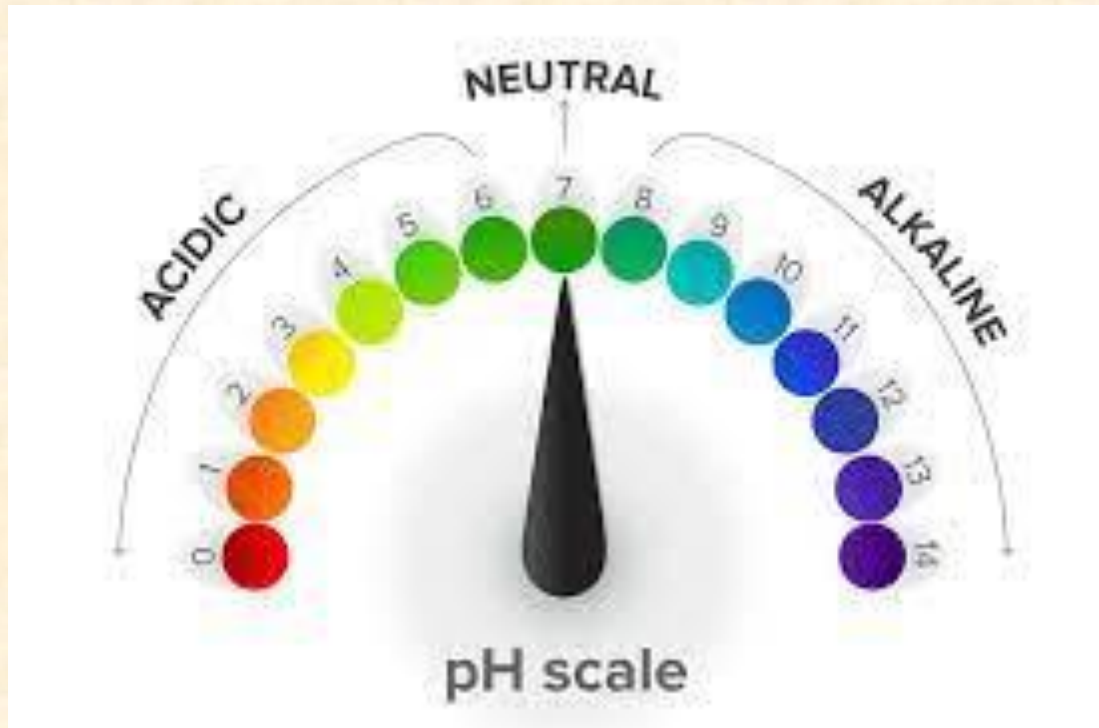


ACID BASE BALANCE BY DR/ HEBA KAREEM

لارم تكون الحماية ضد ال acidosis لانه الجسم بطبيعة الاحوال
مثلا بال glycolysis رح تنتج احماض ف لازم اسوي حماية
ضدها



☆ گا اذہب باقی
مکن کھینچ
acid و base

Maintenance of blood pH

! acidosis is more dangerous

- The normal pH of the blood is maintained in the narrow range of 7.35- 7.45 (slightly alkaline).

If the range is interrupted it may cause neither acidosis or alkalosis

- The body has developed **three lines of defense** to regulate the body's acid-base balance

Bicarbonate to carbonic acid 20:1 > alkalise

- **1- Blood buffers**

Weak acid with strong base

Carbonic acid
Bicarbonate > alkaline

As a buffer there should be alkaline more than acid due to the defence mechanism.

- **2- Respiratory mechanism**

- **3- Renal mechanism**

- **Blood buffers:**

- **A buffer may be defined as a solution of a weak acid & its salt with a strongbase**

Blood contains three buffer systems

base / alkali
↑

- Bicarbonate buffer
- Phosphate buffer
- Protein buffer

bicarbonate % > Carbonic acid
x20, since i want to
treat acidosis

□ Bicarbonate buffer system:

- Sodium bicarbonate & carbonic acid (NaHCO_3^- H_2CO_3) is the most predominant buffer system of ECF.
- Carbonic acid dissociates into hydrogen and bicarbonate ions.



