Lecture No. 9

Role of the kidney in Acid Base Balance

Renal Regulation of Acid-Base Balance

I. Elimination of non-volatile acids (H_2SO_4, H_3PO_4)

II. Secretion of H^+

III. Reabsorption of HCO_3^-

IV. Production of new HCO_3^-

Renal Regulation of Acid-Base Balance

In Summary

- Kidneys eliminate non-volatileacids (H_2SO_4, H_3PO_4) (~ 80 mmol/day)
- Filtration of HCO_3^- (~ 4320 mmol/day)
- Secretion of H^+ (~ 4400 mmol/day)
- Reabsorption of HCO_3^- (~ 4319 mmol/day)
- Production of new HCO_3^- (~ 80 mmol/day)
- Excretion of HCO_3^- (1 mmol/day)

Kidneys conserve HCO_3^- and excrete acidic or basic urine depending on body needs

The Renal control of the Acid-Base Balance:

Reabsorption of filtered HCO_3^-.

- HCO_3^- is very precious: we can't really afford losing it in urine.

(Full reabsorption, primarily in the proximal tubules)

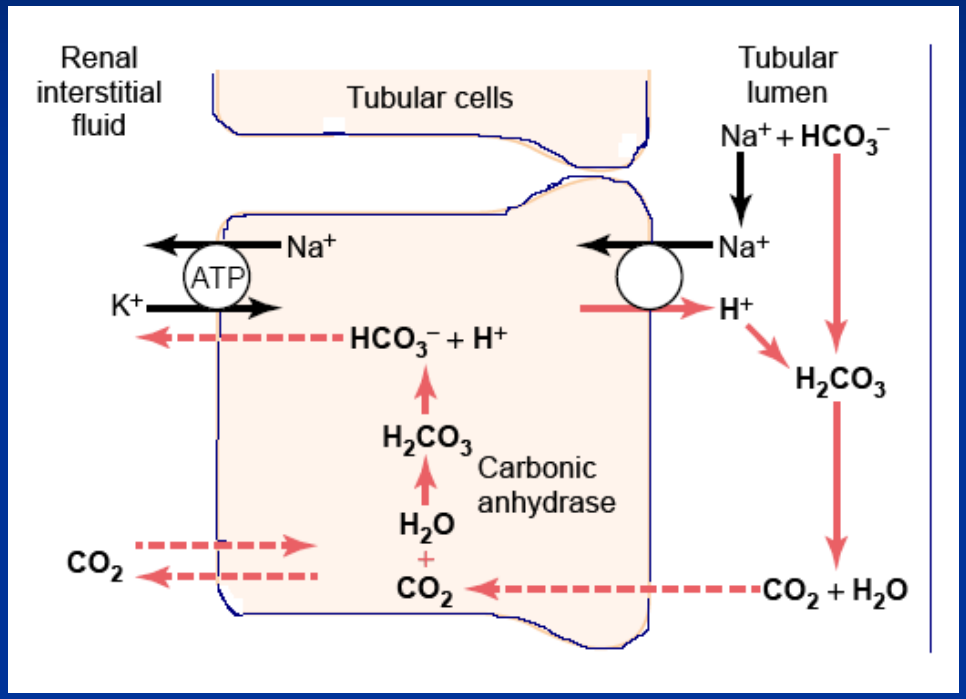
80-90% of the HCO_3^- reabsorption and thus H^+ secretion ccurs at proximal tubule, 10% in thick ascending, 4.9 % in collecting duct and distal tubule, and less than 0.1% is excreted.

- The filtered load of the bicarbonate is equal to
  - 180L/day * 24mEq/L = 4320 mEq/day.

- The clearance is of HCO_3^- is negative→

- Quantity aspect: The reabsorption is more important than the production since its amount (4320) is greater.

Mechanism of HCO_3^- Reabsorption and Na^+ - H^+ Exchange In Proximal Tubule and Thick Loop of Henle and Early distal Tubule



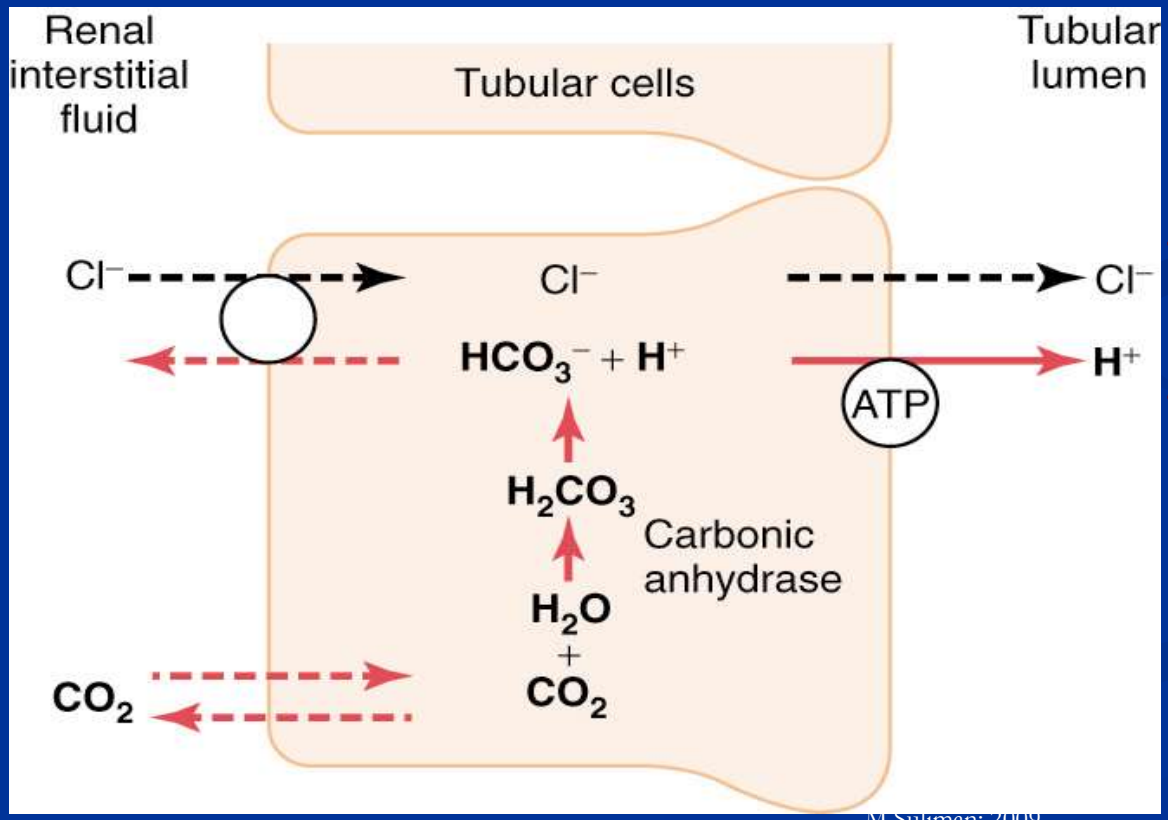
(1) Active secretion of H^+ ions into the renal tubule.

2. Tubular reabsorption of HCO3- by combination with H^+ ions to form carbonic acid, which dissociates to form carbon dioxide and water.

3) Sodium ion reabsorption in exchange for hydrogen ions secreted, by *secondary active hydrogen counter-transport*.

