# Basics of Acid Base Balance

Red: very important.
Green: Doctor's notes.
Pink: formulas.
Yellow: numbers.
Gray: notes and explanation.

**Physiology Team 436 – Renal Block Lecture 9**



For further understanding please check our "Extra Notes" file which contains extra explanation from reference books.

# Objectives

- Lecture: 9
- Define: acid and base.
- Explain what is meant by strong and weak acids and bases.
- List and identify the names/formulas for the common strong acids and strong bases.
- To explain the role of Henderson-Hasselbalch equation in acid-base regulation.

- Lecture 10 & 11

- To define buffer system and discuss the role of blood buffers and to explain their relevant roles in the body.
- To describe the role of kidneys in the regulation of acid-base balance
- To describe the role of lungs in the regulation of acid-base balance
- To explain the principles of blood gas and acid-base analysis
- To interpret blood gas analysis and diagnose various acid base disorders
- Describe causes of acid base disorders
- Understand use of acid base nomograms

# Acid – Base Balance

- Acid – Base balance ( as known as pH HOMEOSTASIS ) : one of the essential functions of the body, it is concerned with the precise regulation of free (unbound) hydrogen ion concentration in body fluids.
- When discussing acid - base balance, we are normally concerned with regulation of H+ ion balance ( although HCO3- plays a vital role in this balance ).
- To avoid disturbances in [H+], and to maintain its homeostasis :
- the amount generated / taken in MUST EQUAL the amount secreted.

Why is control of $[H^+]$ so important?

1- Highly reactive chemical species (protons).

combine easily with negatively charged ions and bases

2- Most enzymes function optimally at pH ~ 7.4

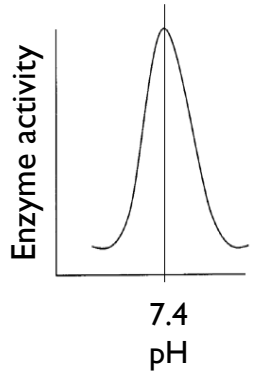
( except gastric enzymes )

So slight deviation in $H^+$ have profound effects on enzyme and protein activity and thus the body's metabolic activity in general.

3- Precise $[H^+]$ regulation is vital because activity of almost all enzyme systems / proteins (inc. ion channels) influenced by pH

(e.g. hydrogen bonding and charge on proteins altered by pH so tertiary structure and function affected.)

4- Acid-base imbalances can cause cardiac arrhythmias and abnormal neuronal excitation due to its affect on $K^+$ levels in the body.




Enzymes of the body can't function properly if the H+ ions balance is disturbed

# Acid – Base Balance
# Definitions ( Bronsted-Lowry )

- **ACIDS :** Molecules containing hydrogen atoms that can release ( Donate ) $H^+$ into solution.
- ( e.g. HCl (hydrochloric aicd) $\Leftrightarrow$ $H^+ + Cl^-$ , $H_2CO_3$ (carbonic acid)).
- **Classified to :**

| Strong acid | Weak acid |
|---|---|
| • all their H+ is dissociated completely in $H_2O$ | • dissociate partially in H2O and are efficient at preventing pH changes |

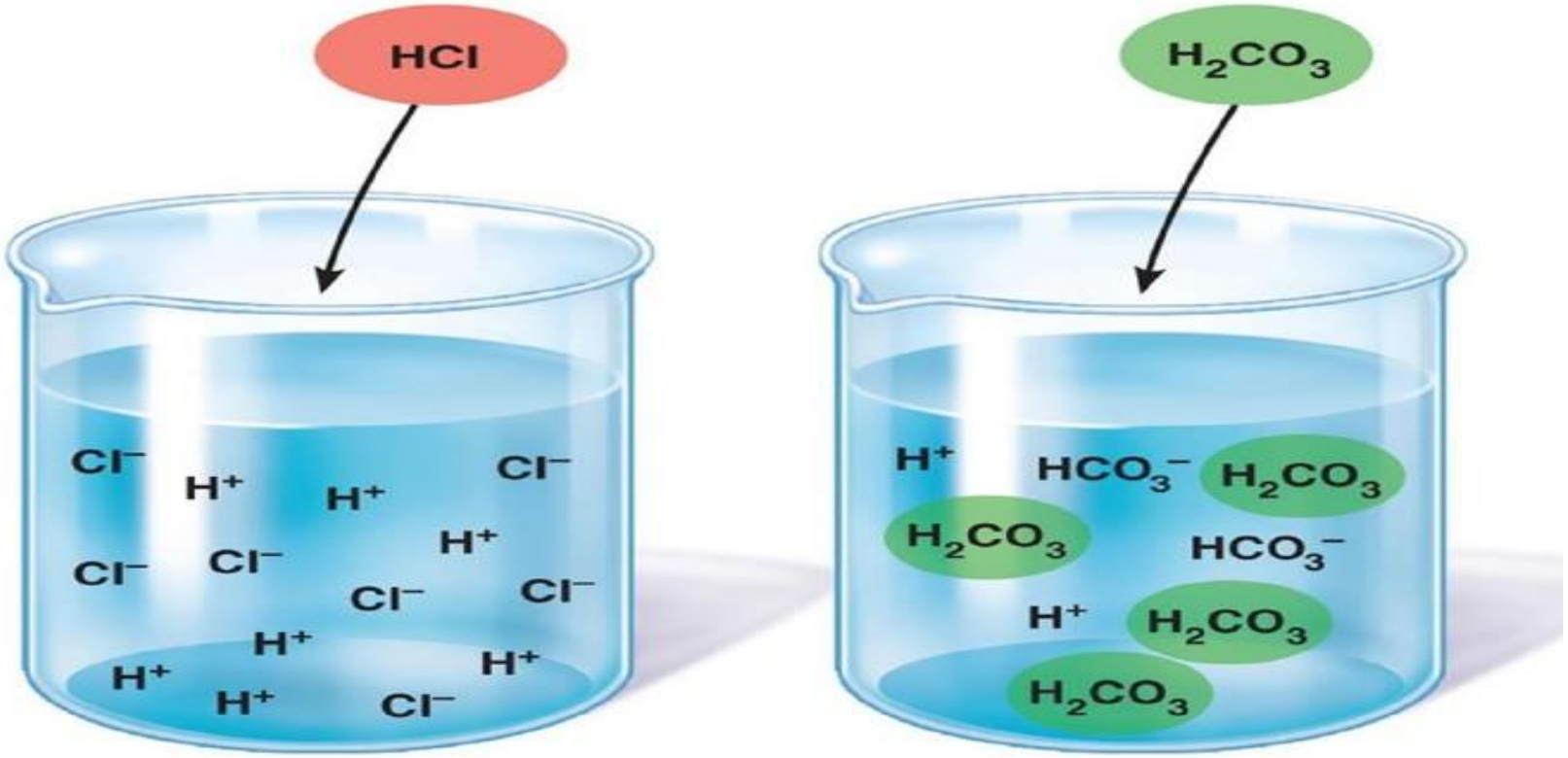
- **BASES (alkalis) :** ions or molecules that can ( Accept ) $H^+$. (e.g., $HCO_3^-$ (Bicarbonate ions), Hydrogen phosphate $HPO_4^{-2}$)
- **Classified to :**