□ The blood pH 7.4, the ratio of bicarbonate to carbonic acid is 20: 1

Acidosis can be due to the decrease in carbonic acid and increase bicarbonate base

□ The bicarbonate concentration is much higher (20times) than carbonic acid in the blood.

□ This is referred to as alkali reserve.
مخزون
↑ another name for buffer system
مندی کثیر base بطلعه کا یکنون بی کثیر acid

Respiratory mechanism for pH regulation

□ A rapid mechanism. But can't be used in long term

□ This is achieved by regulating the concentration of carbonic acid (H_2CO_3) in the blood.

↓

↑ in acidosis so, hyper to fix & avoid its dissolving

hyper → ↑ basic

hypo → ↑ acidic

In the RBC the mechanism is anaerobic due to the absence of mitochondria

The large volumes of CO₂ produced by the cellular metabolic activity. All of this CO₂ is eliminated from the body in the expired air via the lungs



The respiratory centre is highly sensitive to changes in the pH of blood.

Decrease in blood pH causes hyperventilation to blow off CO₂ & reducing the H₂CO₃ concentration

- Respiratory control of blood pH is rapid but only a short term regulatory process, since hyperventilation cannot proceed for long.

Hemoglobin as a buffer

- Hemoglobin binds to H^+ ions & helps to transport CO_2 as HCO_3^- with a minimum change in pH.
- In the lungs, hemoglobin combines with O_2 , H^+ ions are removed which combine with HCO_3^- ^(+H) to form H_2CO_3 & is dissociates ^(-H) to release CO_2 to be exhaled.

Generation of HCO_3^- by RBC → ⊗ mito "cell long"

- Due to lack of aerobic metabolic pathways, RBC produce very little CO_2 .
- The plasma CO_2 diffuses into RBC along the concentration gradient, it combines with water to form H_2CO_3 by **Carbonic anhydrase**.
- In RBC, H_2CO_3 dissociates to produce H^+ & HCO_3^- .
- The H^+ ions are buffered ^① by Hemoglobin.
- As the concentration of HCO_3^- increases in the RBC, it diffuses into plasma along with concentration gradient, in exchange for **Cl^- ions**, to maintain electrical neutrality.



High
higher in plasma

Low

dissolves it

CA dissociation

low
*

High



-ve out

258

-ve in





acidosis, alk

acidosis (acidosis)

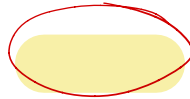
Excretion of H^+ ions

- Kidney is the only route through which the H^+ can be eliminated from the body.
- H^+ excretion occurs in the proximal convoluted tubules & is coupled with generation of HCO_3^- .
- Carbonic anhydrase catalyses the production of carbonic acid (H_2CO_3) from CO_2 & H_2O in renal tubular cells.
- H_2CO_3 then dissociates to H^+ & HCO_3^-
- H^+ ions are secreted into tubular lumen in exchange for Na^+
- Na^+ in association with HCO_3^- is reabsorbed into blood

④ ✓

reabsorbed ١/٢
NOT wasted

3



2

نبداء
موت



+ve

لاذخ

+ve

"sodium reab."

+

HCO_3^-
ضالع اخصه

++

into

H_2CO_3

↑
بترجع
 $\text{CO}_2 + \text{H}_2\text{O}$

HCO_3^+
reabsorbed
before its
thrown out
بس مينا كمالها

Reabsorption of Bicarbonate

- This mechanism is responsible to conserve blood HCO_3^- , with simultaneous excretion of H^+ ions.
- Bicarbonate freely diffuses from plasma into tubular lumen.
- HCO_3^- combines with H^+ , secreted by tubular cells, to form H_2CO_3 .
- H_2CO_3 is then cleaved to form CO_2 and H_2O .
- As the CO_2 concentration builds up in the lumen, it diffuses into the tubular cells along the concentration gradient.