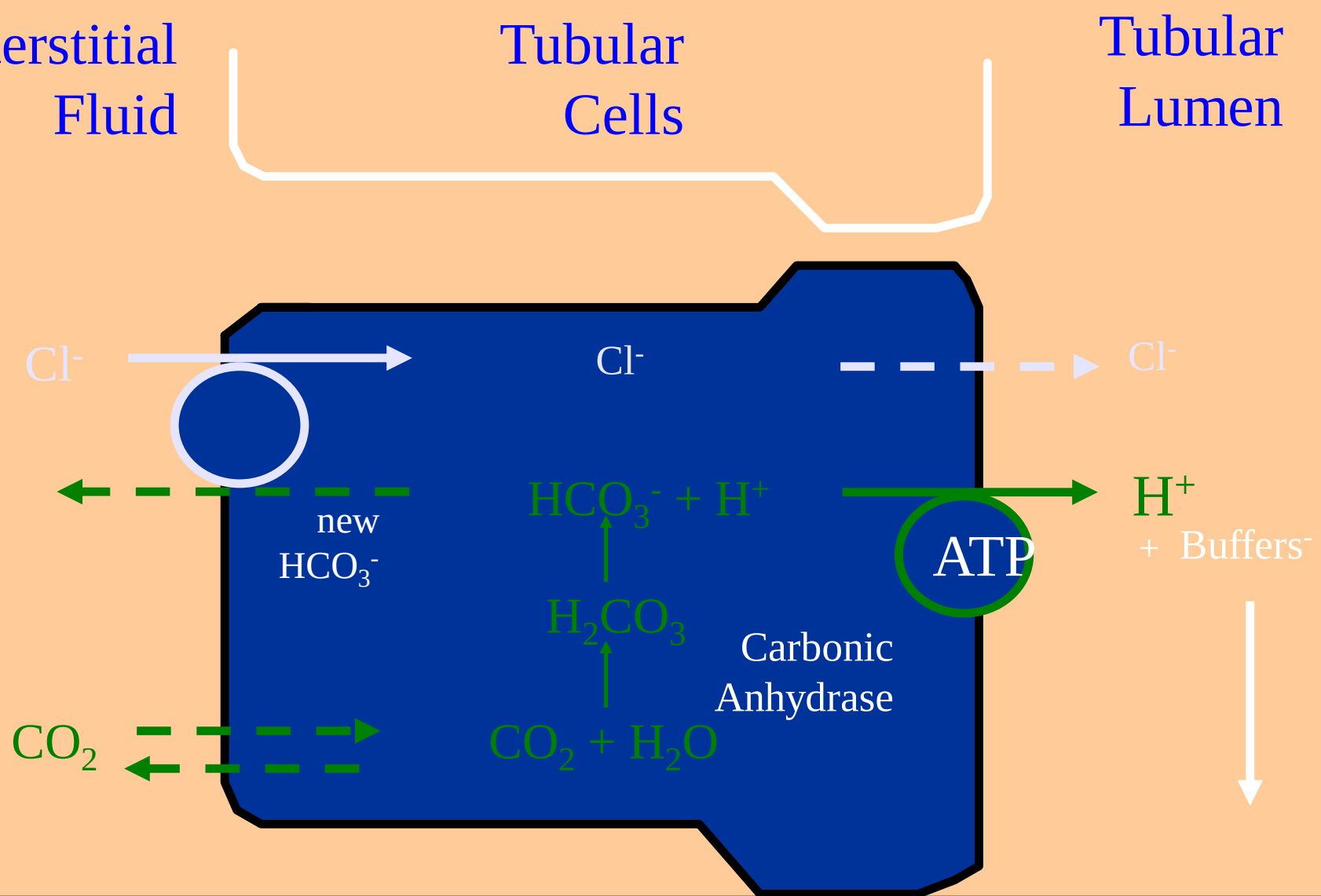
HCO_3^- Reabsorption and H^+ Secretion in Intercalated Cells of Late Distal and Collecting Tubules

H+ secreted by ***primary active transport*** in the intercalated cells of the late distal tubule and the collecting tubules via a H^+ATPase pump.

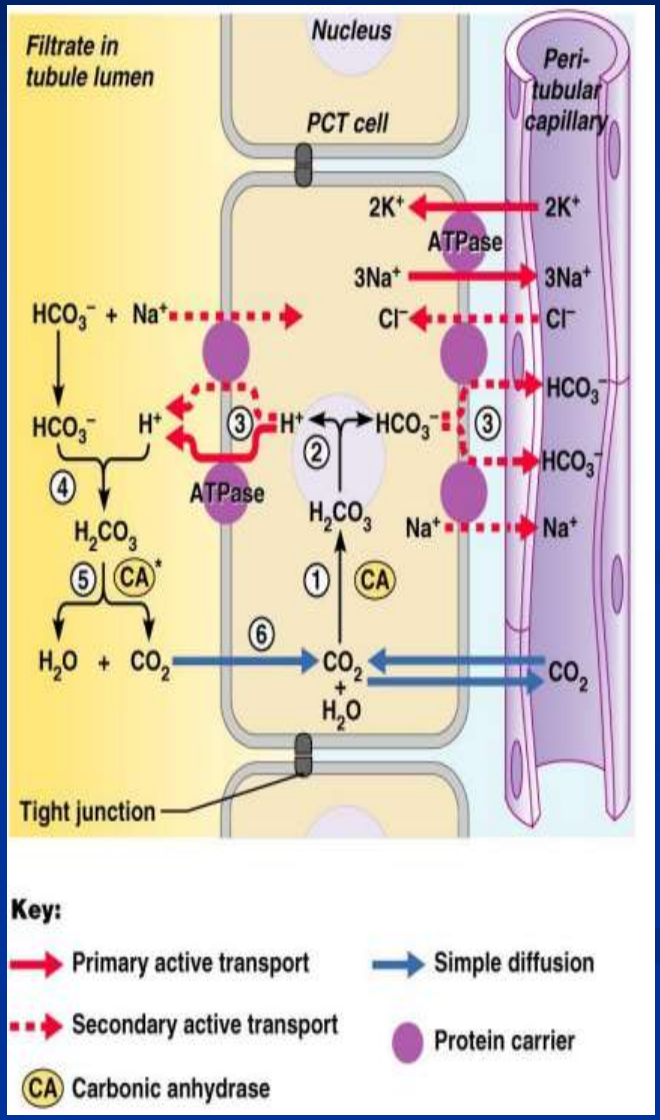


- H+ secretion represents 5% in this part of the tubule.
- H+ secretion in this part of the tubule is important in forming a maximally acidic urine.

In acidosis all HCO_3^- is titrated and excess H^+ in tubule is buffered



- Carbonic acid formed in filtrate dissociates to release carbon dioxide and water
- Carbon dioxide then diffuses into tubule cells, where it acts to trigger further hydrogen ion secretion



Bicarbonate is freely filtered.
Since it is a charged particle, it cannot cross the apical side and cannot be absorbed as it is.

Inside the cell, the $CO_2 + H_2O$ unite by carbonic anhydrase to form H_2CO_3 which dissociates to form $H^+ + HCO_3^-$

There is a bicarbonate carrier, at the basolateral side

H^+ is actively secreted in the tubule, it binds HCO_3^- in the tubular fluid forming CO_2 which diffuses inside the cell.

<u>Sources of Intracellular CO_2:</u>

1. Cellular metabolism
2. Tubular fluid
3. Interstitium

- 4320 molecules of bicarbonate can be reabsorbed by only one proton (H^+), there is no net secretion of hydrogen ions so far. (H+ recycle again and again)

(Net secretion of H+ means bicarbonate gain)

- After complete HCO_3^- reabsorption: any further H^+ secretion is net secretion resulting in:
  - HCO_3^- gain
  - Shift of TF pH below 7.4

- The majority of H^+ secretion occurs in the proximal tubule by Na^+ countertransport mechanism and can cause a concentration difference across the cell membrane up to 5-6 times only: but a tremendous amount of H+ is secreted (95%)….pH of TF at this site is around 6.5 only

•Primary Active Secretion Of H+ in Intercalated Cells (brown cells) of Late Distal Tubules & Collecting Ducts

- In the collecting ducts, we have H^+ pump and the rest of H^+ (5%) is actively secreted and it can increase the concentration difference up to 900 times leading to a very high concentration gradient. pH =4.5 (maximum pH of the urine)

	Reabsorption of HCO3-	Gaining of HCO3-
Proximal parts	**80-85%**	**55 mM**
Distal parts	**15-20%**	**15 mM**

HCO_3^- Gain

→ After absorbing the entire filtered bicarb, we still need additional 80mmol/day. This amount is supplied by the kidneys.

The presence of TF buffers allow us to secrete H+ and make new HCO_3^-: Main TF buffers are phosphate $HPO_4^=$ which blinds H^+ to form $H_2PO_4^-$ → → →

• Most diuretics except carbonic anhydrase inhibitors induce alkalosis be washing H+ from urine, maintaining H+ gradient which lead to continuous secretion and removal of H+.

•Carbonic anhydrase inhibitors: inhibit H+ secretion and thus decrease bicarb production → → → acidosis.

Do we have 80 mMole of phosphate to be excrted in the urine? If yes, then we can gain 80 mMole/D of HCO_3^- → → → The problem is solved!!! We don't worry about the 80 mMole of fixed acids anymore.

Unfortunately, we excrete only 20-30 mMole of phosphate

We still need additional 50-60 mMole of new HCO_3^- through other source→ By ammonium production

Ammonium production

Don't confuse ammonium $NH4^+$ with ammonia NH_3 ,. ammonium is an ion; ammonia is not).

Glutamine from blood enter the proximal cells where it is converted to glutamate then to alpha keto-glutamate which forms 2 NH_4^+ +2HCO_3^-

- Ammonia is secreted into the lumen by counter-transport mechanism in exchange with sodium in proximal tubules, thick ascending loop of Henley and distal tubules.
- In collecting tubules:

H^+ is secreted into the lumen where it combines with NH_3(ammonia) to form NH_4^+ (ammonium).

→ Collecting tubules membrane is much less permeable for ammonium than ammonia, thus NH_4^+ is trapped in the lumen , this is called → ammonia trapping.

- Ammonium production can be induced unlike phosphate buffer system which is fixed.
- Whenever a hydrogen ion secreted into the tubular lumen combines with a buffer other than bicarbonate, the net effect is the addition of new bicarbonate ion to the blood.