| Weak base | Strong base |
|---|---|
| • dissociate easily in $H_2O$ and quickly bind $H^+$ | • accept $H^+$ more slowly (e.g., $HCO_3^-$ and $NH_3$) |

- HCO3- has negative charge → it can accept hydrogen ion to form carbonic acid.
- Proteins are negatively charged, they accept H+ ions and behave as weak bases.

- **Proteins** in body function as weak bases as some constituent amino acids have net negative charge and attract H+ (e.g. HAEMOGLOBIN).

# Acid – Base Balance
# Definitions ( Bronsted-Lowry )

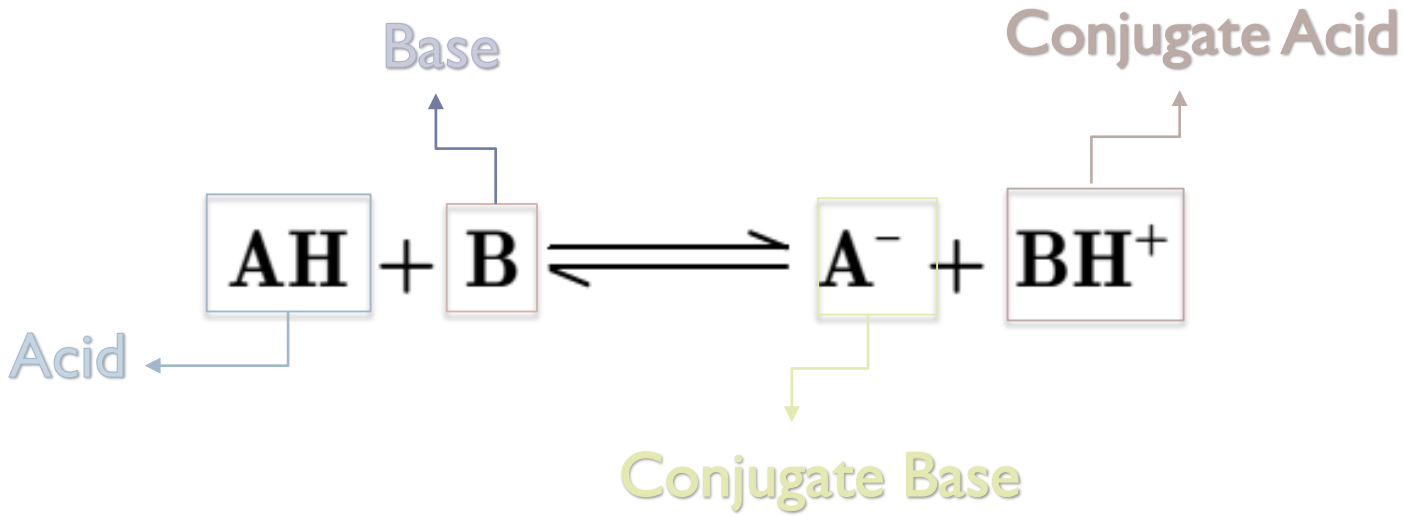



Strong acids dissociate rapidly and release large amounts of $H^+$ in solution.

Weak acids dissociate incompletely and less strongly releasing small amounts of $H^+$ in solution.

**ONLY IN FEMALES' SLIDES**

Free hydrogen ions are extremely unstable. Therefore, for any acid and any base, the equilibrium established is:




Every acid has a conjugate base associated with it, and vice versa.

- A conjugate base : is the particle that is left over after the acid loses its hydrogen ion
- This is general equation but we can replace A with chloride or any other elements so the same equation has to be established.