- In the tubular cell, CO_2 again combines with H_2O to form H_2CO_3 which then dissociates into H^+ & HCO_3^-
- The H^+ is secreted into the lumen in exchange for Na^+ .
- The HCO_3^- is reabsorbed into plasma in association with Na^+ .
- Reabsorption of HCO_3^- is a cyclic process without net excretion of H^+ ^① or generation of new HCO_3^- ^②

⊛

alkalining
urine
until it
matches
blood pH

base



! how much [sodium hydroxide] we add
to urine to match its $\text{pH} = 7.4$ to bloods

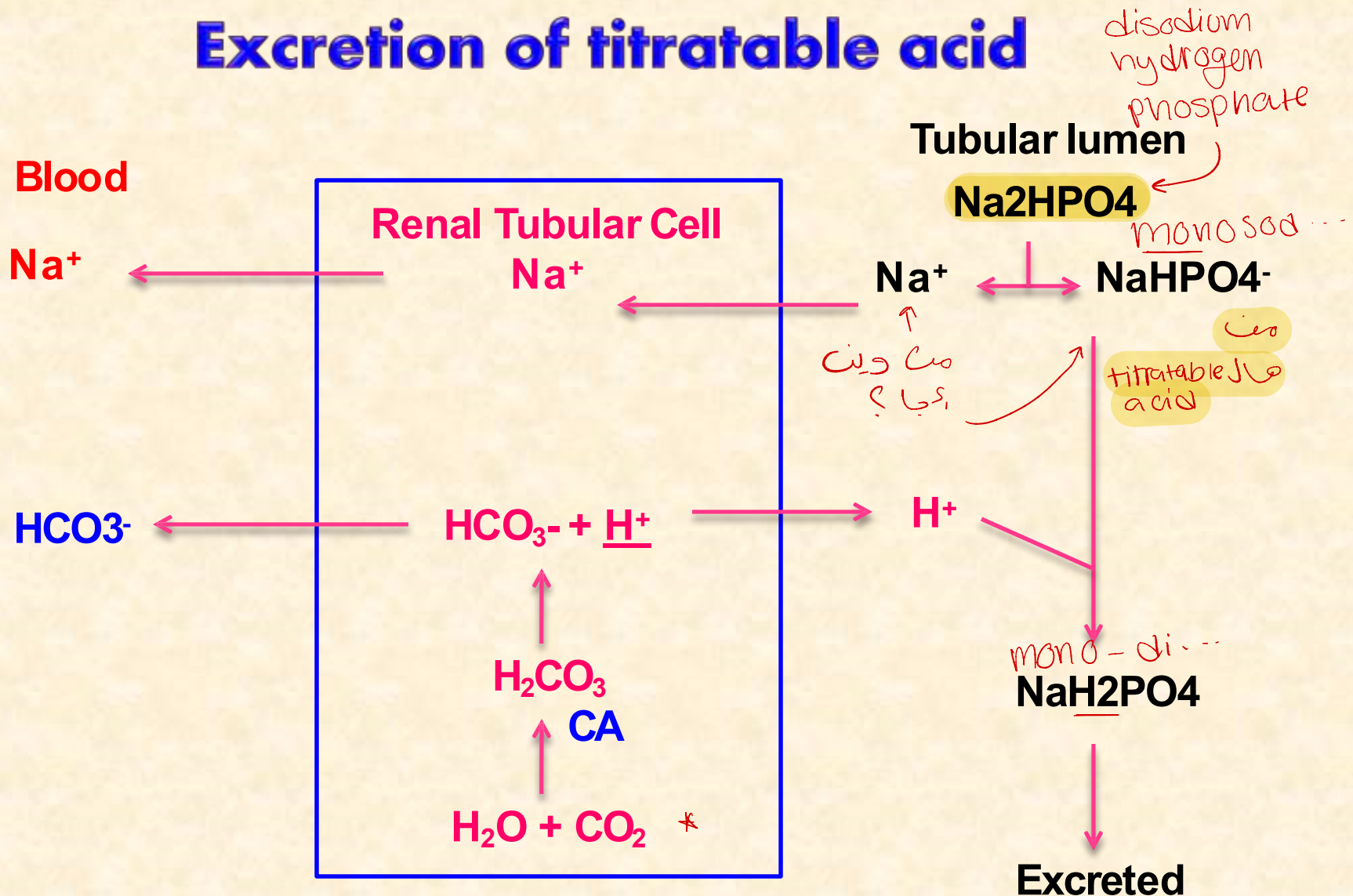
$\text{pH} = 7.35 - 7.45$

- H^+ ions are secreted into the tubular lumen in exchange for Na^+ ion.
- This Na^+ is obtained from the base, disodium hydrogen phosphate (Na_2HPO_4).
- This combines with H^+ to produce the acid, sodium dihydrogen phosphate (NaH_2PO_4), in which form the major quantity of titratable acid in urine is present.
- Tubular fluid moves down the renal tubules,

more and more H^+ ions are added, resulting in the acidification of

urine. Causes a fall in the pH of urine as low as 4.5.