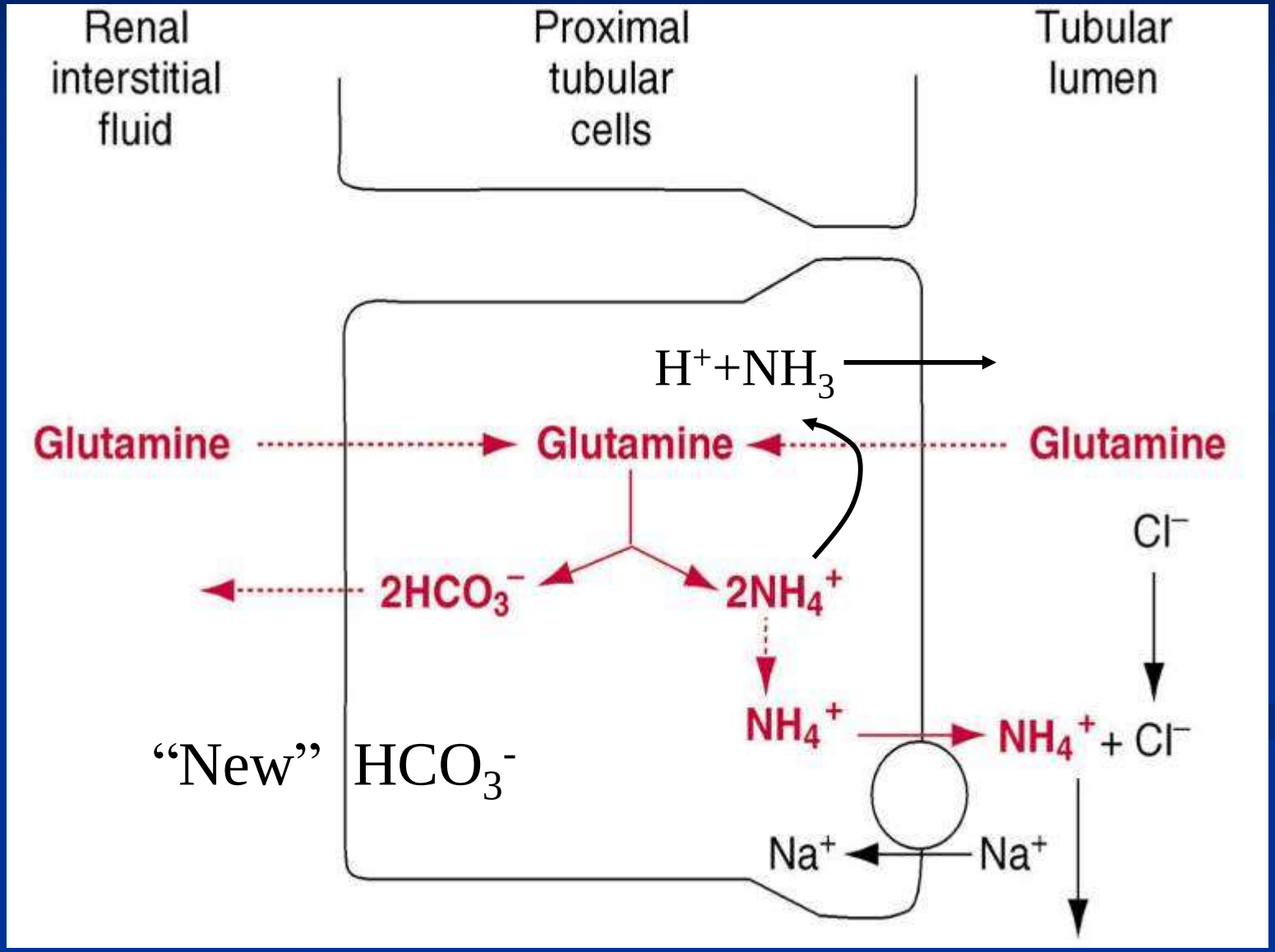
- Low blood pH induces glutaminase enzyme to produce HCO_3^- and NH_4^+, so the urine will be full of ammonium which is secreted in the from of NH_4Cl

-"The kidney can make up to 500 mMole of NH4/D"

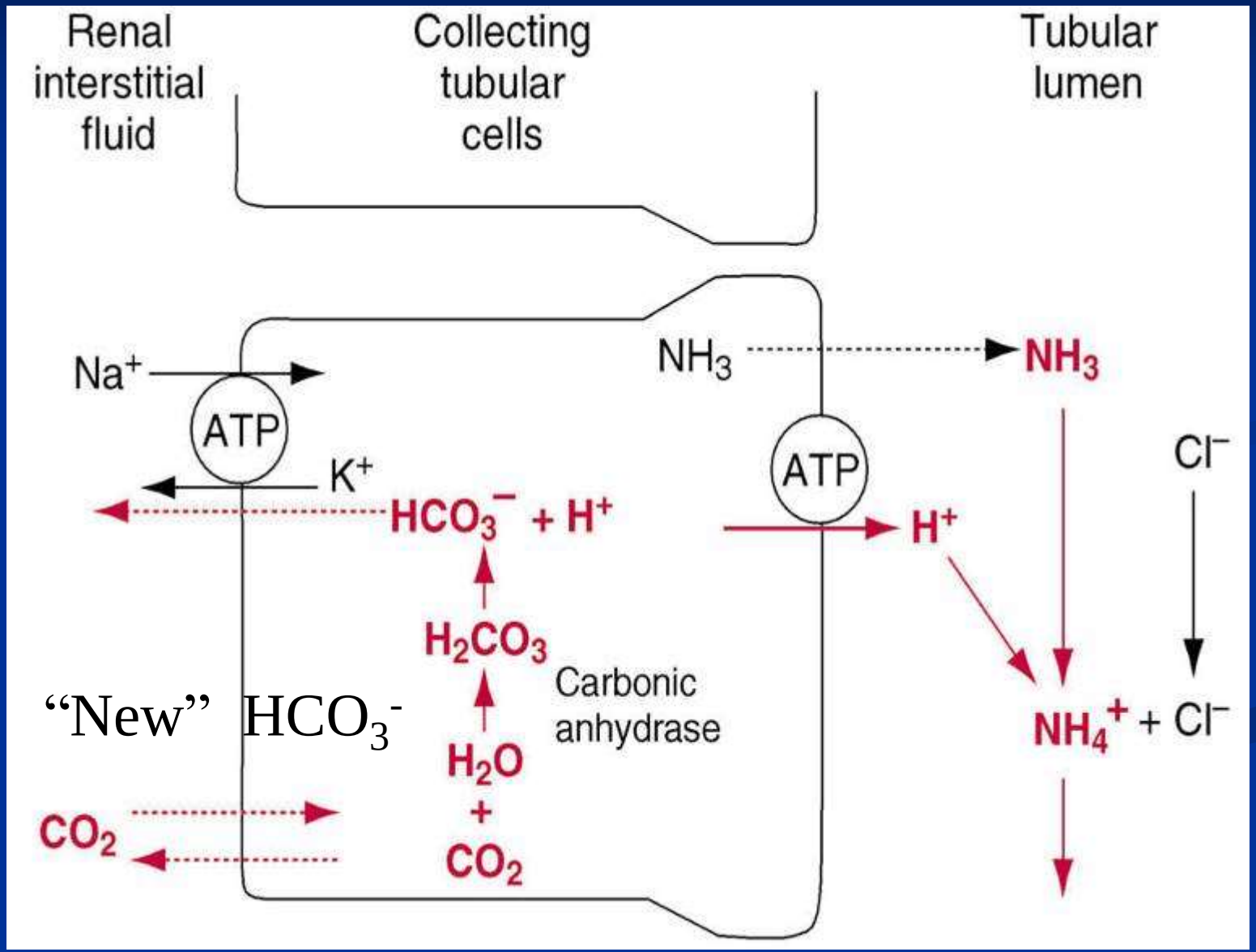
- If the kidney cannot absorb HCO3- or cannot secrete H+ then there is acidosis, this acidosis is called: <u>renal tubular acidosis</u>

