RAAS & Blood Pressure

By Sebastian Wolinski

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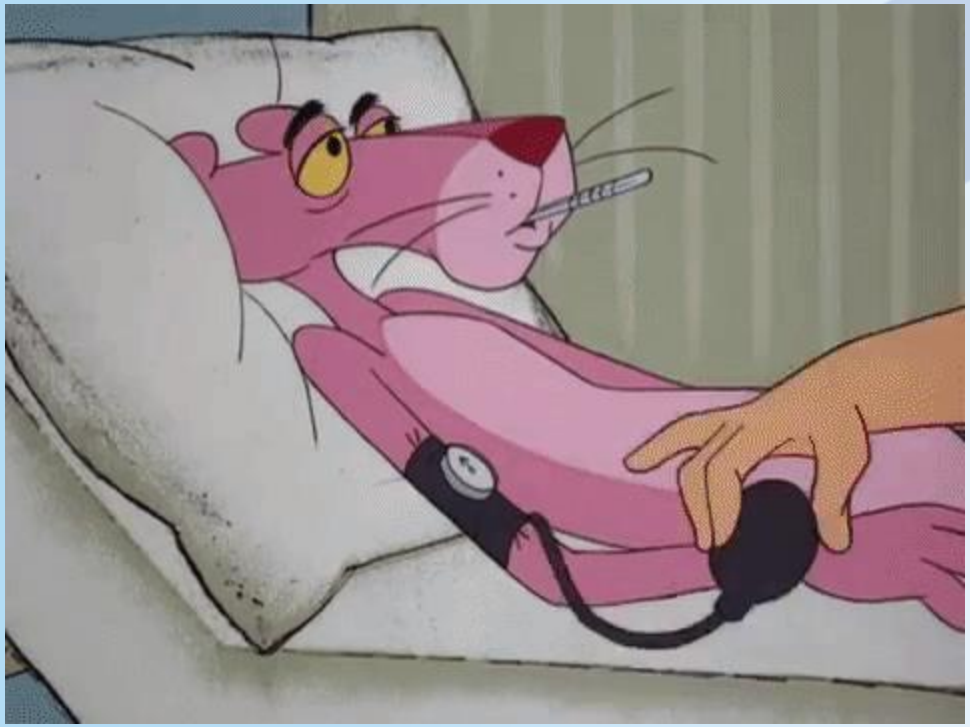
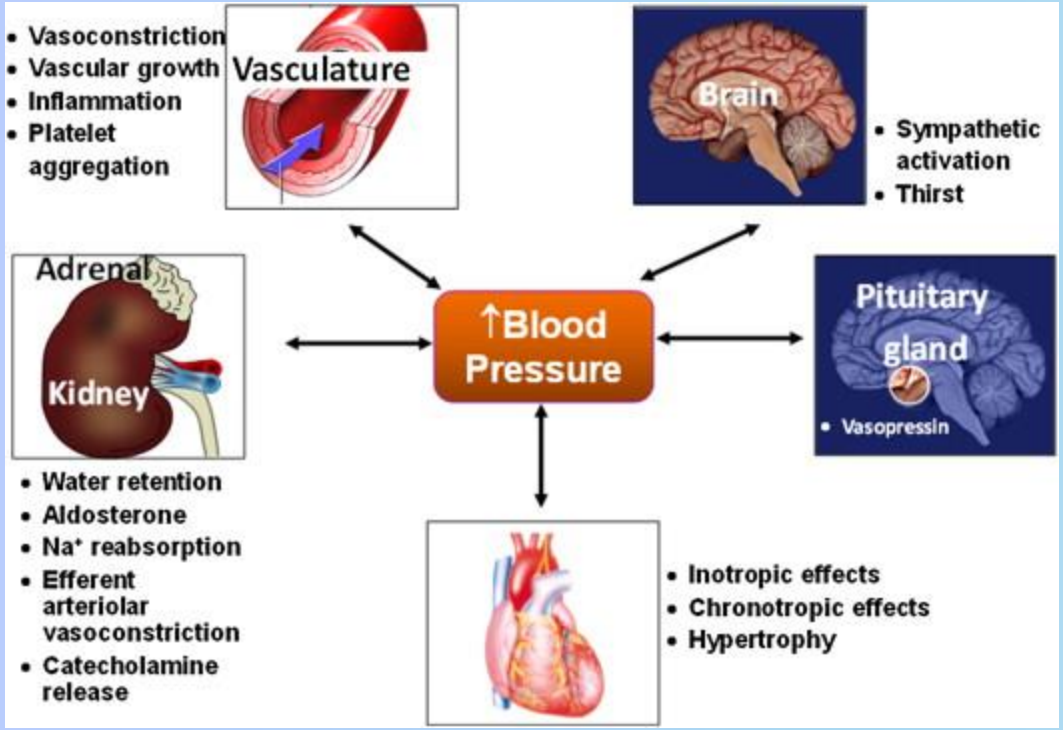


Big Picture



- Blood pressure = Heart + Vessels + Kidneys
- Short-term BP = nerves + heart
- Long-term BP = kidneys + volume
- RAAS = bridge between kidney function and BP

"The heart is emotional. The kidney never forgets."

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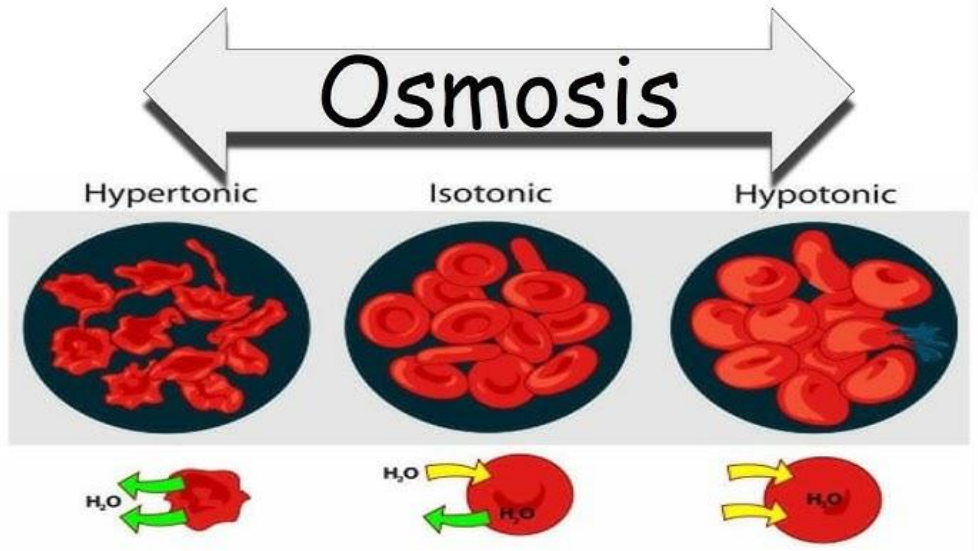
Osmolarity



- Osmolarity = number of solute particles per liter of solution

- Normal plasma osmolarity ≈ **300 mOsm/L**
- Dominated by:
  - **Na^+** (most important)
  - Glucose
  - Urea (minor effect)
- 🧠 **"Water ALWAYS follows salt (Na^+)"**

- 📌 **Exam trap:**
  - Osmolarity ≠ volume
    - You can be **dry but concentrated**

What changes Osmolarity?

- ↑ **Osmolarity**
- Dehydration
- Diarrhea
- Sweating
- Diabetes mellitus (glucose!)

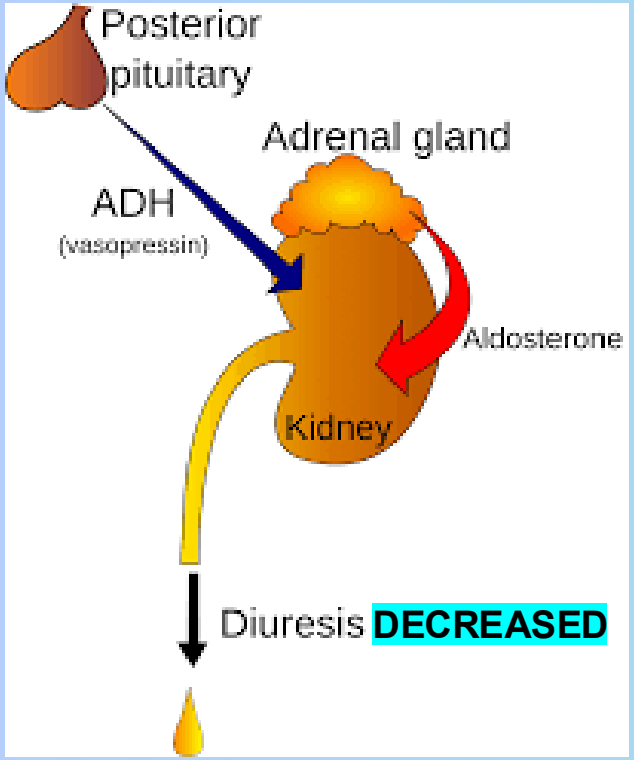
- ↓ **Osmolarity**
- Excess water intake
- SIADH

🧪 **Classic exam question:**

- High plasma osmolarity → which hormone released?