- The hydronium ion concentration can be found from the pH by the reverse of the mathematical operation employed to find the pH

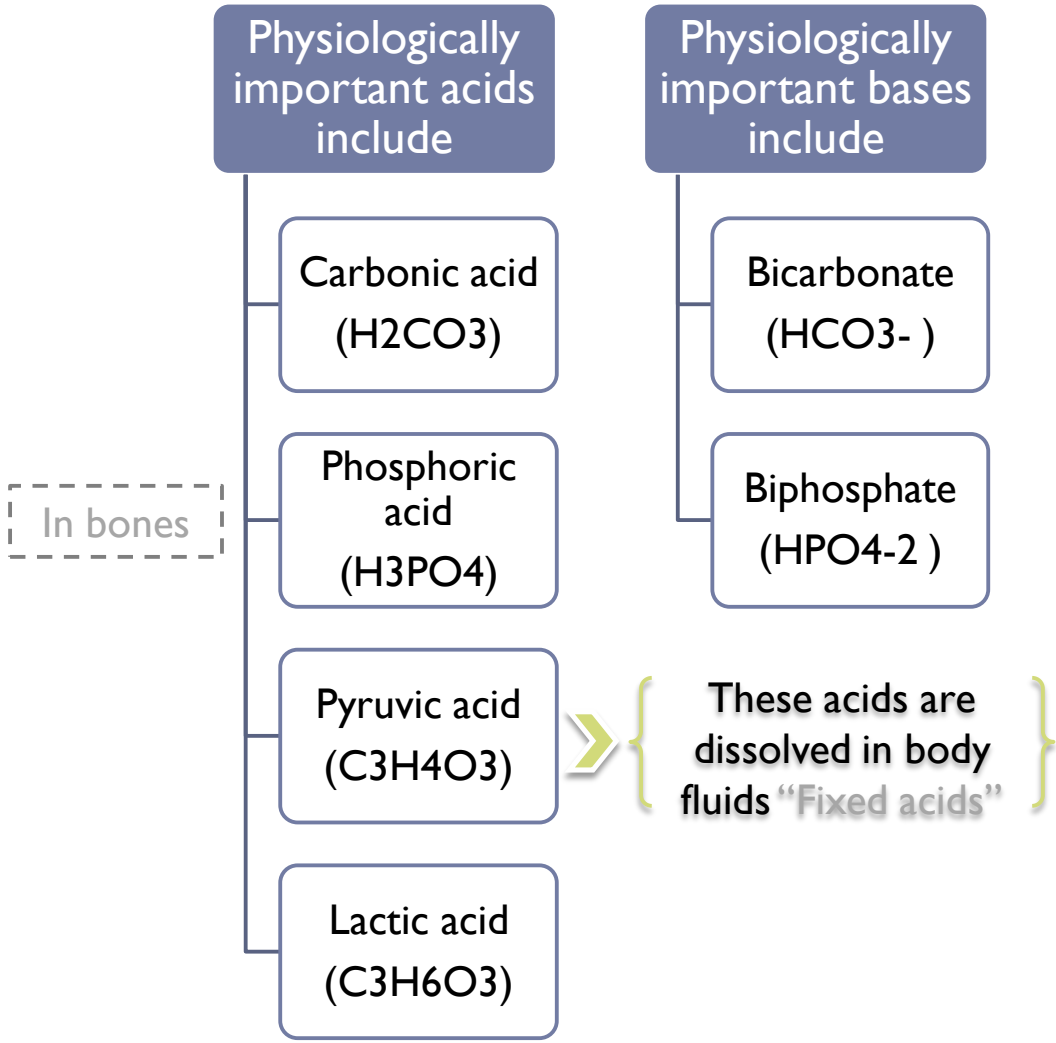
- amphoteric : substances that can act as an acid or as a base.

Water usually is amphoteric compound depend on the added compound (acidic or basic)

- When water behaves as a base, it accepts H+ and forms a hydronium ion; H3O+

When it behaves as an acid, it loses a proton and forms a hydroxide ion; OH-

# Physiologically important Acids and Bases



# pH

- What is pH ?
- The pH of a solution is a measure of the acidity of the solution not the strength of an acid, so it is the $H^+$ ion concentrations. (normal pH= 7.35 – 7.45)
- pH = -log ($H^+$) , When $H^+$ increases > pH decreases and vice versa.
- It is defined as :
- Where [H3O+] is the concentration of hydronium ions in the solution
- The pH of water is 7. This means that a solution of pure water has a $10^{-}7$ mol $dm^{-}3$ of hydronium ions.

**pH depends on two things:**

**The acid in the question**

if we have two equally concentrated solutions of acids, the solution of a strong acid will have a lower pH than that of a weak acid, because it is more fully dissociated and therefore produces more $H_3O^+$ ions. HCl, for example, is completely dissociated.

**The concentration of the solution**

If we have two solutions of the same acid, the more concentrated solution will have more free $H_3O^+$ ions and therefore a lower pH.