- HCO3- added /day = NH_4Cl excretion + titratable acids - HCO3-

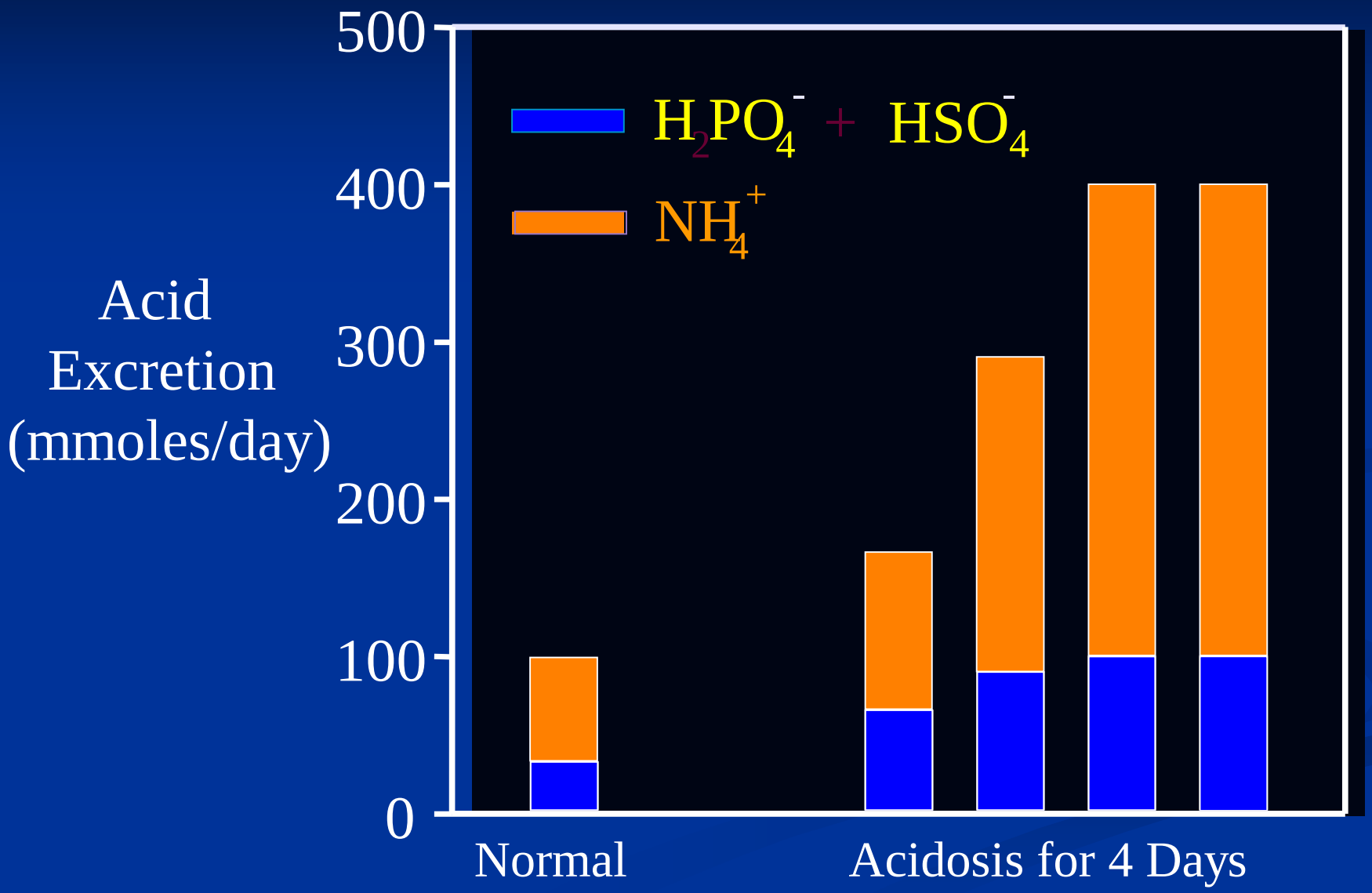
Production and secretion of NH_4^+ and HCO_3^- by proximal, thick loop of Henle, and distal tubules



Buffering of hydrogen ion secretion by ammonia (NH_3) in the collecting tubules.



Phosphate and Ammonium Buffering In Chronic Acidosis



Quantification of Normal Renal Acid-Base Regulation

Total H^+ secretion = 4400 mmol/day

= HCO_3^- reabsorption (4320 mmol/d)

+ titratable acid ($NaHPO_4^-$) (30 mmol/d)

+ NH_4^+ excretion (50 mmol/d)

Net H^+ excretion = 79 mmol/day

= titratable acid (30 mmol/d)

+ NH_4^+ excretion (50 mmol/d)

- HCO_3^- excretion (1 mmol/d)

Normal Renal Acid-Base Regulation

Net addition of HCO_3^- to body
(i.e. net loss of H^+)

Titratable acid	= 30 mmol/day
+ NH_4^+ excretion	= 50 mmol/day
- HCO_3^- excretion	= 1 mmol/day
Total	= 79 mmol/day

Acid-Base Imbalances

- **Acidosis**
  - A condition in which the blood has too much acid (or too little base), resulting in a decrease in blood pH (< 7.35)
- **Alkalosis**
  - A condition in which the blood has too much base (or too little acid), resulting in an increase in blood pH (> 7.45)

HCO_3^-/H^+ ratio in extracellular fluids

- Acidosis ↓ in the ratio
  - Due to a fall in HCO3 (*metabolic acidosis)*
  - Due to an increase in PCO2 (*respiratory acidosis)*
- Alkalosis ↑ in the ratio
  - Due to an increase in HCO3 (*metabolic alkalosis)*
  - Due to a fall in PCO2 (*respiratory alkalosis)*

Classification of Acid-Base Disorders from plasma pH, PCO_2, and HCO_3^-

$$H_2O + CO_2 \longleftrightarrow H_2CO_3 \longleftrightarrow H^+ + HCO_3^-$$

$$pH = pK + \log \frac{HCO_3^-}{\alpha\ PCO_2}$$

Acidosis : pH < 7.4

- metabolic : ↓ HCO_3^-
- respiratory : ↑ PCO_2

Alkalosis : pH > 7.4

- metabolic : ↑ HCO_3^-
- respiratory : ↓ PCO_2

pH disturbances:

- Acidosis is more common than alkalosis.
- metabolic acidosis is more common than respiratory acidosis.

Main cause of metabolic acidosis is not diabetes mellitus because diabetes mellitus type 1 that causes ketoacidosis is not common.

→ The most common cause of M.acidosis is diarrhea.

* Diarrhea treatment: rehydration, correct electrolyte and blood pH

pH disturbance

Metabolic → it is the HCO3- shift

Respiratory → lit is the PCO2 shift

	pH	P_aCO_2	HCO_3^-
M. Acidosis	↓	↓	↓
M. Alkalosis	↑	↑	↑
R. Acidosis	↓	↑	↑
R. alkalosis	↑	↓	↓

To know the type of disorder:

First we look for pH if it increased or decrease, second we look for the cause: is it the HCO3- (metabolic) or the CO2 (respiratory). Third we look for compensation.

Classification of Acid-Base Disturbances

	Plasma			
Disturbance	pH	HCO_3^-	pCO_2	Compensation
metabolic acidosis	↓	↓	↓	ventilation → ↓PCO2 renal HCO_3 production
respiratory acidosis	↓	↑	↑	renal HCO_3 production
metabolic alkalosis	↑	↑	↑	ventilation renal HCO_3 excretion
respiratory alkalosis	↑	↓	↓	renal HCO_3 excretion

Metabolic acidosis:

Metabolic Acidosis:

Non-respiratory acidosis is better term, but metabolic acidosis is most commonly used.

1. Renal tubular acidosis

2. ↑ HCO3- loss: diarrhea is the most common cause of M. acidosis, another cause is deep vomiting.

3. ↑ H+ production: as in D.M,

4. also ingestion of Aspirin or when acetoacetic acids are produced from fats.

→ Acidosis stimulate respiratory center causing hyperventilation, decreasing $PaCO_2$ as a compensation.

- **Acute metabolic acidosis (not for long period of time) is not accompanied with respiratory compensation.**

* **Respiratory compensation starts to act after minutes, full effect after hours.**

Metabolic acidosis

- Metabolic Acidosis : ↓ (HCO_3^- / PCO_2 in plasma)
  (↓ pH, ↓ HCO_3^-)

  - aspirin poisoning (↑ H^+ intake)
  - diabetes mellitus (↑ H^+ production)
  - diarrhea (↑ HCO_3^- loss)
  - renal tubular acidosis (↓ H^+ secretion, ↓ HCO_3^- reabs.)
  - carbonic anhydrase inhibitors (↓ H^+ secretion)

Anion Gap as a Diagnostic Tool.

In body fluids: total cations = total anions

Cations (mEq/L)		Anions (mEq/L)	
Na^+	(142)	Cl^-	(108)
		HCO_3^-	(24)
		Unmeasured	
K^+	(4)	Proteins	(17)
Ca^{++}	(2-3)	Phosphate,	
Mg^{++}	(2)	Sulfate,	
		lactate, etc	(4)
Total	(153)		(153)

Anion Gap

- Definition: Difference between calculated serum cations and anions
- The difference between $[Na^+]$ and the sum of $[HCO_3^-]$ and $[Cl^-]$.
  - **$[Na^+] - ([HC0_3^-] + [Cl^-]) =$**
    - 142 - (24 + 108) = 10
      - Normal = 12 $\pm$ 2
      - Range 8 - 16
- Clinicians use the anion gap to identify the cause of metabolic acidosis.

- Anion gap in metabolic acidosis:

We test electrolyte and ABG (arterial blood gases)

- <u>ABG measures:</u>

  PO2

  PCO2

  Hemoglobin saturation

  HCO3-

  pH

•What ever the disturbance in electrolyte the plasma will remain electro-neutral, that means anions = cations.

- Acetoacetic acid is not routinely measured, but Na+, K+, Cl-, HCO3- are measured

Anion GAP : $(Na^+) = (Cl^- + HCO_3^-) + (8\text{-}16$ mM as unmeasured anions or the so called anion gap).