✓ **ADH**

ADH: The water-saver



- **Anti-Diuretic Hormone (vasopressin):**
- Released from **posterior pituitary**
- Triggered by:
  - ↑ Osmolarity (MOST IMPORTANT)
  - ↓ Blood volume

- **Effect:**
- Collecting duct → **aquaporins inserted**
- ↑ Water reabsorption
- ↓ Urine volume

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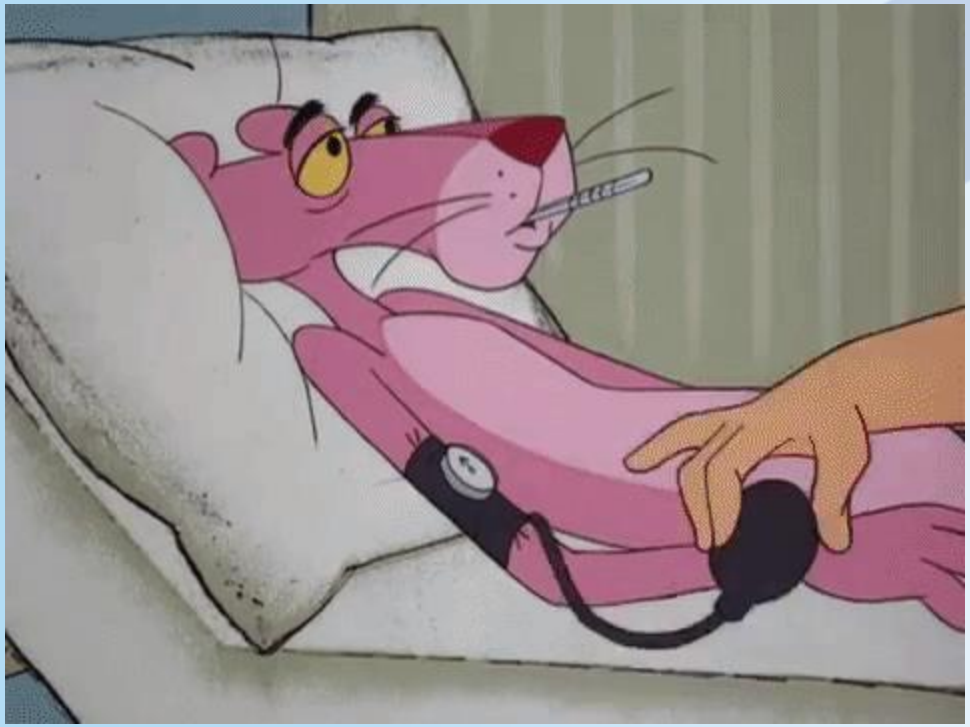
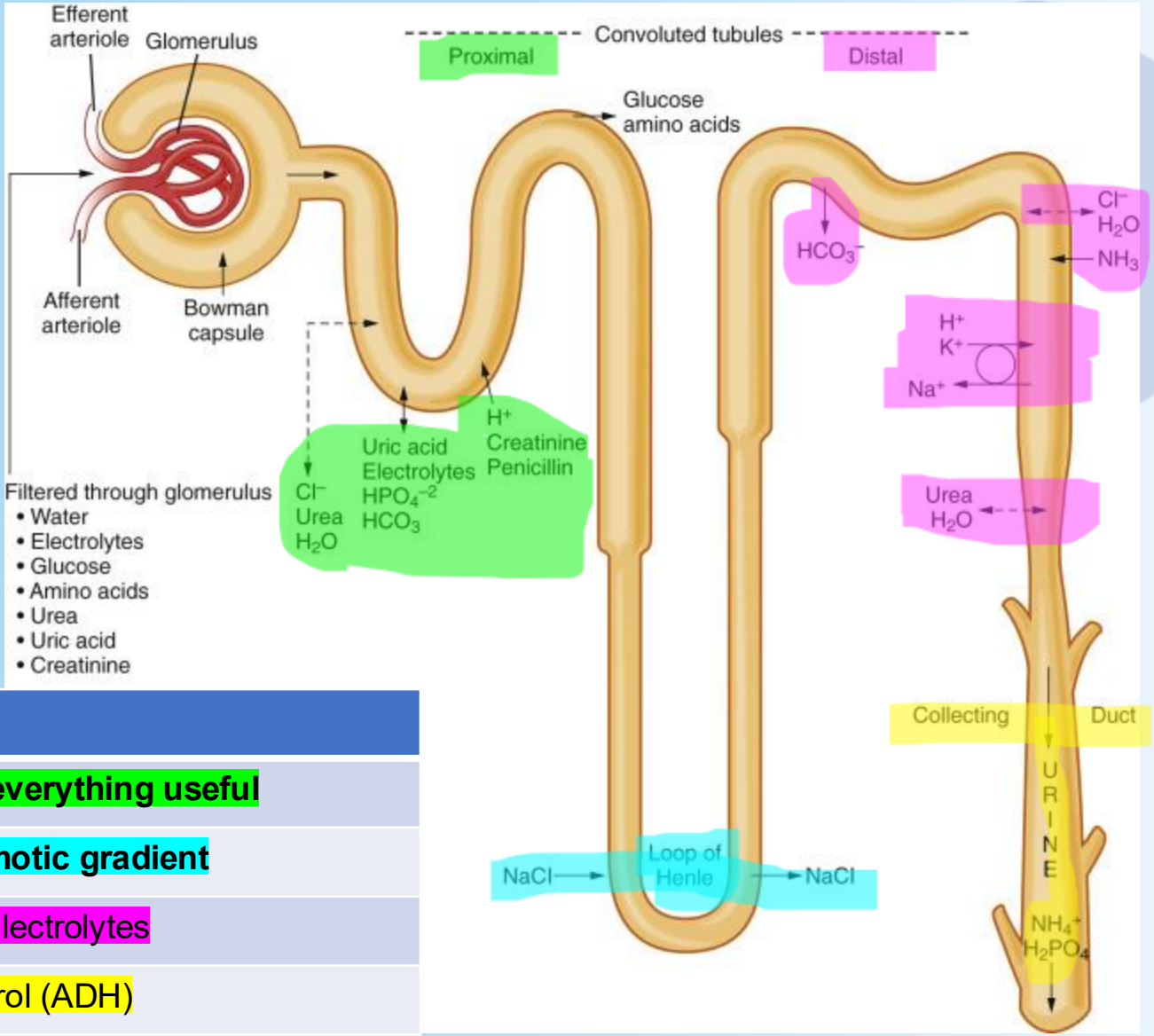
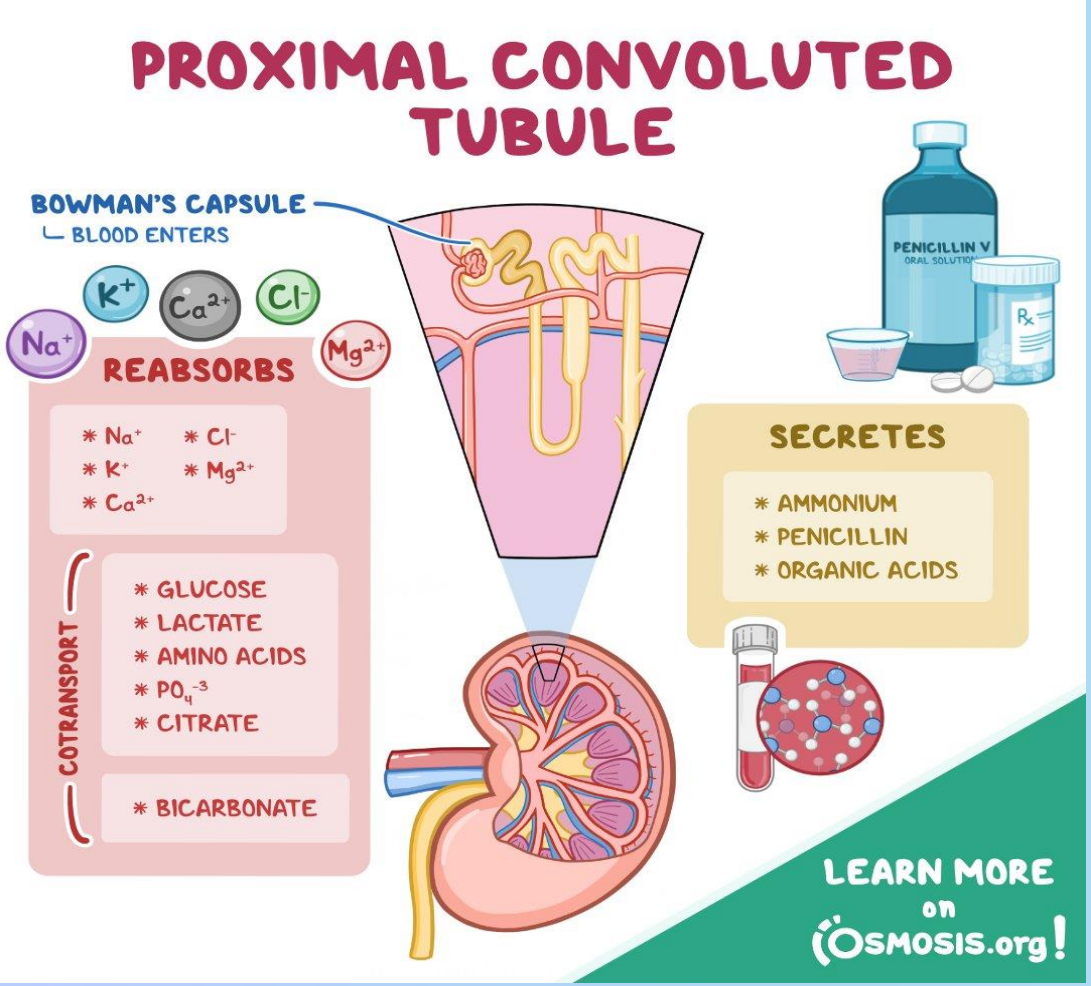