Excretion of titratable acid

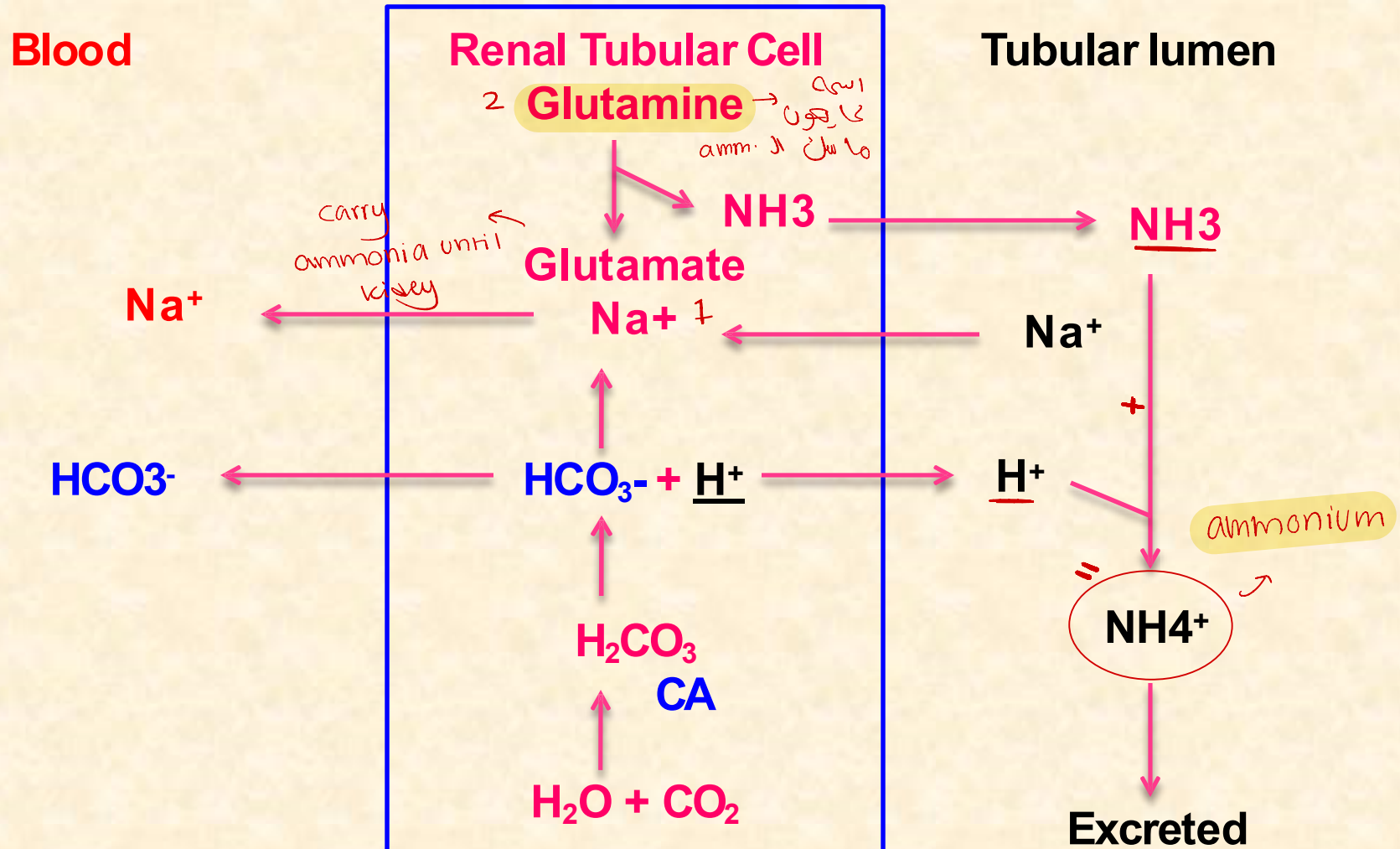


Excretion of ammonium ions

- The H^+ ion combines with NH_3 to form ammonium ion (NH_4^+).
- The renal tubular cells deaminate glutamine to glutamate and NH_3 by the action of enzyme glutaminase.
- The liberated NH_3 diffuses into the tubular lumen where it combines with H^+ to form NH_4^+ .
- Ammonium ions cannot diffuse back into tubular cells and excreted into urine.

Ammonia is toxic
One of the ways in excreting it is by urine

Excretion of ammonium ions



Acidosis

Blood PH tend to ↓

1820

مازاد خالی

Results from **formation of excessive acids**
More than the capacity of the body to eliminate them

↑ carbonic
↓ bicarb

Respiratory ⊕
acidosis

Metabolic
acidosis

Respiratory acidosis

Q.
cases.

hypo ventilation ❗ أسباب

↑ **CO₂ (CO₂ RETENTION)** due to

- ❑ Bronchial asthma
- ❑ Chronic bronchitis
- ❑ Emphysema
- ❑ Pneumonia
- ❑ Respiratory centre inhibition
- ❑ Asphexia

If lung is abnormal kidney will fix the case

↑ **CO₂** → ↑ **blood H₂CO₃**

Respiratory acidosis

$\uparrow \text{CO}_2$ \swarrow \uparrow blood H_2CO_3
 \searrow HCO_3^- not changed