If cation >>> anions :like in diabetes Mellitus there are excess unmeasured anions (α-ketegluteric acid, acetacetats…we don't measure them).

If anion gab> 16 then it is increased like in cases of ketoacidosis.

- Ketocidosis

→ Cl- and HCO3- are decreased in concentration so the anion gap is increased.

<u>Diarrhea</u> :

In this state we lose $NaHCO_3$

So decreased Na+ and HCO3- and therefore normal anion gap.

Anion Gap as a Diagnostic Tool

One more time: In body fluids:
total cations = total anions
$Na^+ = Cl^- + HCO_3^- +$ unmeasured anions

unmeasured anions = $Na^+ - Cl^- - HCO_3^-$ = anion gap

= 142 - 108 - 24 = 10 mEq/L

Anion Gap in Metabolic Acidosis

- loss of HCO_3^- and normal anion gap

anion gap = Na^+ - ↑Cl^- - ↓HCO_3^-

hyperchloremic metabolic acidosis

↑ anion gap = Na^+ – Cl^- – ↓HCO_3^-

normochloremic metabolic acidosis

i.e. diabetic ketoacidosis, lactic acidosis, salicylic acid poisoning, etc.

Use of "Anion Gap" as a Diagnostic Tool for Metabolic Acidosis

Increased Anion Gap (normal Cl^-)

- diabetes mellitus (ketoacidosis)
- lactic acidosis
- aspirin (acetysalicylic acid) poisoning
- methanol poisoning
- starvation
- Renal Failure

Normal Anion Gap (increased Cl^-, hyperchloremia)

- diarrhea
- renal tubular acidosis
- Addison' disease
- carbonic anhydrase inhibitors

The most common causes of increased Anion Gap in Metabolic Acidosis

- Salicylates raise the gap to 20.
- Renal failure raises gap to 25.
- Diabetic ketoacidosis raises the gap to 35-40.
- Lactic acidosis raises the gap to > 50.
- Largest gaps are caused by ketoacidosis and lactic acidosis.

Laboratory values for an uncontrolled diabetic patient include the following:

arterial pH = 7.25

Plasma HCO_3^- = 12

Plasma PCO_2 = 28

Plasma Cl^- = 102

Plasma Na^+ = 142

Metabolic Acidosis with Respiratory Compensation

What type of acid-base disorder does this patient have?

What is his anion gap ?