Nephron Overview



Segment	Main Job
PCT	Reabsorb **everything useful**
Loop of Henle	Create **osmotic gradient**
DCT	Fine-tune electrolytes
Collecting duct	Water control (ADH)

Ion Movement: PCT



- **Proximal Convoluted Tubule (PCT)**
- Reabsorbs:
  - **Na^+**
  - **Glucose**
  - **Amino acids**
  - **HCO_3^-**
- Water follows Na^+

- **"PCT = Pretty Close to Total reabsorption"**

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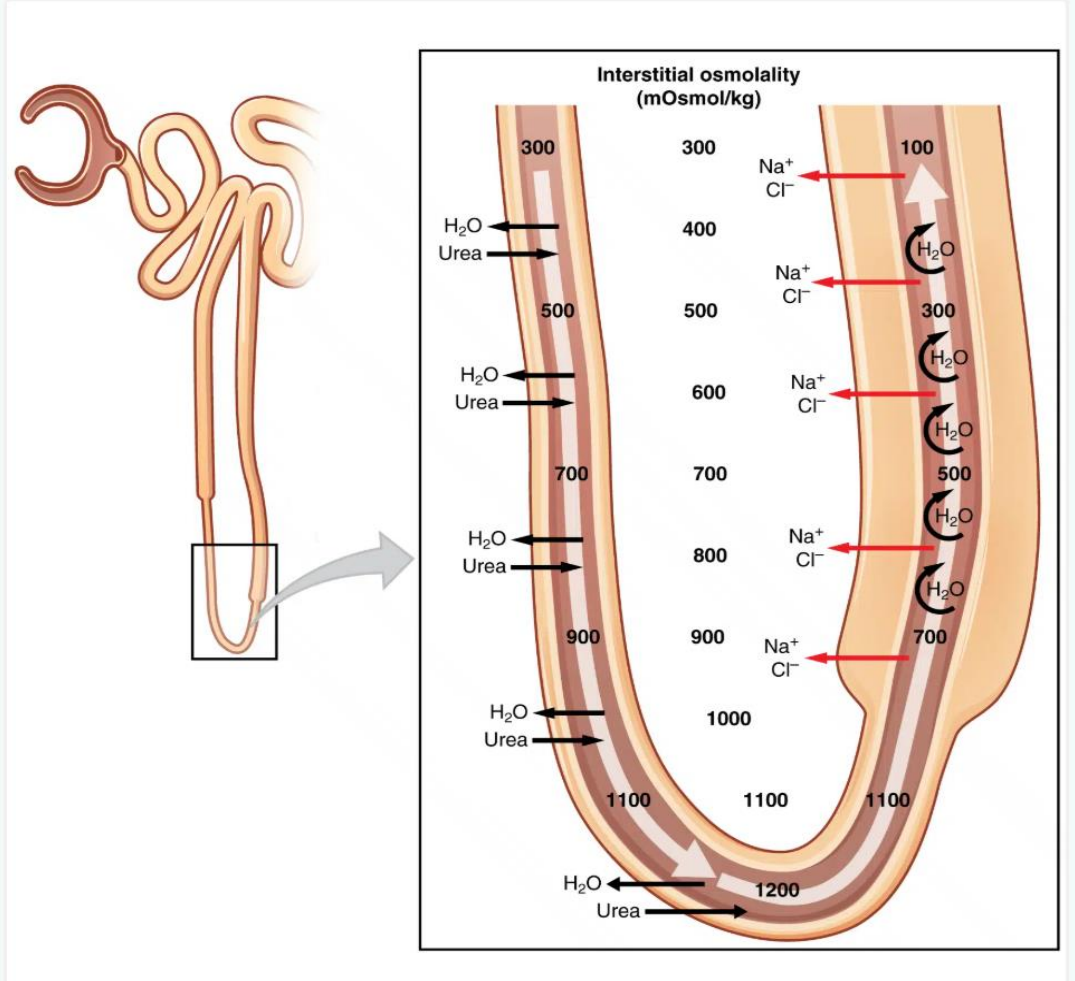
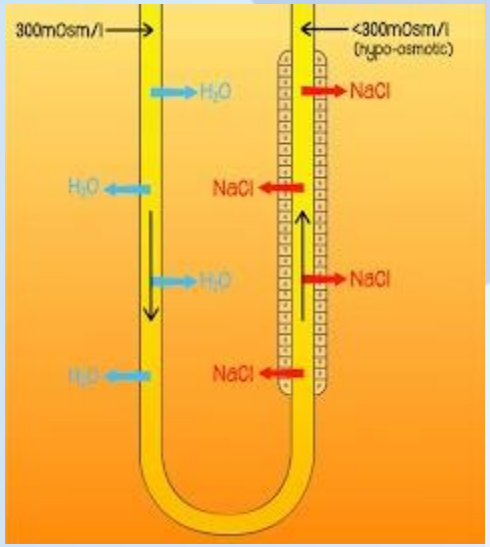
Loop of Henle: Dilute vs Concentrate





- **Descending limb**
  - Water OUT
  - No Na^+ movement
- **Ascending limb**
  - Na^+, K^+, Cl^- OUT
  - No water
- 🧠 **Mnemonic:**
  “Down = water leaves, Up = salt leaves”