 $\rightarrow \downarrow \text{HCO}_3^-/\text{H}_2\text{CO}_3$ (N=20:1)
 $\rightarrow \downarrow$ blood PH

(Uncompensated respiratory acidosis [acidemia])

How to compensate?

Kidney reabsorbs more HCO_3^-

Till normal $\text{HCO}_3^-/\text{H}_2\text{CO}_3$ (20:1)

\rightarrow PH reach 7.4

NOT hypervent.
cuz lung is not
healthy

Metabolic acidosis

↑ workout
kidney failure +
no reabs. ←

diarrhea →

↑ acids or ↓ bases (HCO_3^-) in blood

↓ blood HCO_3^-

blood H_2CO_3 not changed

→ ↓ $\text{HCO}_3^-/\text{H}_2\text{CO}_3$ (N=20:1)

→ ↓ blood PH

(Uncompensated metabolic acidosis [acidemia])

How to compensate?

↓ PH → ++ chemoreceptors in respiratory centre → hyperventilation → loss of CO_2 → ↓ H_2CO_3

Till normal $\text{HCO}_3^-/\text{H}_2\text{CO}_3$ (20:1)

→ PH reach 7.4 (Compensated metabolic acidosis)

Causes of Metabolic acidosis

1- ↑ blood acids

Lactic acidosis
Uncontrolled Diabetes inscp that will cause increase in ketone bodies

↑ production

- ↑ lactic acid in muscular exercise
- ↑ ketone bodies in Ketosis due to Diabetes mellitus
- ↑ acids from metabolism of different food stuffs (diet) as pyruvic , lactic, phosphoric and nucleic acids.