Anion gap = 142 - 102 -12 = 28

Answer

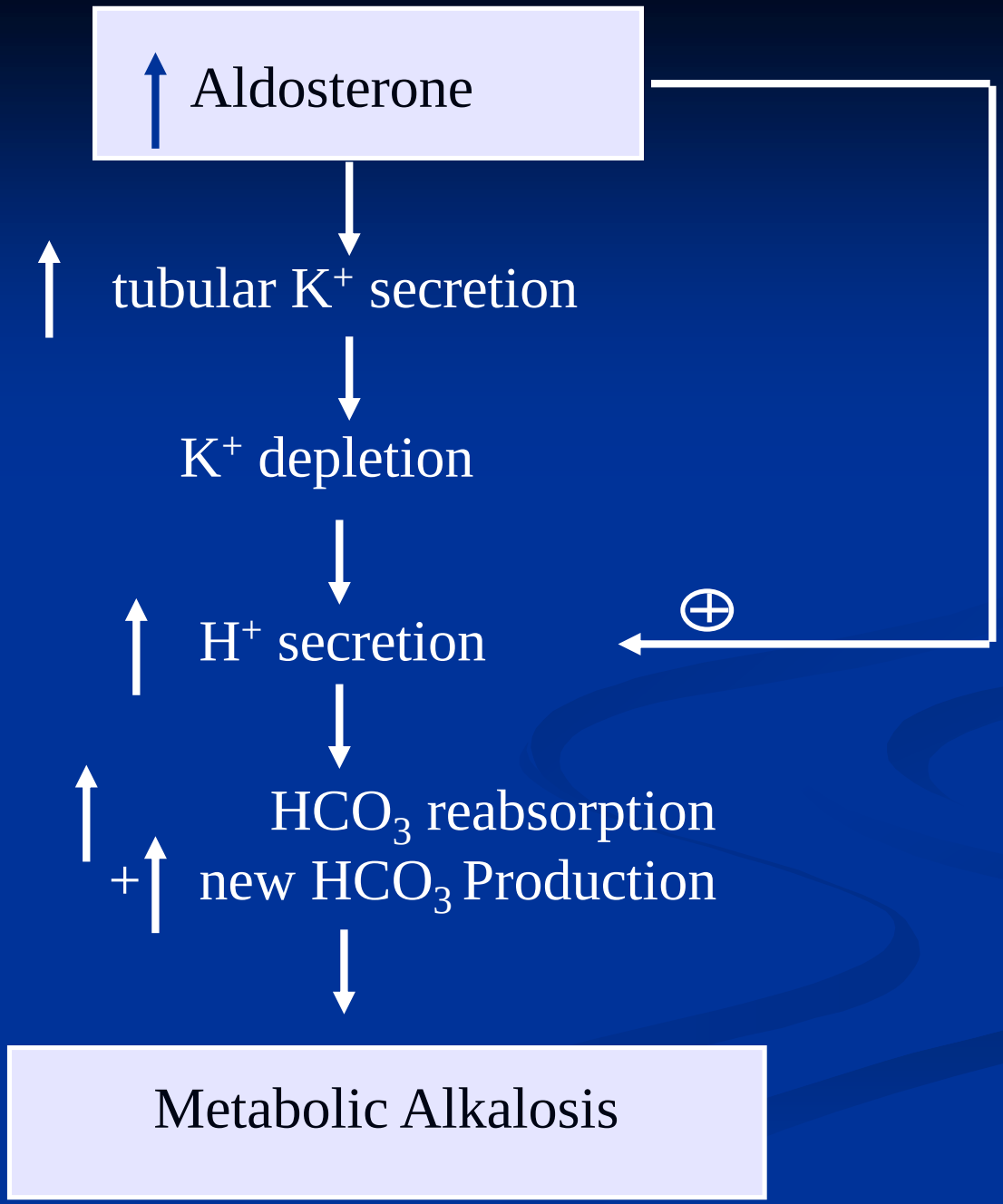
A. diarrhea

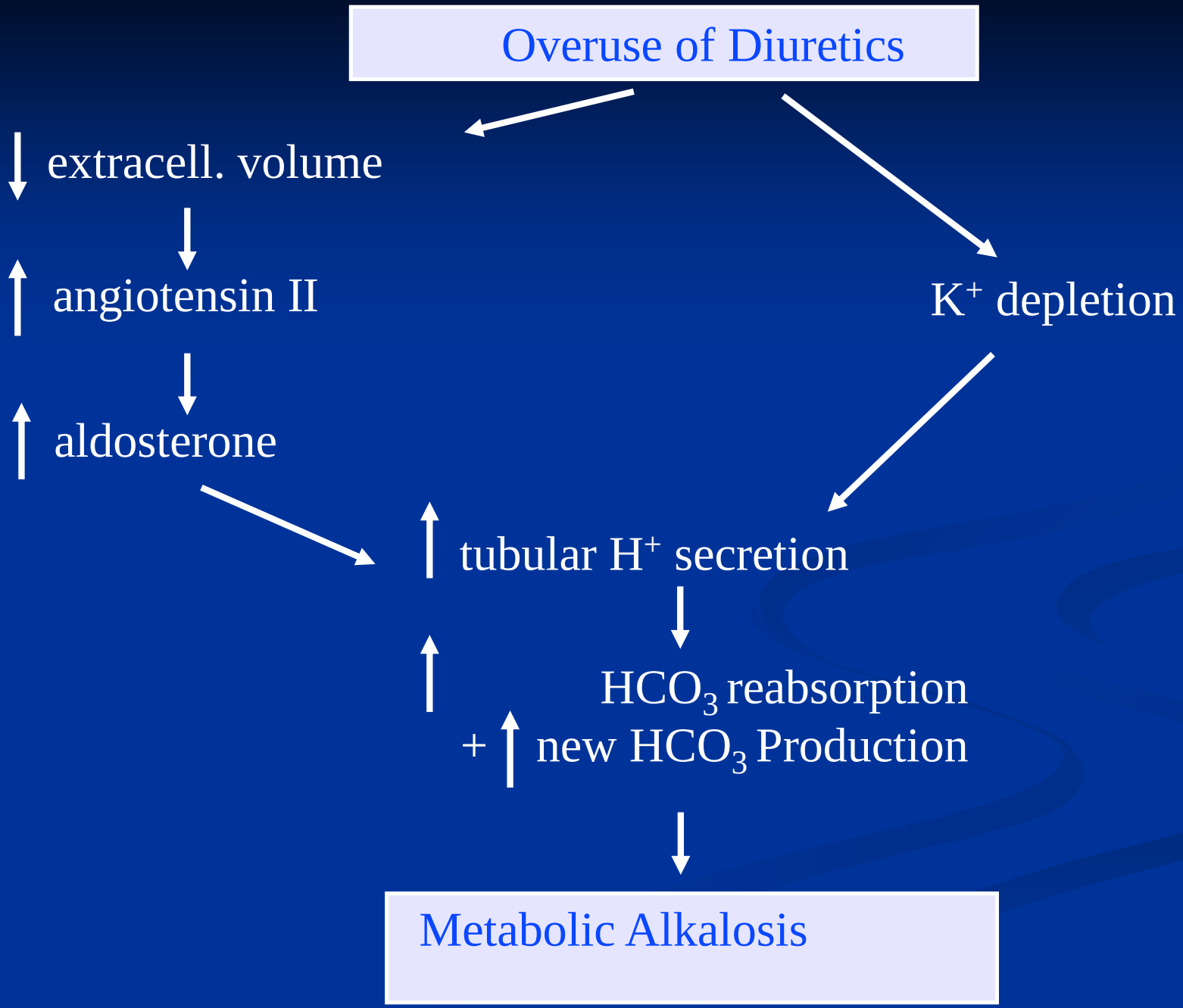
B. diabetes mellitus

C. Renal tubular acidosis

D. primary aldosteronism

- **Metabolic Alkalosis:** "not common"
- Metabolic Alkalosis : ↑ (HCO_3^- / PCO_2 in plasma)
  (↑ pH, ↑ HCO_3^-)
- 1. Diuretics with the exception of C.A inhibitors :↑ flow → ↑ TF flow rate → ↑ H+ secretion.
- 2. ↑ aldostrerone.
- 3. Vomiting of gastric content only (Pyloric stenosis)
- 4. Administration of $NaHCO_3$.


↑ Aldosterone
↑ tubular K^+ secretion
K^+ depletion
↑ H^+ secretion
⊕
↑ HCO_3 reabsorption
+ ↑ new HCO_3 Production
Metabolic Alkalosis


Overuse of Diuretics
↓ extracell. volume
↓
↑ angiotensin II
↓
↑ aldosterone
K+ depletion
↑ tubular H+ secretion
↓
↑ HCO3 reabsorption
+ ↑ new HCO3 Production
↓
Metabolic Alkalosis

Respiratory acidosis

- Respiratory Acidosis : ↓ (HCO_3^- / PCO_2 in plasma)
  (↓ pH, ↑ PCO_2)

  - brain damage
  - pneumonia
  - emphysema
  - other lung disorders

Respiratory acidosis

Respiratory here does not mean the lung: it means CO_2 → (CO_2 is the cause like in dialysis).

Causes:

1. Gas exchange (↓ Ability of the lung to eliminate CO_2 such as): pneumomia , lack of lung tissue, airway obstruction, ↓ surface area.

2. CNS Damage to the respiratory CNTR. trauma , tumors.

3. Respiratory muscles: phrenic paralysis, diaphragmatic fatigue

Respiratory Alkalosis

- - Psychoneurosis. psychic (fear, pain, etc)
- - high altitude

Renal Compensations for Acid-Base Disorders

- Acidosis:
  - increased H^+ secretion
  - increased HCO_3^- reabsorption
  - production of new HCO_3^-
- Alkalosis:
  - decreased H^+ secretion
  - decreased HCO_3^- reabsorption
  - loss of HCO_3^- in urine

Renal Compensation for Acidosis

Increased addition of HCO_3^- to body by kidneys
(increased H^+ loss by kidneys)

Titratable acid	= 35 mmol/day (small increase)
NH_4^+ excretion	= 165 mmol/day (increased)
HCO_3^- excretion	= 0 mmol/day (decreased)
Total	= 200 mmol/day

This can increase to as high as 500 mmol/day

Renal Compensation for Alkalosis

Net loss of HCO_3^- from body
(i.e. decreased H^+ loss by kidneys)

Titratable acid	=	0 mmol/day (decreased)
NH_4^+ excretion	=	0 mmol/day (decreased)
HCO_3^- excretion	=	80 mmol/day (increased)
Total	=	80 mmol/day