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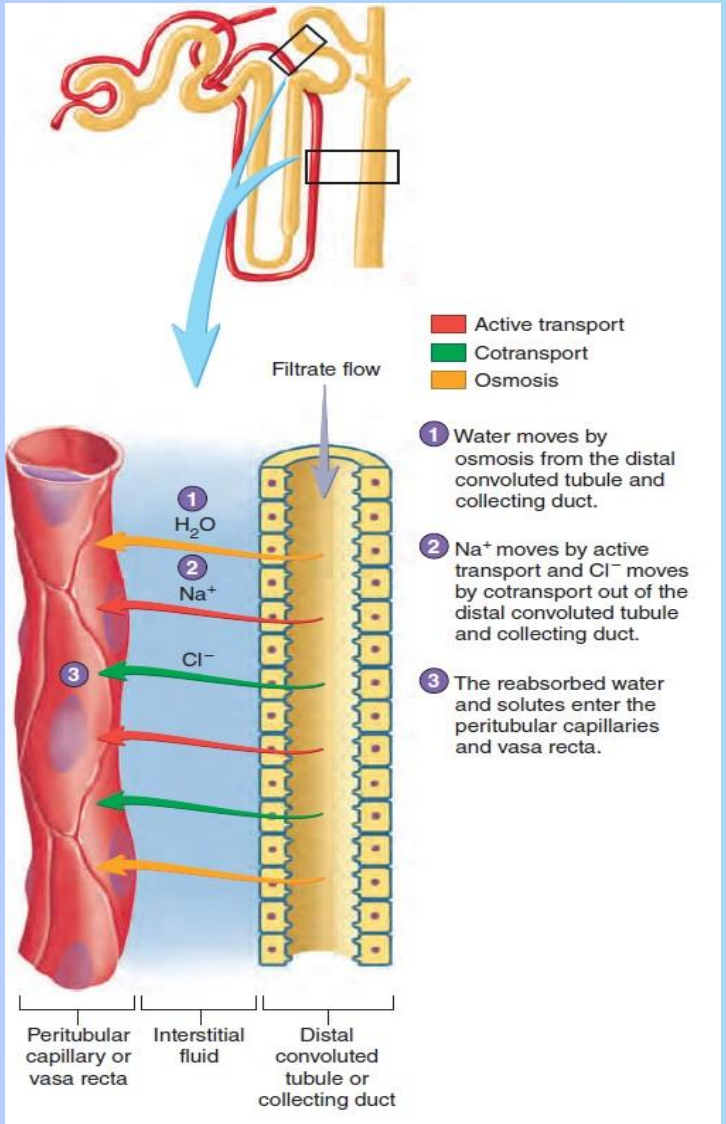
Distal Convoluted Tubule & Collecting Duct



PROCESS Figure 18.13 AP R Reabsorption in the Distal Convoluted Tubule and Collecting Duct

Approximately 19% of the filtrate is reabsorbed from the distal convoluted tubule and collecting duct.

- **DCT**
  - Na^+ reabsorption (aldosterone sensitive)
  - Ca^{2+} reabsorption (PTH)
- **Collecting duct**
  - ADH dependent water reabsorption
  - Final urine concentration
- 📌 **Exam pearl:**
- Urine entering collecting duct is **hypotonic**

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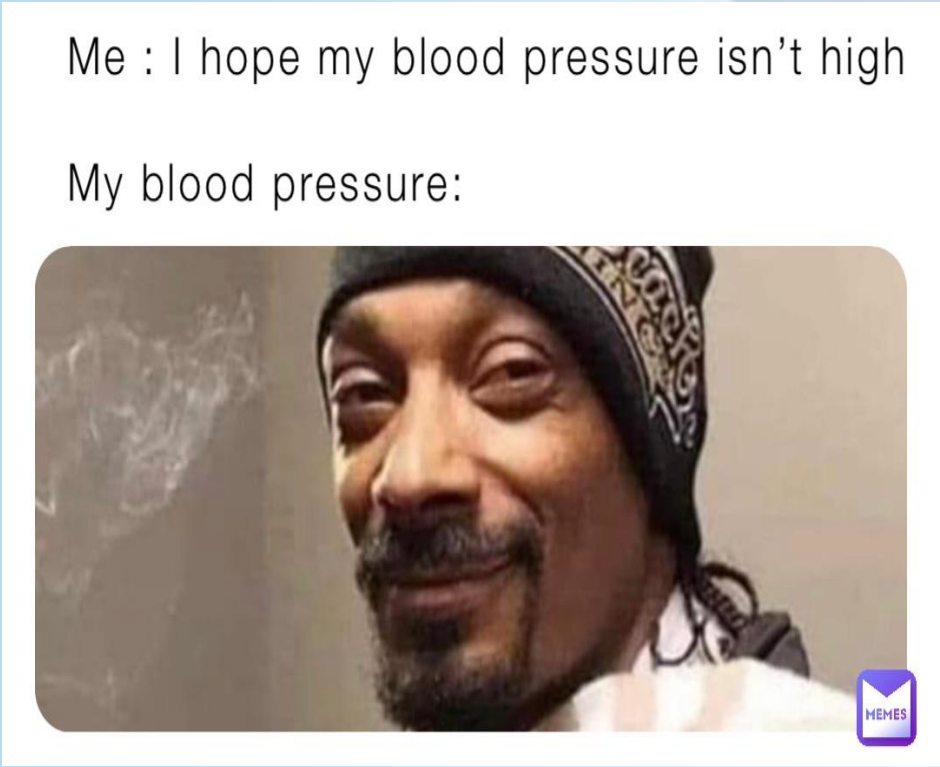
Segway: Why This Matters for BP

- **BP depends on volume**
- Volume depends on **Na^+** concentration
- Na^+ reabsorption depends on **kidney function**
- Kidney controlled by **RAAS**
- 🧠 “Salt pulls water → water raises BP”



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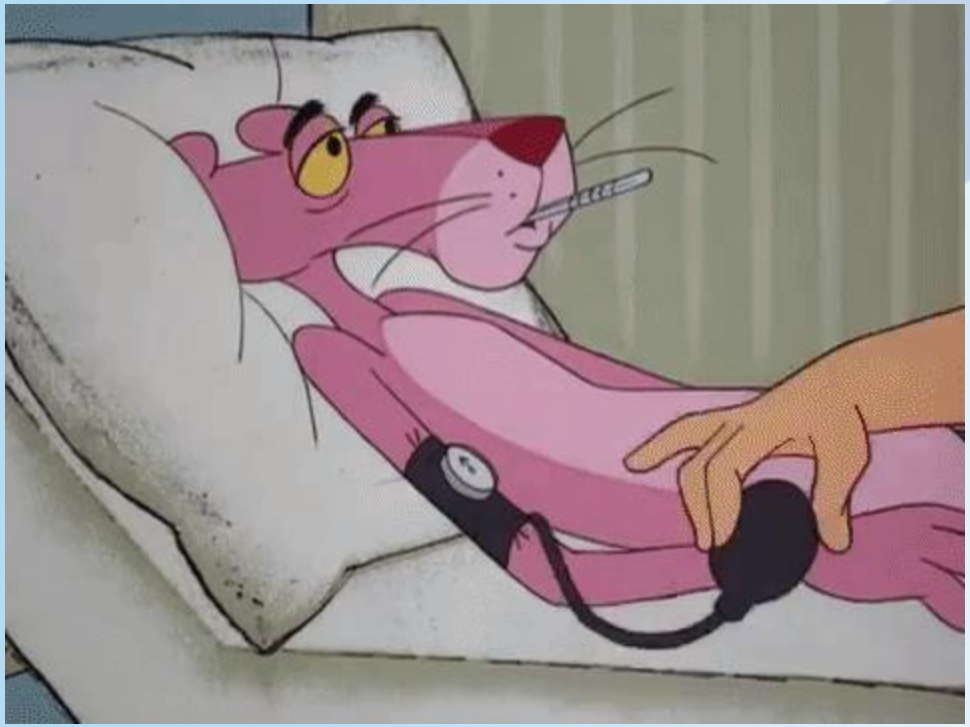
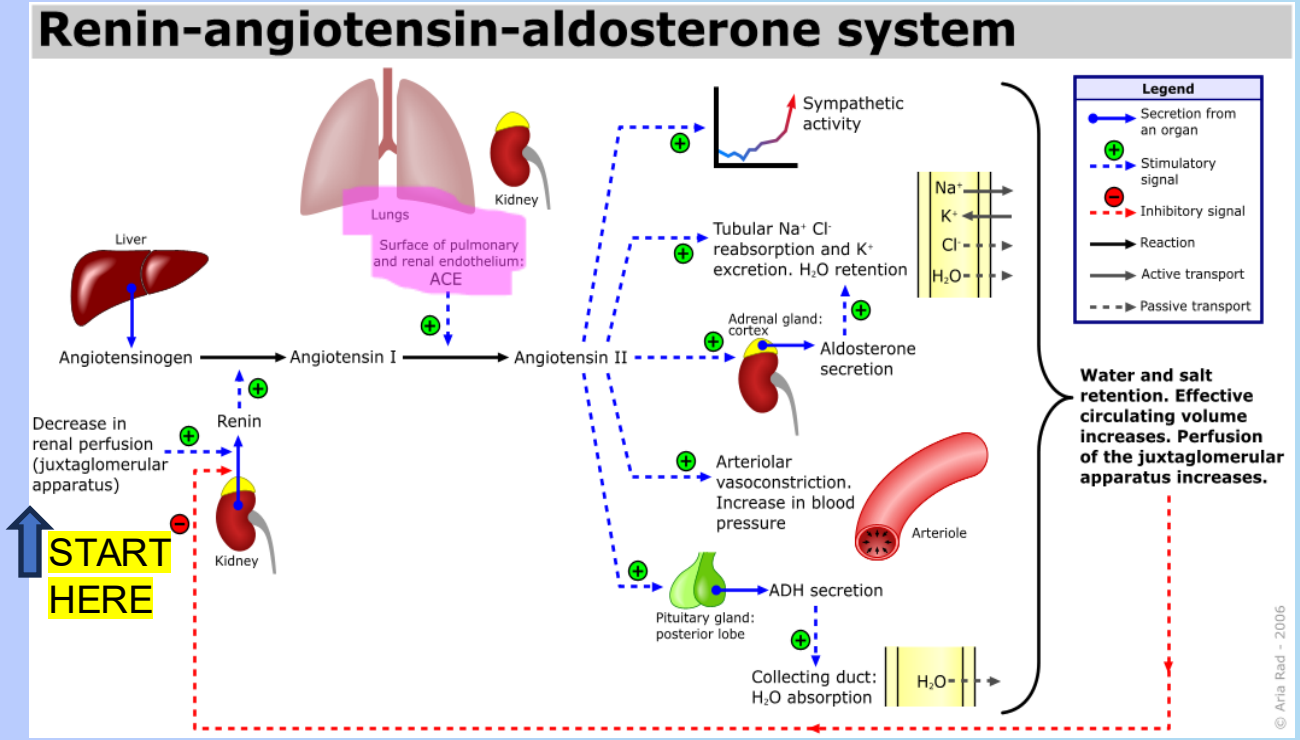