For example, if we have 2 concentrations of the same acid (HCL) :
HCl = 5 mEq / HCl = 10 mEq
The HCl with 10 mEq will have lower pH thus it will be more acidic

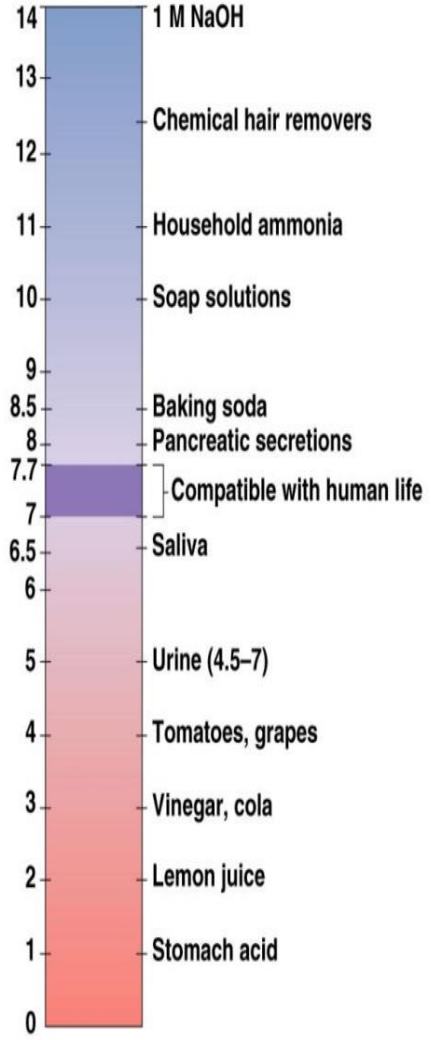
# Acid – Base Balance pH Scale (Sørensen, 1909)

- Relative to other ions, [H+] of body fluids kept VERY LOW
- e.g., ECF [Na+] ≈ 145 mM/L
- ECF [H+] ≈ 0.00004 mM/L (40nM)
- (~ 3.5 million fold difference).
- Because $[H^+]$ so low, easier to express
- $[H^+]$ on a logarithmic scale ⇒ pH units.

$$pH = \log \frac{1}{[H^+]} = - \log [H^+]$$

- Free hydrogen ions in the body are very low that's why we use Logarithm to calculate it.

- Normal pH = -log [0.00000004]
  = 7.4




- **Guyton corner :**

Equally important, the normal variation in H+ concentration in extracellular fluid is only about one millionth as great as the normal variation in sodium ion (Na+) concentration. Thus, the precision with which H+ is regulated emphasizes its importance to the various cell functions.

- **Linda corner:**

When using pH instead of H+ concentration, there are two points of caution. First, because of the minus sign in the logarithmic expression, a mental reversal is necessary: As H+ concentration increases, pH decreases, and conversely. Second, the relationship between H+ concentration and pH is logarithmic, not linear. Thus, equal changes in pH do not reflect equal changes in H+ concentration.

# Acid – Base Balance pH Scale (Sørensen, 1909)

**table 2.5 Hydrogen Ion Concentrations and pH**

| Grams of $H^+$ per Liter | pH | |
|---|---|---|
| 0.00000000000001 | 14 | ↑ Increasingly basic |
| 0.0000000000001 | 13 | |
| 0.000000000001 | 12 | |
| 0.00000000001 | 11 | |
| 0.0000000001 | 10 | |
| 0.000000001 | 9 | |
| 0.00000001 | 8 | Neutral–neither acidic nor basic |
| 0.0000001 | 7 | |
| 0.000001 | 6 | |
| 0.00001 | 5 | |
| 0.0001 | 4 | |
| 0.001 | 3 | Increasingly acidic |
| 0.01 | 2 | |
| 0.1 | 1 | ↓ |
| 1.0 | 0 | |

Note that a change of 1 pH unit = 10x change in [H+] ($log_{10}$ scale)

# Acid – Base Balance pH Scale (Sørensen, 1909)

- pH INVERSELY related to [H+]

as [H+] ↑, pH ↓ – acidosis (below 7.35)

as [H+] ↓, pH ↑– alkalosis (above 7.45)

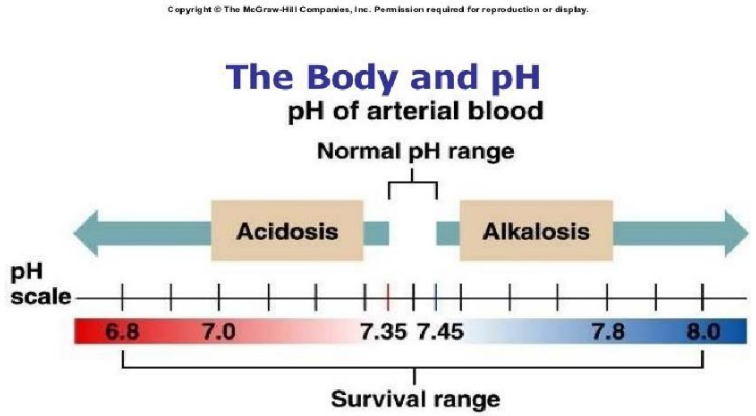
- Normal BLOOD pH

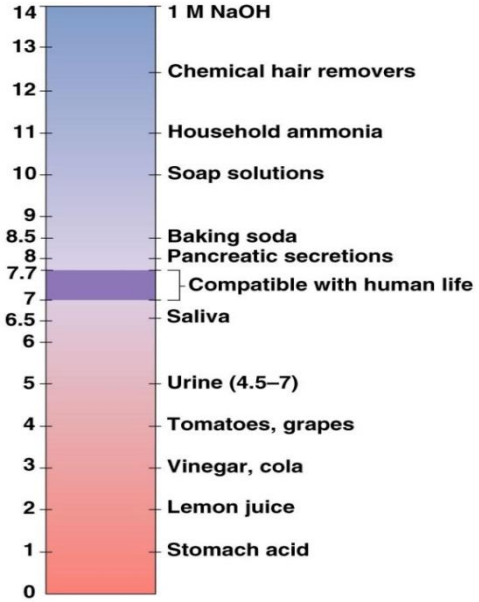
range for adults = 7.35 – 7.45

maintained by chemical buffer systems, kidneys and lungs.

