) **Pons:** facilitation

• Role of higher centers:

- Keeping the micturition reflex partially inhibited all the time except when there is a desire for micturition.
- Prevent micturition even when the reflex is initiated until appropriate time.

MCQ

Which spinal cord segments form the spinal micturition center?

a) T11-L2

b) S1-S5

c) S2-S4

d) L4-L5

e) T1-T12

Answer: c