Renin-Angiotensin-Aldosterone System: Trigger Points



- **RAAS activated when:**
- ↓ BP
- ↓ Renal perfusion
- ↓ NaCl delivery to macula densa
- ↑ Sympathetic tone (β_1)

- 📌 **Key Starting enzyme: Renin**

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RAAS Cascade (Memorize Cold)

Renin (kidney)

↘

Angiotensinogen

↘

Ang I

↘

ACE (lungs)

↘

Ang II

↗

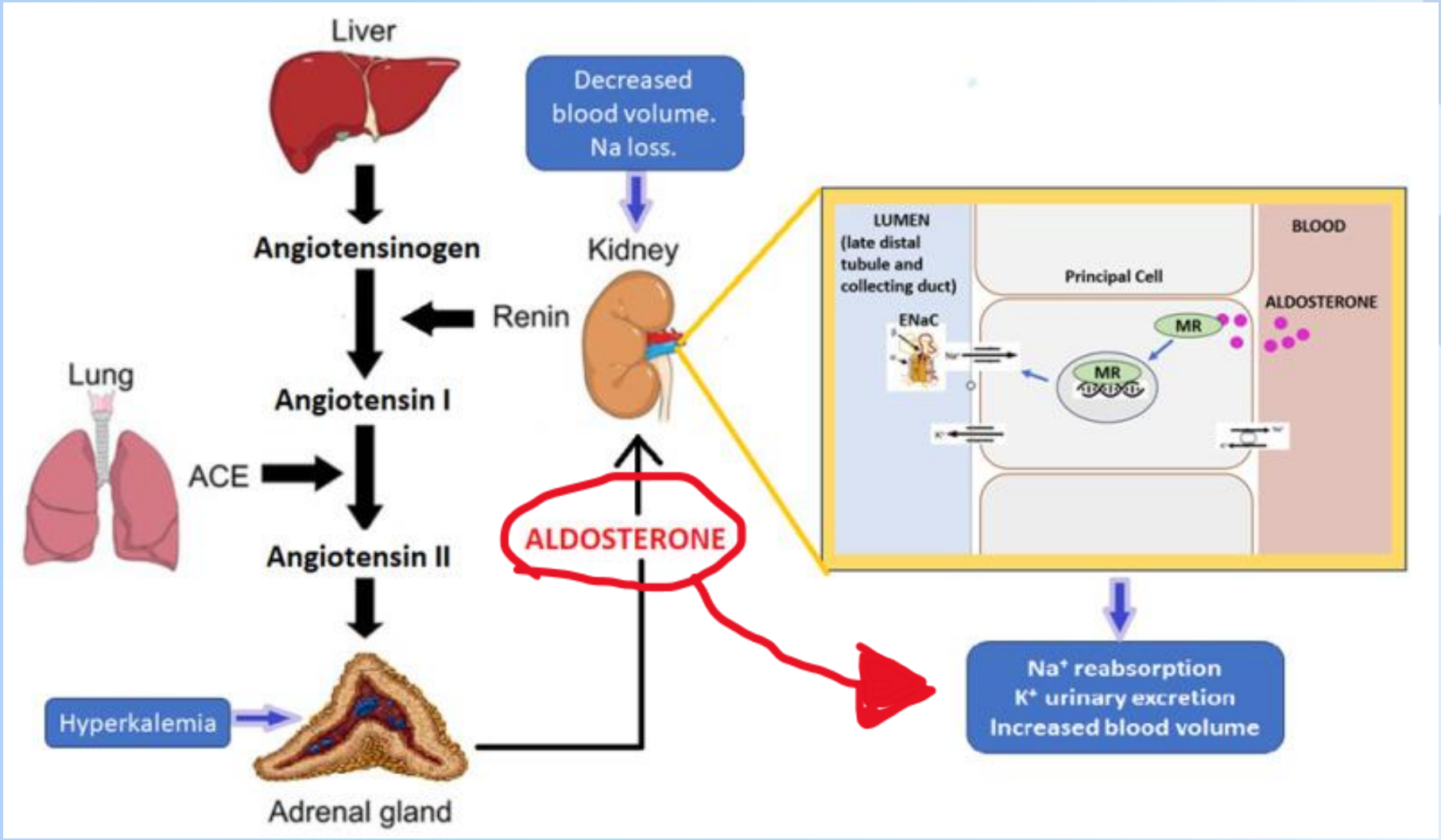
- Angiotensin II effects:
  - Vasoconstriction
  - Aldosterone release
  - ADH release
  - ↑ Sympathetic tone

- **Aldosterone**

- **Acts on:**
  - Distal tubule
  - Collecting duct

- **Effects:**
  - ↑ Na^+ reabsorption
  - ↑ Water retention
  - ↑ K^+ excretion
  - ↑ H^+ excretion

- 🧠 **Mnemonic:**
  "Aldo saves Salt, dumps K^+ & H^+"

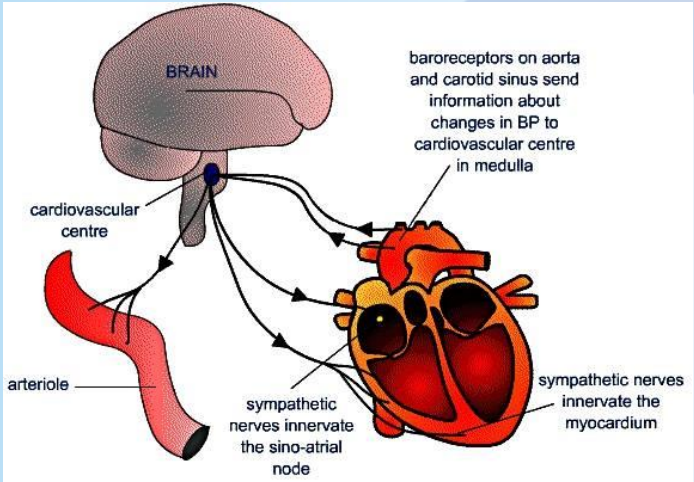

Liver
Decreased blood volume. Na loss.
Angiotensinogen
Kidney
Renin
Lung
Angiotensin I
ACE
Angiotensin II
ALDOSTERONE
Hyperkalemia
Adrenal gland
LUMEN (late distal tubule and collecting duct)
Principal Cell
BLOOD
ENaC
MR
MR
ALDOSTERONE
Na⁺ reabsorption
K⁺ urinary excretion
Increased blood volume

Short vs. Long-Term BP Control

Short term

- **Baroreceptors (carotid sinus + aortic arch):**
  - ↑ BP → ↑ firing → parasympathetic = lower blood press.
  - ↓ BP → ↓ firing → sympathetic = raise blood press.
- **Effects:**
- HR ↑
- Contractility ↑
- Vasoconstriction ↑