In pH, even the slight change is taken in consideration, as when pH = 7.34 it is acidic ..






- The normal pH is slightly basic which may be because the body does a lot of acidic processes so we are at more risk of acidosis.
- Increasing or decreasing pH (even with a small range) should be fixed immediately to prevent damage.

As when someone takes Aspirin overdose it makes the blood very acidic and the pH becomes below 6.8 causing death if gastric lavage is not performed ..

pH range Compatible with human life = (6.8-7.8).
It likely cause Death: if the pH is higher or less than this range

# Acid – Base Balance pH Scale (Sørensen, 1909)

**Table 31-1** pH and $H^+$ Concentration of Body Fluids

| | $H^+$ Concentration (mEq/L) | pH |
|---|---|---|
| Extracellular fluid | | |
| Arterial blood | $4.0 \times 10^{-5}$ | 7.40 |
| Venous blood | $4.5 \times 10^{-5}$ | 7.35 |
| Interstitial fluid | $4.5 \times 10^{-5}$ | 7.35 |
| Intracellular fluid | $1 \times 10^{-3}$ to $4 \times 10^{-5}$ | 6.0-7.4 |
| Urine | $3 \times 10^{-2}$ to $1 \times 10^{-5}$ | 4.5-8.0 |
| Gastric HCl | 160 | 0.8 |

Approximate pH Values of Various Body Fluids

| Compartment | pH |
|---|---|
| Gastric secretions (under conditions of maximal acidity) | 0.7 |
| Lysosome | 5.5 |
| Chromaffin granule | 5.5 |
| Neutral $H_2O$ at 37°C | 6.81 |
| Cytosol of a typical cell | 7.2 |
| Cerebrospinal fluid | 7.3 |
| Arterial blood plasma | 7.4 |
| Mitochondrial inner matrix | 7.5 |
| Secreted pancreatic fluid | 8.1 |

There are a range of pH values within different body fluids - dependent on function.

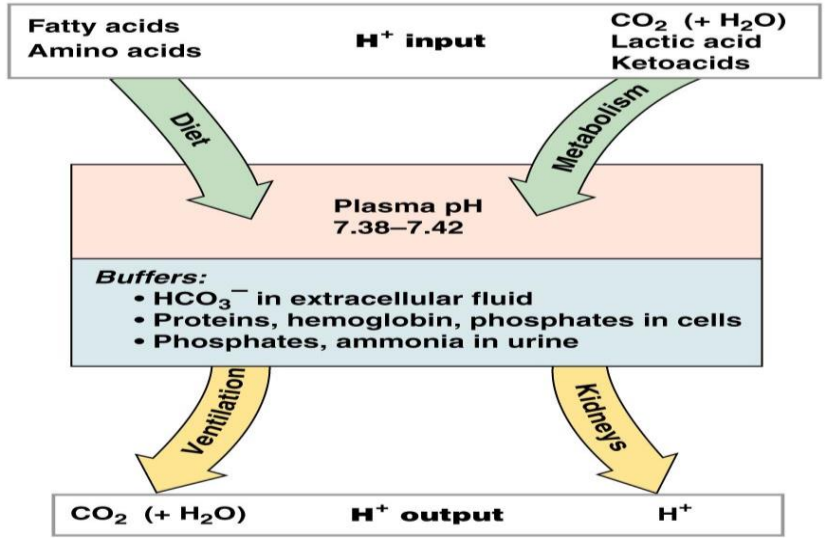
# Sources of H+

- The body generally PRODUCES more acids than bases.
- 1) Cellular aerobic metabolism produces 15,000 mmol CO2/day
- $CO2 + H20 \leftrightarrow H2CO3 \leftrightarrow H^+ + HCO3^-$ (volatile acid)
- Normally all volatile acid excreted by the lungs.

- 2) DIET : incomplete anarobic metabolism of carbohydrates (lactate), lipids (ketones) and proteins (H2SO4 , H3PO4 ) generates fixed (non-volatile) acids ~50 -100 mEq per day.
- In order to maintain balance, acids need to be BUFFERED and/or EXCRETED.

- CO2 is an important source of acidity.
  - Buffer
  - Excretion by lungs CO2
  - If there's no wash out of CO2 it will bind to water > High $H^+$> Acidosis

# Hydrogen and pH Balance in the Body




Catabolism of **SULFUR** containing amino acid AA gives $H_2SO_4$(sulfuric acid

Catabolism of **phospholipids/ phosphoproteins** gives $H_3PO_4$ ( phosphoric acid)

# How is [H+] Controlled?

- Three systems involved :

1) BUFFERS
   - First defense
   - second to second regulation of [H+]

2) Excretion of $CO_2$ ($\downarrow H_2CO_3$) by LUNGS (removal of volatile acid)
   - Second defense
   - regulation in minutes - to – hours

3) Excretion of $H^+$ ($\uparrow HCO_3^-$) by KIDNEYS (fixed acids)
   - Third defense
   - regulation over several hours to days
   - slowest, but most POWERFUL, of body's acid-base regulatory systems.

- Buffers has the ability to do immediate regulation for the pH but it only limits the change without eliminating it.
- Lungs work better with respiratory problems (like respiratory acidosis and alkalosis)

# Henderson-Hasselbalch Equation

Details are not important just know how to calculate the pH

هذي معادلة تقيس البي اتش باستخدام العلاقة بين تركيز ثاني أكسيد الكربون والبايكاربونيت في الدم .. يعني نحتاج فيها Blood sample

- It's a Relative concentrations of CO2 and HCO3 - in plasma / ECF , determine pH.