HCO_3^- excretion can increase markedly in alkalosis

Renal Responses to Respiratory Acidosis

$H_2O + CO_2 \longleftrightarrow H_2CO_3 \longleftrightarrow H^+ + HCO_3^-$

Respiratory acidosis : ↓ pH ↑ PCO_2 ↑ HCO_3^-

↑ PCO_2 → ↑ H^+ secretion → complete HCO_3^- reabs.
+
excess tubular H^+

↓ pH

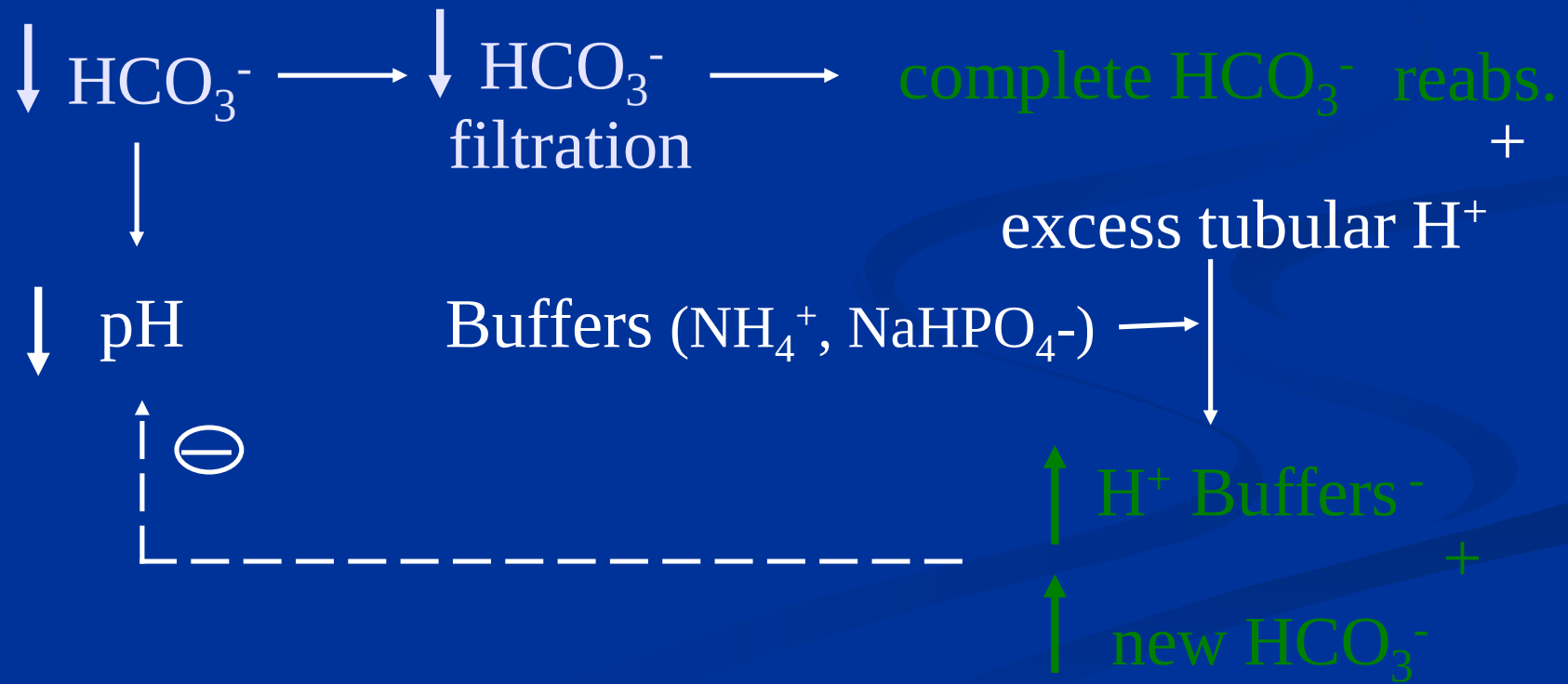
Buffers (NH_4^+, $NaHPO_4^-$) →

↑ H^+ Buffers$^-$
+
new HCO_3^-

⊖

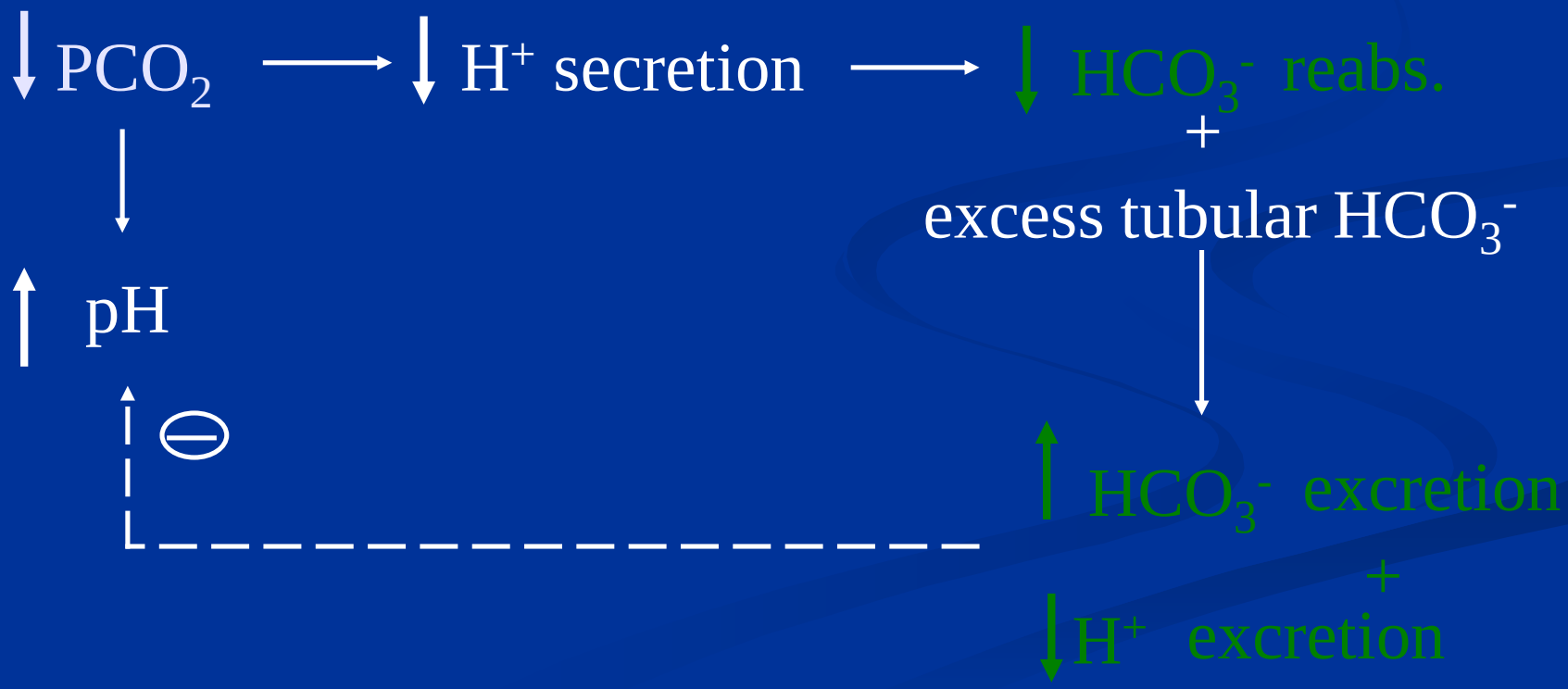
Renal Responses to Metabolic Acidosis

Metabolic acidosis : ↓ pH ↓ pCO_2 ↓ HCO_3^-



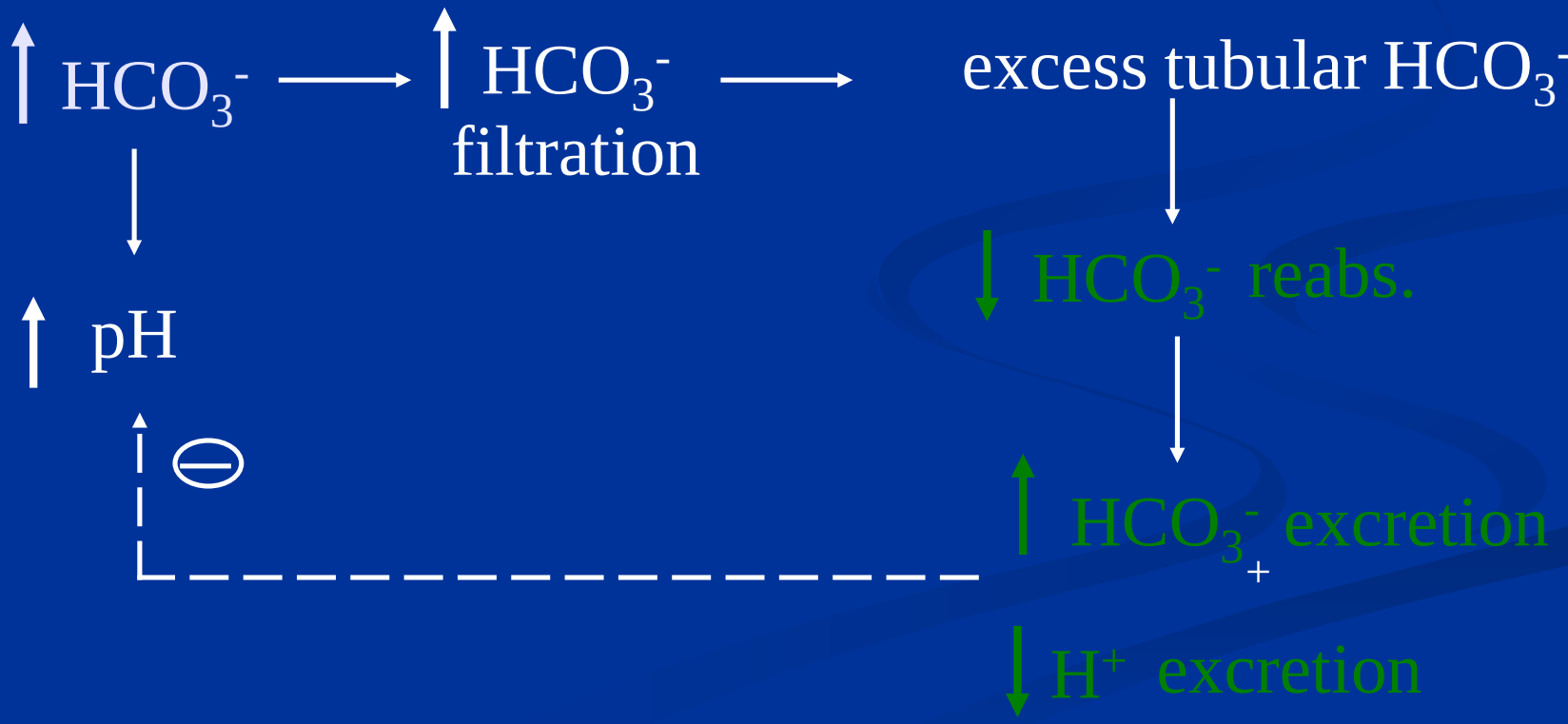
Renal Responses to Respiratory Alkalosis

Respiratory alkalosis : ↑ pH ↓ pCO_2 ↓ HCO_3^-



Renal Responses to Metabolic Alkalosis

Metabolic alkalosis : ↑ pH ↑ PCO_2 ↑ HCO_3^-



- Simple Versus Mixed `Acid-Base Imbalance:
- - Mixed (complex) disorder (either term can be used).
- *M. Acidosis For every ↓ 1 mEq HCO3- → 1.2 mm Hg PCO2 ↓ too.
- **M. Alkalosis For every 1 mEq↑ in HCO3-→ 0.7 mmHg ↑ in PCO2
- ***R. Acidosis
- Acute: For every 10 mmHg ↑ in PCO2 → 1 mEq ↑ in HCO3-
- Chronic For every 10 mmHg ↑ in PCO2 →3.5 mEq↑ in HCO3-
- ****R. Alkalosis
- Acute For every 10 mmHg ↓ PCO2 → 2 mEq ↓ HCO3-
- Chronic For every 10 mmHg ↓ PCO2 → 5 mEq ↓ HCO3-

- * if PCO2 ↓ more than expected→ superimposed R. alkalosis too.
- * if PCO2 ↓ less than expected→ superimposed R. acidosis too.
- ** if PCO2 ↑ more than expected→ superimposed R. acidosis too.
- ** if PCO2 ↑ less than expected→ superimposed R. alkalosis too.
- *** if HCO3 ↑ more than expected→ superimposed M. alkalosis too.
- *** if HCO3 ↑ less than expected→ superimposed M. acidosis too.
- **** if HCO3 ↓ more than expected→ superimposed M. acidosis too.
- **** if HCO3 ↓ less than expected→ superimposed M. alkalosis too.
- *** In metabolic acidosis respiratory system compensate more than metabolic alkalosis because acidosis induce hyperventilation while alkalosis induce hypoventilation which may be opposed by hypoxia

Question

The following data were taken from a patient:

urine volume = 1.0 liter/day
urine HCO_3^- concentration = 2 mmol/liter
urine NH_4^+ concentration = 15 mmol/liter
urine titratable acid = 10 mmol/liter

- What is the daily net acid excretion in this patient ?
- What is the daily net rate of HCO_3^- addition to the
- extracellular fluids ?

Answer

The following data were taken from a patient:

urine volume = 1.0 liter/day

urine HCO_3^- concentration = 2 mmol/liter

urine NH_4^+ concentration = 15 mmol/liter

urine titratable acid = 10 mmol/liter

net acid excretion = Titr. Acid + NH_4^+ excret - HCO_3^-

= (10 x 1) + (15 x 1) - (1 x 2)

= 23 mmol/day

net rate of HCO_3^- addition to body = 23 mmol/day

Question

A plasma sample revealed the following values in a patient:

pH = 7.12

PCO_2 = 50

HCO_3^- = 18

diagnose this patient's acid-base status :

acidotic or alkalotic ? Acidotic

respiratory, metabolic, or both ? Both

Mixed acidosis: metabolic and respiratory acidosis

Mixed Acid-Base Disturbances

Two or more underlying causes of acid-base disorder.

pH= 7.60
PCO_2 = 30 mmHg
plasma HCO_3^- = 29 mmol/L

What is the diagnosis?