- **Long-term: RAAS**
  - Days to weeks



System	Speed
Baroreceptors	Seconds
Sympathetic	Minutes
RAAS	Days - Weeks

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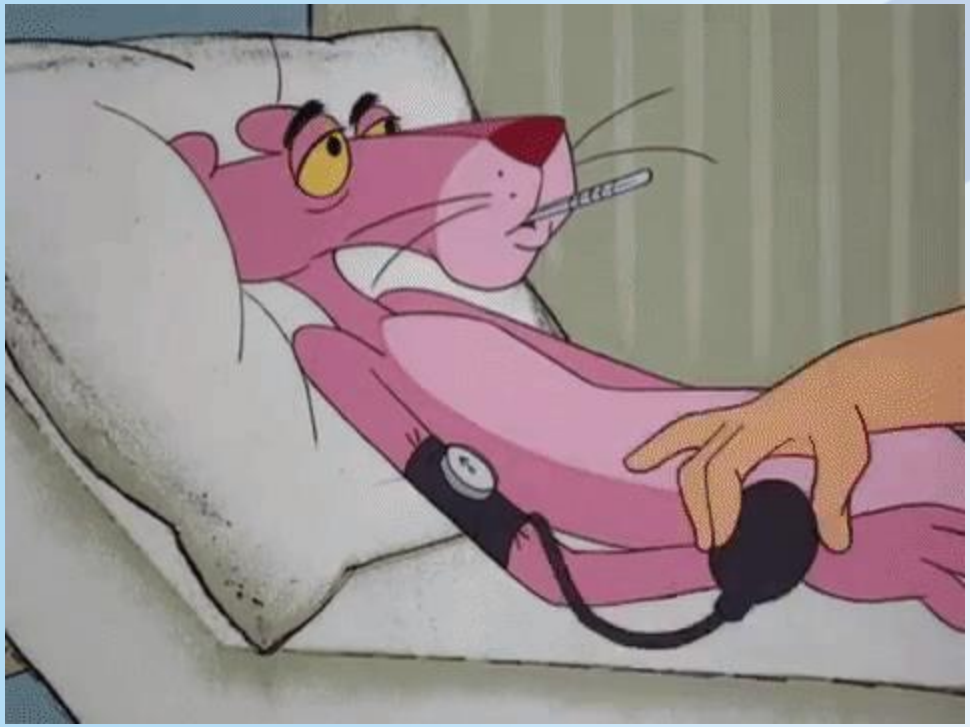

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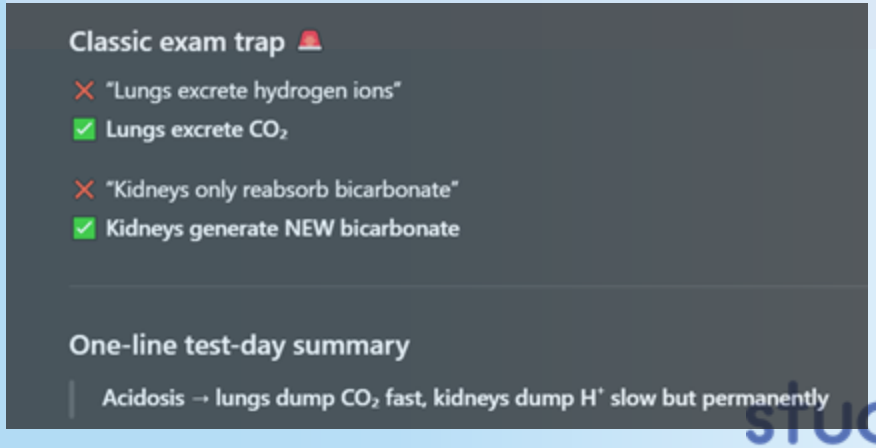
Metabolic Acidosis vs. Alkalosis

Acidosis

- ↓ pH ↓ HCO_3^-
- So how does the body remove Acid? (H+)
- **Lungs hyperventilate**
  - **Kussmaul Respiration**
  - ↓ CO_2 → ↓ H^+ → **pH rises**
  - $H^+ + HCO_3^- \rightleftharpoons H_2CO_3 \rightleftharpoons CO_2 + H_2O$
- **Kidneys excrete H^+**
  - $NH_3 + H^+ \rightarrow NH_4^+$
  - NH_4^+ gets trapped and excreted

Acidosis cont'd

- **More Kidney Action**
  - H^+ binds to **phosphate (HPO_4^{2-})**
  - Excreted as **$H_2PO_4^-$**
    - For every H^+ excreted → **1 new HCO_3^- added to blood**
    - 📌 Limited by available phosphate



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Metabolic Acidosis vs. Alkalosis

ALKALOSIS

- ↑ pH ↑ HCO_3^-
  - Kidneys excrete HCO_3^-
  - Lungs hypoventilate

- **Respiratory Compensation**
  - ↓ Ventilation
  - ↑ CO_2 retained
  - $CO_2 + H_2O \rightarrow \uparrow H^+$

- pH pulled **back down toward normal**
  - 📌 *Limited by hypoxia!*
    (You can't stop breathing forever just to fix pH)

ALKALOSIS cont'd

Kidney Response (Long-term)

- ↓ H^+ secretion
- ↓ NH_4^+ production
- ↑ **Bicarbonate excretion in urine**

- 📌 Urine becomes **alkaline**

SIMPLIFYING ACID-BASE DISORDERS

Condition	pH	$PaCO_2$	HCO_3^-	Causes
Respiratory Acidosis	↓	↑ (>45)	normal/ ↑	COPD, pneumonia, drug overdose, hypoventilation
Respiratory Alkalosis	↑	↓ (<35)	normal/ ↓	Hyperventilation, anxiety, high altitude
Metabolic Acidosis	↓	normal/ ↓	↓ (<22)	DKA, renal failure, diarrhea, lactic acidosis
Metabolic Alkalosis	↑	normal/ ↑	↑ (>26)	Vomiting, diuretics, excessive antacid use

Question

A 17-year-old male experiences an anxiety attack and is told by a doctor to place a paper bag over his mouth and breathe in and out of the bag.
As he continues to breathe into this bag, his rate of breathing continues to increase. Which of the following is responsible for the increased ventilation?

A. Increased alveolar PO_2
B. Increased alveolar PCO_2
C. Decreased arterial PCO_2
D. Increased pH

If an anxiety attack leads to hyperventilation, what changes to blood pH can we observe? →

By breathing into a paper bag, we don't allow as much CO2 to escape into the environment. Instead, it is inhaled back into the lungs → useful in hyperventilation

A. Increased Alveolar PO2 would be observed without a paper bag, and continued hyperventilation
C. Decreased arterial PCO2 would also be observed with continued hyperventilation
D. Increased pH would be observed with continued hyperventilation (less CO2 = less acidic blood = more alkaline products in blood)

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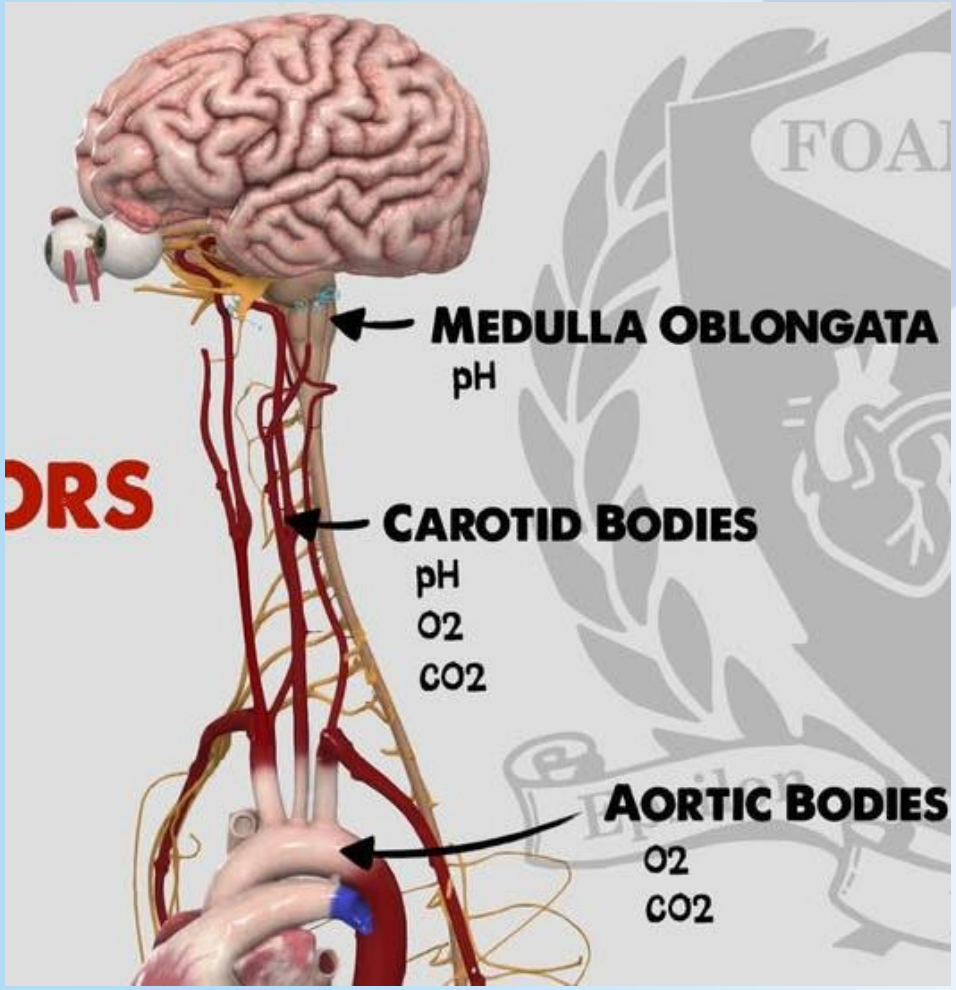
C. Decreased arterial PCO_2
D. Increased pH