- Show the relationship between pH, hydrogen ion concentration and the ratio of buffer membrane in a solution.

$$\mathbf{pH} = pK' + \frac{\log [HCO_3^-]}{S\ (PCO_2)}$$

$$\mathbf{pH} = 6.1 + \frac{\log [HCO_3^-]}{0.03\ (PCO_2)}$$

$$7.4 = 6.1 + 1.3$$

**PH=** is the negative logarithm of H+ in mol/L.(tells us how acidic a solution is).
**PK=** is negative logarithm of overall dissociation constant for the reaction = 6.1 in health. (tells us how acidic or not a given hydrogen atom in a molecule is).
**S=** is solubility of CO2 in solution = 0.03 at 37°C. In **health**, **[HCO3-]** = 24 mmol/L & **PCO2** = 40 mm Hg

# What happen to the pH using H-H ?

- In case if the HCO3 in Plasma remains normal

1. If Pco2 increased, the ratio of [HCO3]P/ 0.03 Pco2 will decrease which lead to acidosis.
2. If Pco2 decrease, the ratio will increase and pH will increase causing alkalosis.

- In case the Pco2 remains normal

1. Increase bicarbonate in plasma causes an increase in the ratio which leads to alkalosis.
2. Decrease in bicarbonate in plasma causes a decrease in the ration which leads to acidosis.

MEDICINE
KING SAUD UNIVERSITY

Physiology Team 436

Revised by
شوق الأحمري & طراد الوكيل

Renal Block

# Body Fluid Buffers

Don't worry about the number of slides; they are mostly pictures to help you understand

Red: very important.
Green: Doctor's notes.
Pink: formulas.
Yellow: numbers.
Gray: notes and explanation.