↓ excretion

- failure of excretion by the kidney in chronic renal failure

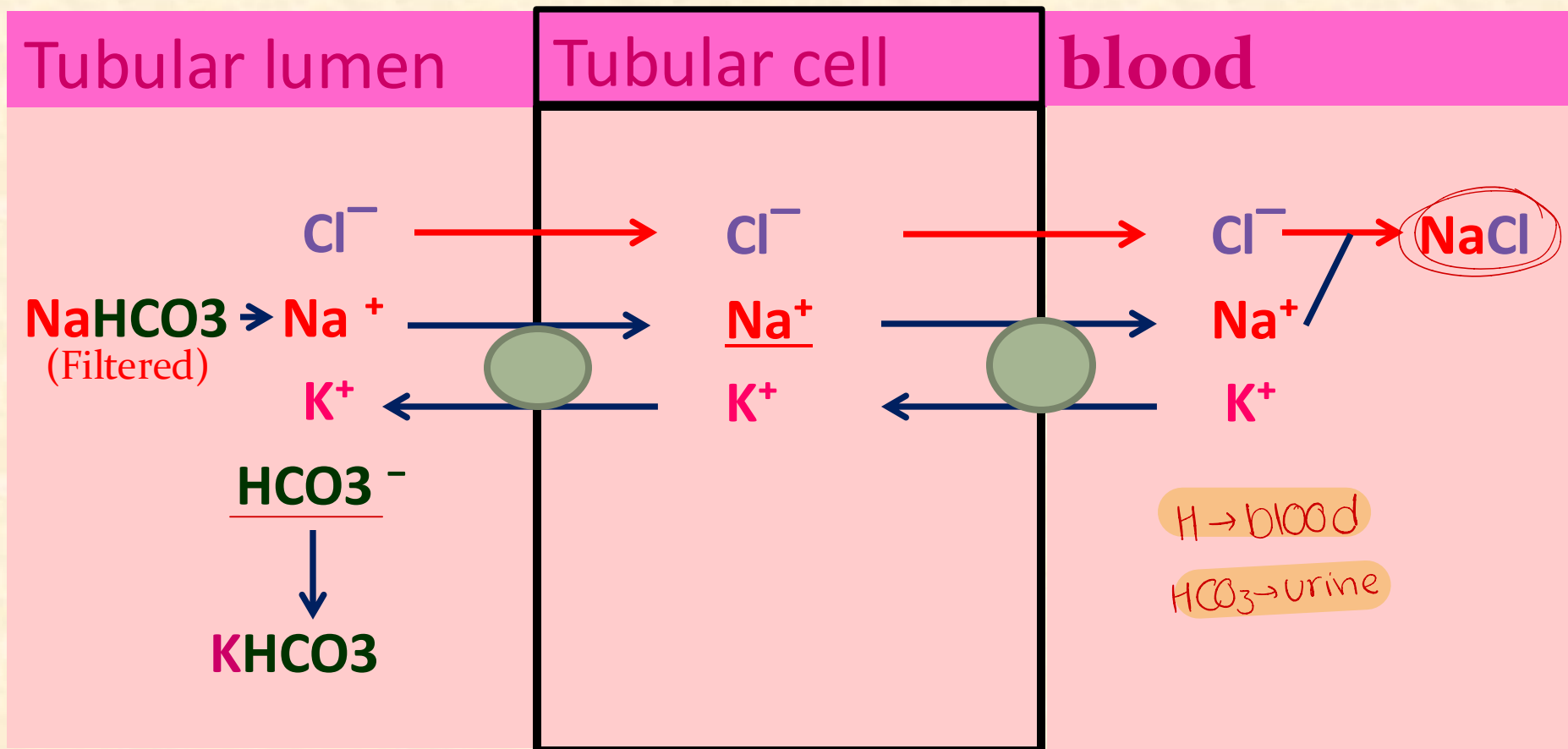
Causes of Metabolic acidosis

1- ↑ base loss

- ☐ ✓ Diarrhea: Intestinal juices are alkaline being rich in Na^+ & K^+ bicarbonate
 - ☐ ✓ Vomiting: due to low intestinal obstruction higher → gastric secretions are lost
 - ☐ Hyperkalemia: ✱ ✱ ✱
 - * ↑ renal tubular reabsorption of Na^+ in exchange with K^+ Na
K
←
→ H
 - stop of Na^+ / H^+ exchange
 - * Na^+ reabsorption will be in the form of NaCl not NaHCO_3 > HCO_3^- will be excreted in the form of KHCO_3 in urine.
 - HCO_3^- loss in urine → metabolic acidosis (Alkaline urine)
 - ↑ Cl in blood → hyperchloremic acidosis (Acidic blood)
- The alkaline urine & acidic blood is called paradoxical acidosis

Causes of Metabolic acidosis

1- ↑ blood acids



Alkalosis

Blood PH tend to ↑

Results from **formation of excessive bases**
More than the capacity of the body to neutralize & eliminate them

*hyper*vent
**Respiratory
alkalosis**

**Metabolic
alkalosis**

Respiratory alkalosis

آبازان hypervent

↑ CO₂ loss due to

- ☐ fever
- ☐ encephalitis
- ☐ high altitude
- ☐ late stages of salicylate poisoning ^{aspirin}
- ☐ hysterical hyperventilation → following emotional trauma

↓ CO₂ →

↓ blood H₂CO₃

وقت یزید
بمبارد
هتیر لانه

Respiratory alkalosis

↓ CO₂ → ↓ blood H₂CO₃
 ↘ HCO₃⁻ not changed
→ ↑ HCO₃⁻/H₂CO₃ (N=20:1)
→ ↑ blood PH

(Uncompensated respiratory alkalosis [alkalemia])

How to compensate?

-- of renal tubular reabsorption of HCO₃⁻

Kidney excretes more HCO₃⁻

Till normal HCO₃⁻/H₂CO₃ (20:1)

→ PH reach 7.4