Respiratory Compensation

- ↑ CO_2 = ↓ pH

- Central chemoreceptors respond to **Cerebral Spinal Fluid pH**
- Peripheral chemoreceptors respond to O_2
- **Exam** 📌 Most potent respiratory stimulus:
  - ✓ **Low CSF pH**



Ventilation-Perfusion Matching

Goal:
Match air (ventilation) with blood (perfusion) to maximize gas exchange

- **What happens when alveolar PO_2 is LOW?**
- **Scenario:**
  - Obstructed airway (mucus, tumor, pneumonia)
  - Poor ventilation → ↓ alveolar PO_2
  - **Pulmonary arteriole response = Vasoconstriction**

- **Why?**
  - No point sending blood to an alveolus with no oxygen
  - Blood is **redirected** to better-ventilated alveoli

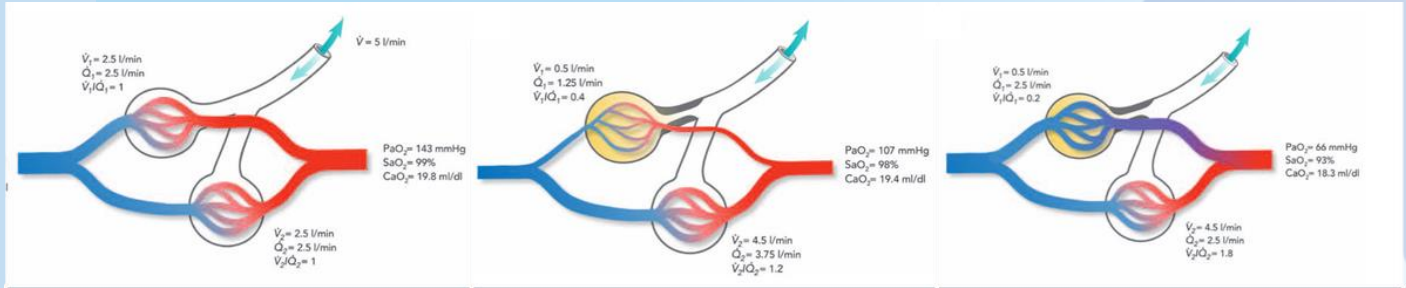
- 📌 This is called **Hypoxic Pulmonary Vasoconstriction (HPV)**

Ventilation-Perfusion Matching cont'd

Why is this OPPOSITE to systemic circulation?

- **Systemic tissues:**
  - Low O_2 → vasodilation (to bring **MORE blood**)
- **Lungs:**
  - **Low O_2 → vasoconstriction (to SEND blood AWAY**, blood won't receive enough O2)

📌 **Exam pearl:**
Pulmonary circulation behaves **opposite** to systemic



Normal. Notice that the V/Q is 1 is all segments.	Hypoxic vasoconstriction redirects blood (Q) from areas of poor ventilation to those with high ventilation (V). This tends to bring V/Q more towards normal.	WithOUT hypoxic vasoconstriction, the well ventilated segments have decreased Q. This leads to high V/Q (i.e., deadspace) ventilation and worsened hypercarbia.

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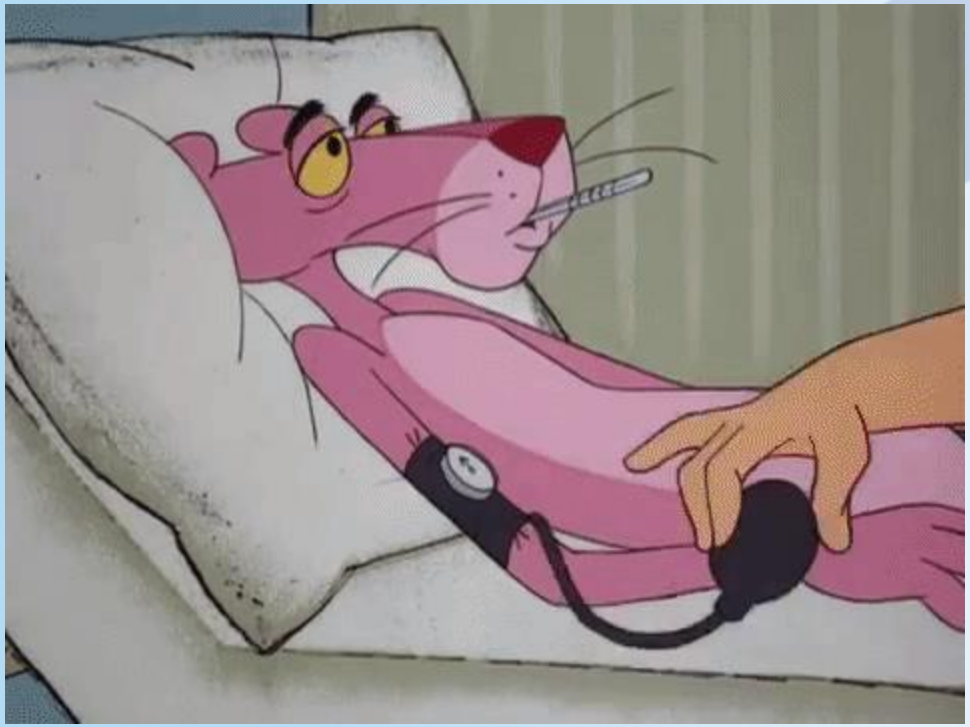

Questions to Bring it all Together

QUESTION 1

A 23-year-old man with type 1 diabetes mellitus is brought to the emergency department with nausea, abdominal pain, and rapid breathing. Laboratory studies show:

pH: **7.12**	[7.35 – 45]
HCO_3^-: **10 mEq/L**	[22 - 26]
PCO_2: **25 mmHg**	[35 – 45]

Which of the following renal processes is most responsible for long-term correction of his acid–base disturbance?

A. Increased bicarbonate reabsorption in the proximal tubule
B. Decreased hydrogen ion secretion in α-intercalated cells
C. Increased ammonium (NH_4^+) excretion
D. Increased chloride reabsorption in β-intercalated cells
E. Increased aldosterone secretion

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🧠 **Step-by-step reasoning**

- This patient has **metabolic acidosis** (low pH, low HCO_3^-)
- Lungs are already compensating → **low PCO_2**
- **Long-term correction = kidneys**
- Only mechanism that:
- Removes **fixed acid,** Generates **new bicarbonate &** Can be upregulated massively

→ **NH_4^+ excretion**

🚨 **Why the others are wrong**

A: Reabsorbing bicarbonate does NOT remove acid
B: H^+ secretion increases, not decreases
D: That happens in alkalosis
E: Aldosterone may worsen alkalosis

🎯 **Ammonium excretion is the kidney's most powerful weapon against chronic metabolic acidosis.**

QUESTION 2

A 45-year-old woman presents with weakness and lightheadedness after several days of persistent vomiting. Labs show:
pH: **7.55** [7.35 – 45]
HCO_3^-: **36 mEq/L** [22 - 26]
Serum chloride: **low**
Urine chloride: **low**

Which of the following mechanisms is primarily responsible for maintaining her alkalosis?