**Physiology Team 436 – Renal Block Lecture 10**

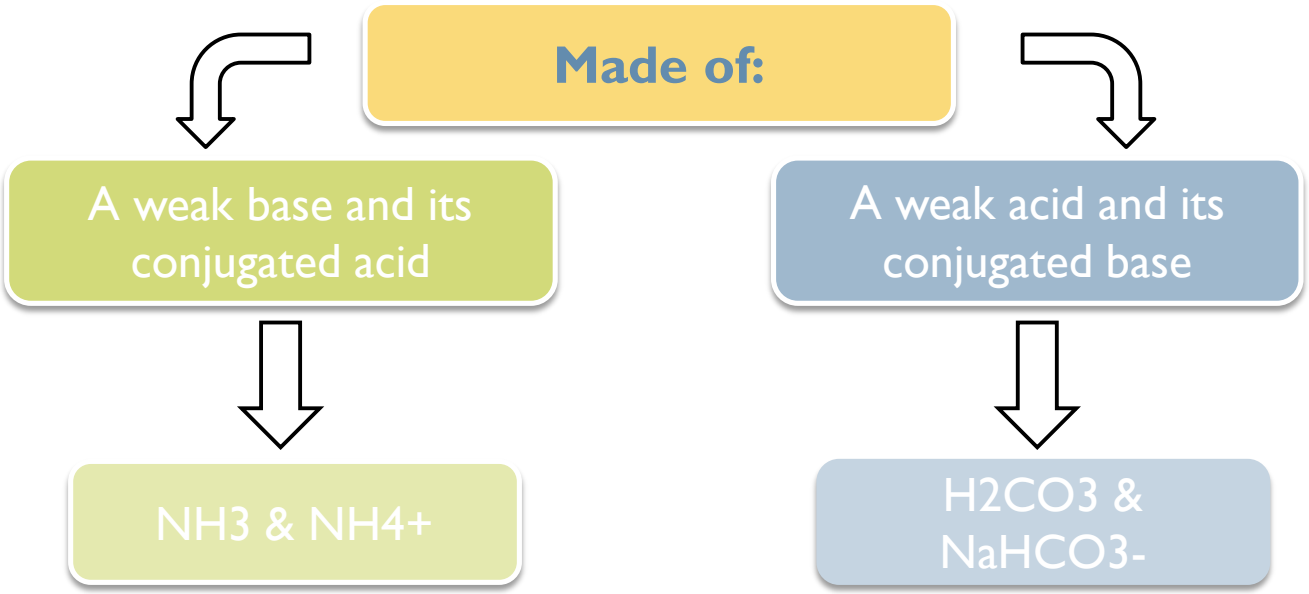


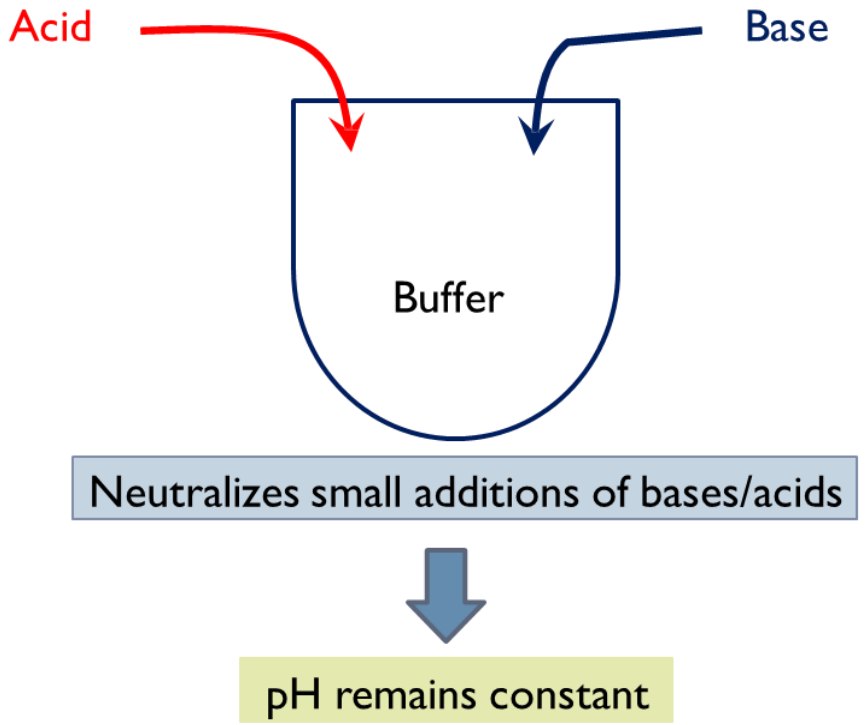
For further understanding please check our "Extra Notes" file which contains extra explanation from reference books.

**ONLY IN FEMALES' SLIDES**

# What is a Buffer?

- A buffer: a solution that resists changes in pH upon addition of small amount of acids and bases.
- A buffer: is a mixture of a weak acid and a weak base that are in equilibrium.



# Control of [H+] - Buffers

- Buffer is substance that stabilizes (limits the change of) [H+] when H+ ions are added or removed from a solution.
- They do not eliminate H+ from body , REVERSIBLY bind H+ until balance is re-established.
- General form of buffering reaction usually in form of conjugate acid-base pair:

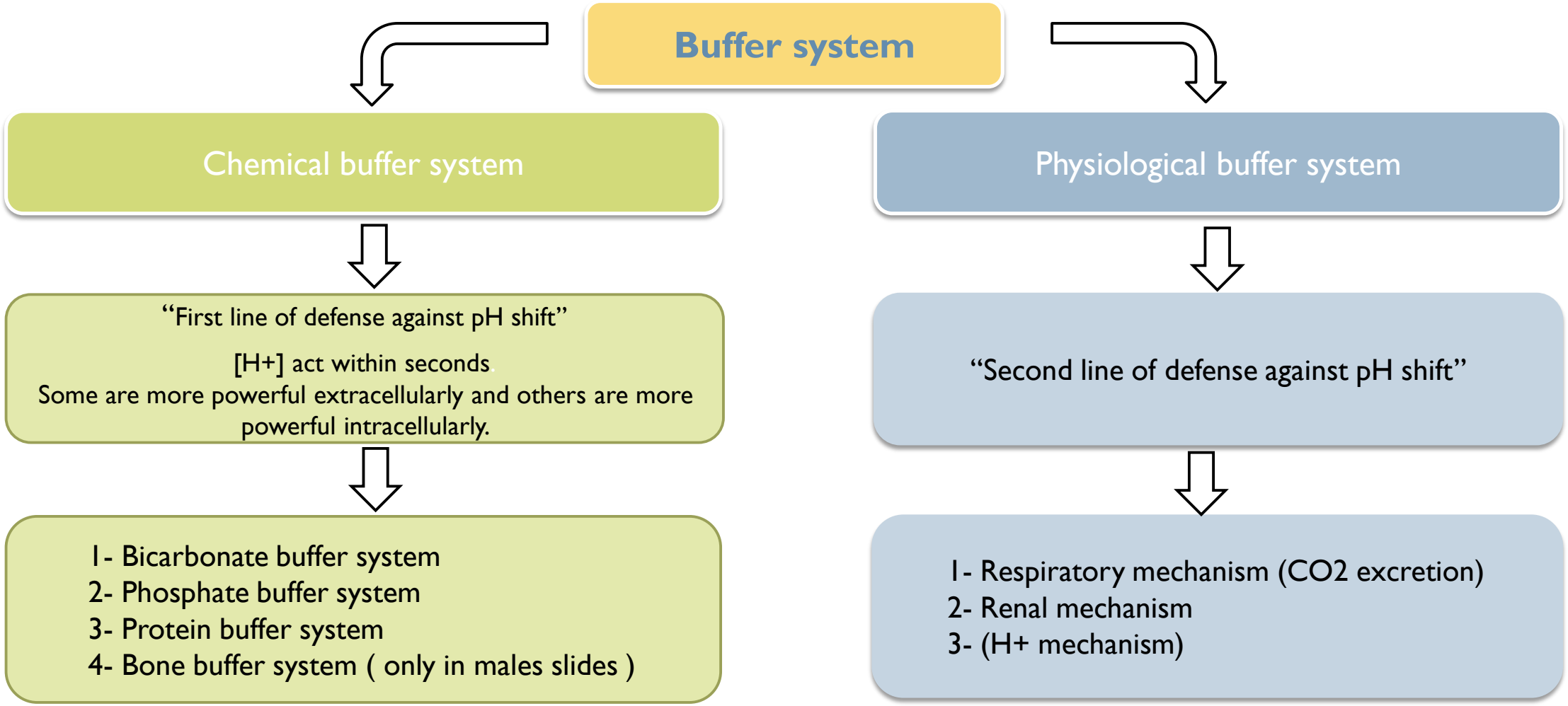
HA = undissociated acid
A- = conjugate base (any anion)


- Reaction direction and dissociation rate dependent on effective concentration of each chemical species.
- If :

[H+ ]↑ then equation moves Leftwards.

[H+ ]↓ then equation moves Rightwards - minimises changes in [H+].