(Compensated respiratory alkalosis)

Urine will be alkaline because of ↑ secretion of K⁺ & HCO₃⁻ in urine

Metabolic alkalosis

↑ bases or ↓ acids in blood

↘ ↑ blood HCO_3^-
↘ blood H_2CO_3 not changed

→ ↑ $\text{HCO}_3^-/\text{H}_2\text{CO}_3$ (N=20:1)

→ ↑ blood PH

(Uncompensated metabolic alkalosis [acidemia])

How to compensate?

↑ PH → - - - chemoreceptors in respiratory centre → hypoventilation → CO_2 retention → ↑ H_2CO_3

Till normal $\text{HCO}_3^-/\text{H}_2\text{CO}_3$ (20:1)

→ PH reach 7.4 (Compensated metabolic alkalosis)

Causes of Metabolic alkalosis

1- ↑absorption of bases

- ❑ Intake of high vegetable and fruit diet: They contain Bicarbonate salts and citrate salts. Citrate salts will be transformed into bicarbonate salts by krebs cycle
- ❑ Intake of drugs containing bicarbonate & citrate salts (drugs used for treatment of hyperacidity & peptic ulcer)

Causes of Metabolic alkalosis

2- ↑loss of acids

نشاط الدواء tube مع ال saline

□ Prolonged suction of gastric juice

→ after treating suicidal attempt after drug overdose

□ Vomiting due to high intestinal obstruction

الفرق
! ٨٥

□ Hypokalemia:

* ↓renal tubular reabsorption of Na^+ in exchange with K^+

→ instead there is Na^+ / H^+ exchange

* Na^+ reabsorption will be in the form of NaHCO_3 not NaCl →

Cl^- loss in urine in the form of NH_4Cl → hypochloremia and acidic urine

H out

Na in

HCO_3 in

↑ NaHCO_3 in blood → alkalosis (alkaline blood)

The acidic urine & alkaline blood is called paradoxical alkalosis

□ Cushing syndrome: → Na & water retention & K excretion → hypokalemia