A. Increased ammonium production
B. Activation of pendrin due to high chloride levels
C. Volume depletion leading to increased aldosterone secretion
D. Decreased hydrogen ion reabsorption in the proximal tubule

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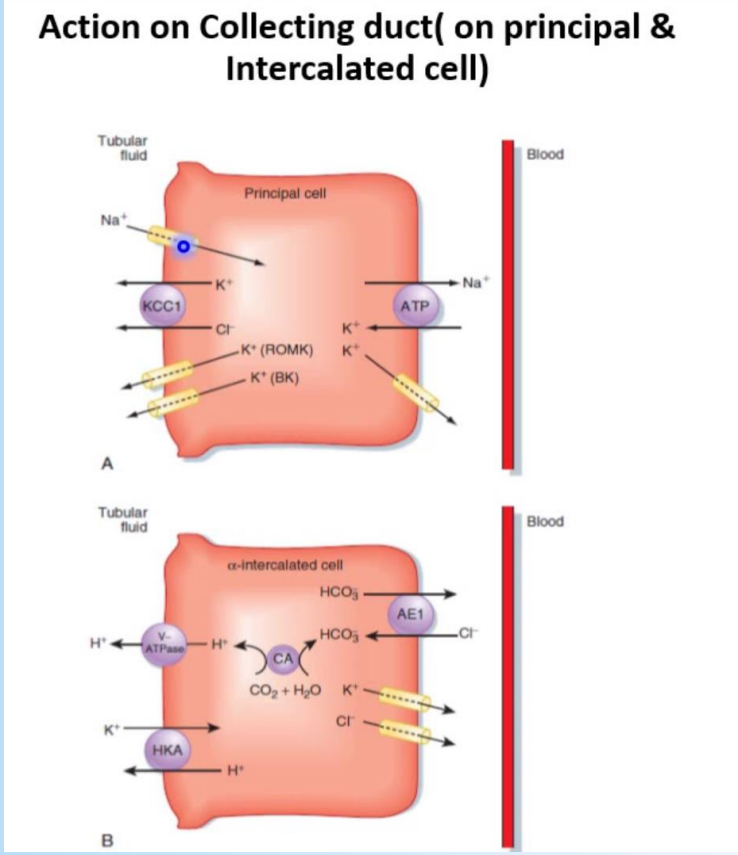

Step-by-step reasoning

- **Vomiting** → loss of HCl (hydrogen-chloride)
- This is chloride-responsive **metabolic alkalosis**
- Volume depletion → RAAS activation
- Aldosterone: ↑H^+ secretion ↑HCO_3^- generation

Why the others are wrong

A: NH3 → NH4 (ammonium) helps excrete ACID
B: High chloride? We have low Cl-
D: in the PCT, H+ is EXCRETED to lumen, not reabsorbed

Chloride depletion and volume depletion lock in metabolic alkalosis.

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QUESTION 3

A patient with lobar pneumonia has consolidation of the right lower lung.

Which of the following best describes the physiologic response in the affected lung region?

A. Vasodilation of pulmonary arterioles due to hypoxia
B. Vasoconstriction of pulmonary arterioles due to hypoxia
C. Bronchoconstriction due to increased PO_2
D. Increased perfusion to improve oxygen delivery
E. Increased ventilation of the affected alveoli

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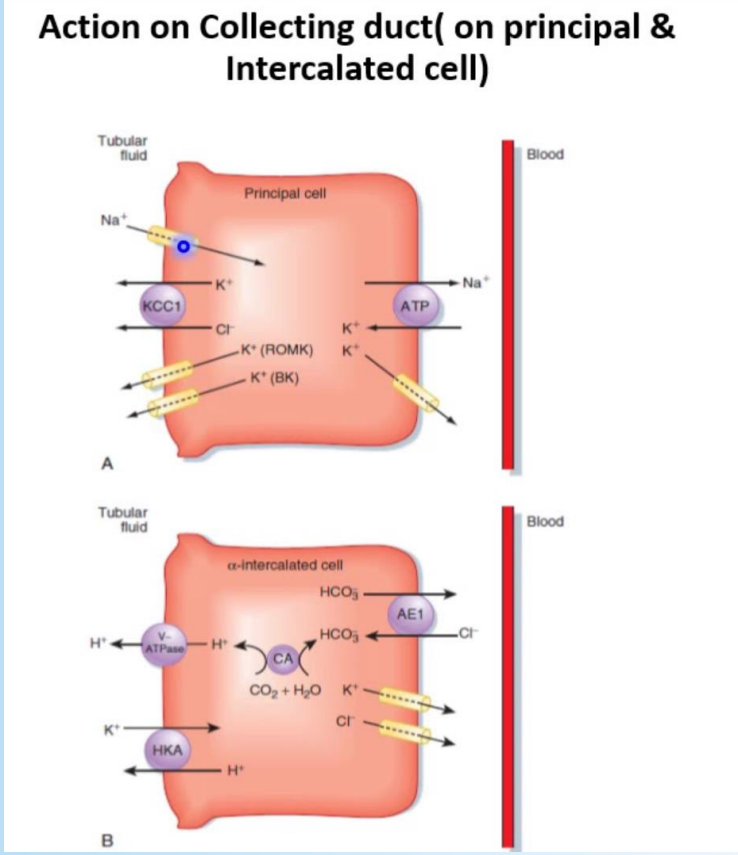


🧠 Step-by-step reasoning

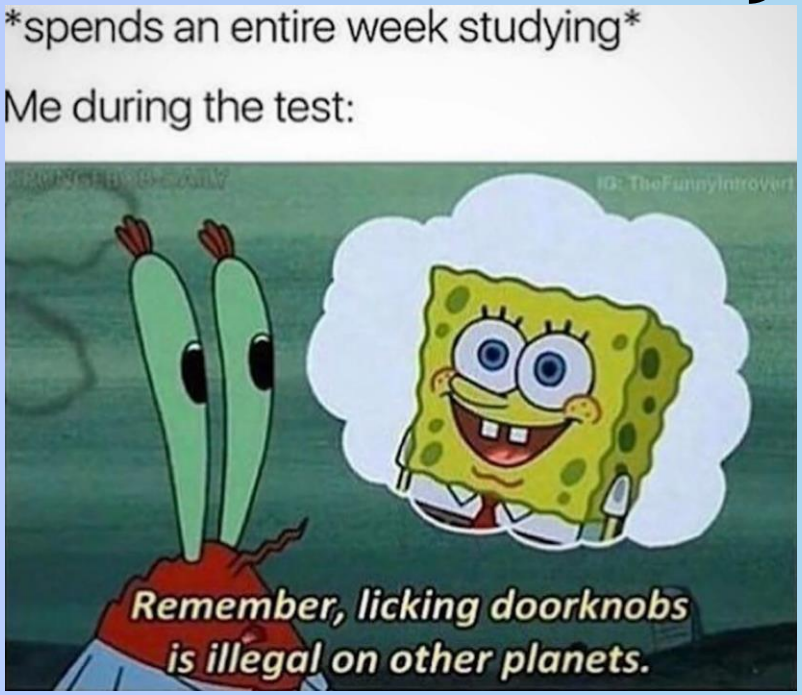
- Pneumonia → ↓ alveolar PO_2
- Pulmonary circulation responds to **O_2**
- Low O_2 → **hypoxic pulmonary vasoconstriction**
- Blood is redirected to better-ventilated alveoli

Systemic vessels: low O_2 → dilation

Pulmonary vessels: low O_2 → constriction

🎯 **Pulmonary vessels constrict in response to hypoxia to optimize ventilation–perfusion matching.**

Thanks for listening! Try not to RAAiSe your BP on test day

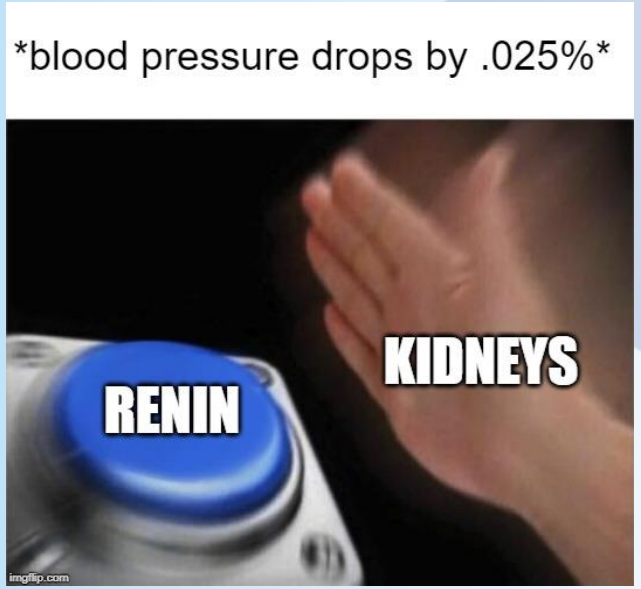


Heart: * fails to pump blood to the body*

Kidney: * activates RAAS to increase volume*

Heart: * pumps even worse*

Kidney:



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