Mixed Alkalosis

- Metabolic alkalosis : increased HCO_3^-
- Respiratory alkalosis : decreased pCO_2

Question

A patient presents in the emergency room and the following data are obtained from the clinical labs:

plasma pH= 7.15, HCO_3^- = 8 mmol/L, PCO_2= 24 mmHg

This patient is in a state of:

1. metabolic alkalosis with partial respiratory compensation
2. respiratory alkalosis with partial renal compensation
3. **metabolic acidosis with partial respiratory compensati**
4. respiratory acidosis with partial renal compensation

Laboratory values for a patient include the following:

arterial pH = 7.34
Plasma HCO_3^- = 15
Plasma PCO_2 = 29
Plasma Cl^- = 118
Plasma Na^+ = 142

Metabolic Acidosis with Respiratory Compensation

What type of acid-base disorder does this patient have?
What is his anion gap ?

Anion gap = 142 - 118 - 15 = 9 (normal)

Which of the following are the most likely causes of his acid-base disorder?

a. diarrhea

b. diabetes mellitus

c. aspirin poisoning

d. primary aldosteronism

Indicate the Acid -Base Disorders in Each of the Following Patients

pH	HCO_3^-	PCO_2	Acid-Base Disorder ?
7.34	15	29	Metabolic acidosis
7.49	35	48	Metabolic alkalosis
7.34	31	60	Respiratory acidosis
7.62	20	20	Respiratory alkalosis
7.09	15	50	Acidosis: respiratory + metabolic

Steps to an Arterial Blood Gas Interpretation…how to diagnose?

Step One

Assess the pH to determine if the blood is within normal range, alkalotic or acidotic. If it is:

above 7.45, the blood is alkalotic.

If it is below 7.35, the blood is acidotic.

Step Two

If the blood is alkalotic or acidotic, we now need to determine if it is caused primarily by a respiratory or metabolic problem. To do this, assess the **$PaCO_2$** level. Remember that with a respiratory problem, as the pH decreases below 7.35, the PaCO2 should rise. If the pH rises above 7.45, the PaCO2 should fall. Compare the pH and the PaCO2 values. If pH and PaCO2 are indeed moving in *opposite directions*, then the problem is primarily respiratory in nature.

Step Three

Finally, assess the HCO_3 value. Recall that with a metabolic problem, normally as the pH

increases, the HCO3 should also increase. Likewise, as the pH decreases, so should the HCO3.

Compare the two values. If they are moving *in the same direction*, then the problem is primarily metabolic in nature. The following chart summarizes the relationships between pH, PaCO2 and HCO3.

	pH	PaCO2	HCO3
Respiratory Acidosis	↓	↑	normal
Respiratory Alkalosis	↑	↓	normal
Metabolic Acidosis	↓	normal	↓
Metabolic Alkalosis	↑	normal	↑

Example

Suha is a 45-year-old female admitted to the E.R with a severe asthma attack. She has been experiencing increasing shortness of breath since admission three hours ago. Her arterial blood gas result is as follows:

PATIENT: Maha

DATE: 5/4/2016 1:43 am

pH = 7.22

$PaCO_2$ = 55

HCO3- = 25

Follow the steps:

1. Assess the pH. It is low (normal 7.35-7.45); therefore, we have acidosis.

2. Assess the PaCO2. It is high (normal 35-45) and in the opposite direction of the pH.

3. Assess the HCO3. It has remained within the normal range (22-26).

	pH	PCO2	HCO3
Respiratory Acidosis	↓	↑	Normal

Acidosis is present (decreased pH) with the $PaCO_2$ being increased, reflecting a primary *respiratory* problem.
For this patient, we need to improve the ventilation status by providing oxygen therapy, mechanical ventilation, administering bronchodilators, etc

Example 2

Maher is a 55-year-old male admitted to E.R with a recurring bowel obstruction. He has been experiencing intractable vomiting for the last several hours despite the use of antiemetics. Here is his arterial blood gas result:

PATIENT: Maher

DATE: 5/4/2016 02:30

pH = 7.50

PaCO2 = 42

HCO3- = 33

1. Assess the pH.=7.5 It is high (normal 7.35-7.45), therefore, indicating alkalosis.
2. Assess the PaCO2.=42 It is within the normal range (normal 35-45).
3. Assess the HCO3=33. It is high (normal 22-26) and moving in the same direction as the pH.

	pH	PCO2	HCO_3^-
Metabolic Alkalosis	↑	normal	↑

Alkalosis is present (increased pH) with the HCO_3^- increased, reflecting a primary *metabolic* problem.

Treatment of this patient might include the administration of I.V. fluids and measures to reduce the excess base.

Compensation

When a patient develops an acid-base imbalance, the body attempts to compensate. That the lungs and the kidneys are the primary buffer response systems in the body.

The body tries to overcome either a respiratory or metabolic dysfunction in an attempt to return the pH into the normal range.

A patient can be uncompensated, partially compensated, or fully compensated. When an acid-base disorder is either uncompensated or partially compensated, the pH remains outside the normal range.

In fully compensated states, the pH has returned to within the normal range,

although the other values may still be abnormal.

Be aware that neither system has the ability to overcompensate.

In our first two examples, the patients were uncompensated. In both cases, the pH was outside of the normal range, the primary source of the acid-base imbalance was readily identified, but the compensatory buffering system values remained in the normal range.

partial compensation, review the following three steps:

1. Assess the pH. This step remains the same and allows us to determine if an acidotic or alkalotic state exists.

2. Assess the PaCO2. In an uncompensated state, we have already seen that the pH and PaCO2 move in opposite directions when indicating that the primary problem is respiratory. But

what if the pH and PaCO2 are moving *in the same direction*?

We would then conclude that the primary problem was metabolic. In this case, the *decreasing* PaCO2 indicates that the lungs, acting as a buffer response, and are attempting to correct the pH back into its normal range by decreasing the PaCO2 ("blowing off the excess CO2").

3. Assess the HCO_3^-. In our original uncompensated examples, the pH and HCO_3^- move in the same direction, indicating that the primary problem was metabolic. But what if our results show the pH and HCO_3^- moving in opposite directions?

We would conclude that the primary acid-base disorder is respiratory, and that the kidneys, again acting as a buffer response system, are compensating by retaining HCO_3^-.

Fully Compensated States

	pH	PaCO2	HCO3
Respiratory Acidosis	normal, but <7.40	↑	↑
Respiratory Alkalosis	normal, but >7.40	↓	↓
Metabolic Acidosis	normal, but <7.40	↓	↓
Metabolic Alkalosis	normal, but >7.40	↑	↑

Partially Compensated States

	pH	$PaCO_2$	HCO_3
Respiratory Acidosis	↓	↑	↑
Respiratory Alkalosis	↑	↓	↓
Metabolic Acidosis	↓	↓	↓
Metabolic Alkalosis	↑	↑	↑

Example 3

Maher is admitted to the hospital. He is a kidney dialysis patient who has missed his last two appointments at the dialysis center. His arterial blood gas values are reported as follows:

pH= 7.32 (7.35-7.45)

$PaCO_2$= 32 (35-45)

HCO_3^- =18 (22-26)

	pH	PaCO2	HCO_3^-
Metabolic Acidosis	↓	↓	↓

Metabolic problem means low HCO_3^- …….therefore low pH… in compensation … over ventilation & subsequent decrease in $PaCO_2$

Example 4

Maha is a patient with chronic COPD being admitted for surgery. Her admission labwork reveals an arterial blood gas with the following values:

pH =7.35 (7.35-7.45)
PaCO2 =48 (35-45)
HCO_3^- =28 (22-26)

Fully compensated respiratory acidosis

pH	PaCO2	HCO3 -
normal, but <7.40	↑	↑

Factors that may affect results of ABG tests

Hyperventilation (rapid and deep breathing). This can cause lower than usual PaCO2 levels. While this is a symptom of several diseases, hyperventilation can also occur due to pain or anxiety.

Smoking. Tobacco smoke contains about 2,000 gases and chemicals. These toxins can interfere with test results.

Carbon monoxide inhalation. Carbon monoxide (CO) is a colorless, odorless, tasteless gas that is produced from the incomplete burning of fuels (e.g., from home furnaces, car engines and indoor barbecues). High CO levels in the body can cause CO poisoning and quickly lead to death. CO levels in the body will also affect an ABG test.

In addition, some types of medications may affect ABG results:

Antacids (especially those containing bicarbonate)

Diuretics.

Antibiotics.

Corticosteroids. Such as *cortisone*.

Patients should tell their physician before the test about any medications they are taking.

THANK YOU