# Buffer system



# Chemical buffering system

1- Bicarbonate buffer system :

Most important buffering system and the main ECF buffer system.

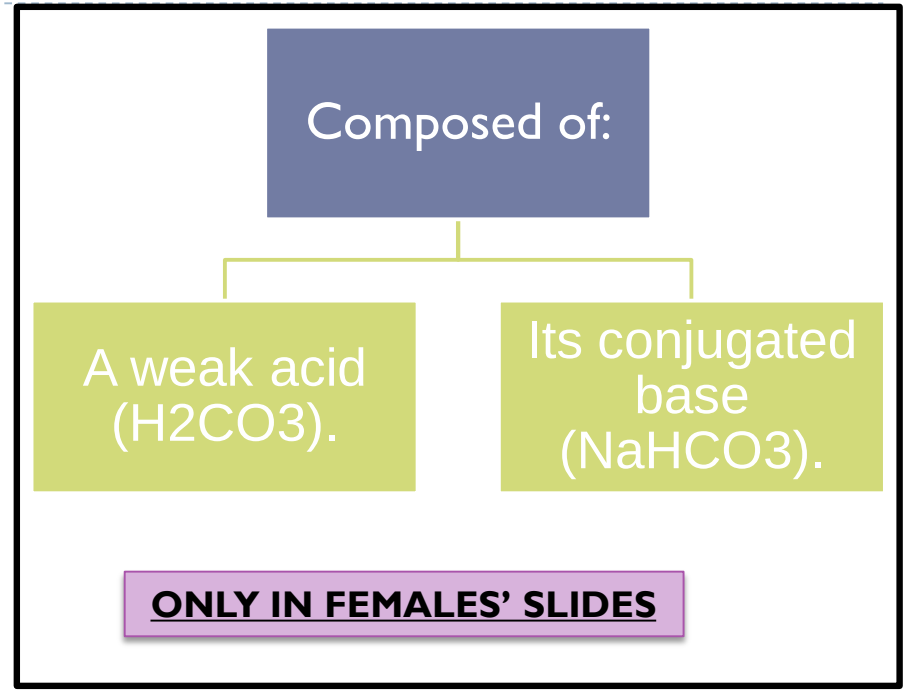
Works by acting as proton acceptor for carbonic acid.

$$CO_2 + H_2O \xrightleftharpoons{CA} H_2CO_3 \rightleftharpoons H^+ + HCO_3^-$$

$$NaHCO_3 \rightleftharpoons Na^+ + HCO_3^-$$

**Using HH equation : ( *Henderson-Hasselblach* )**

$$pH = pK + \log_{10}\frac{[HCO_3^-]}{[H_2CO_3]}$$




- To maintain pH of **7.4**, $HCO_3^-$ : $H_2CO_3$ = **20:1** – if ratio changes, so too will pH.
- When enough [$H^+$] added to halve [$HCO_3^-$], pH would drop to 6.0, **BUT**, $H_2CO_3 \Leftrightarrow H_2O + CO_2 \rightarrow$ ventilation↑ and $CO_2$ is removed.

∴ buffering means that **pH only drops to ~ 7.2**.

[H2CO3] very low (6800 x less than HCO3-), difficult to measure but directly proportional to dissolved arterial [CO2] = Pco2 x solubility coefficient (0.03 for CO2).

# Chemical Buffering System

- Buffering power of CO2/HCO3- system (against acids but not bases) usually only limited by depletion of HCO3-

**As pH of a $CO_2/HCO_3^-$ solution depends on the ratio of $HCO_3^-$ : $Pco_2$ rather than $[HCO_3^-]$ and :**

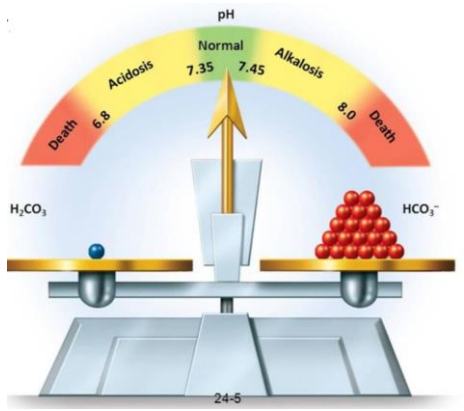
a) **$[HCO_3^-]$** is controlled mainly by *kidneys.*
b) **$Pco_2$** is controlled by *lungs*

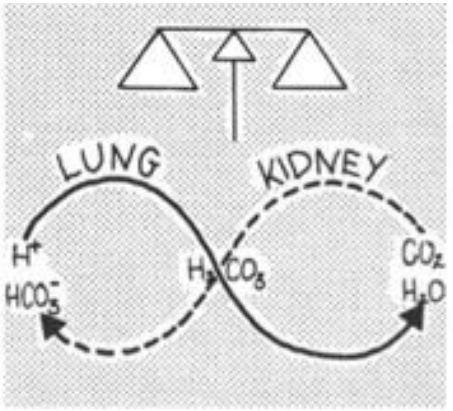
**pH can be expressed as "**

$$pH = \text{constant} + \frac{\text{kidneys}}{\text{lungs}}$$

- ↑↑ $HCO_3^-$ will ↑↑ pH
- ↑↑ PCO2 will ↓↓ pH

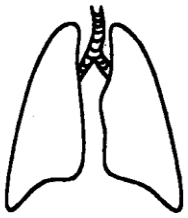
Ratio of $\frac{HCO3^-}{PCO2}$ is ≈ 20:1



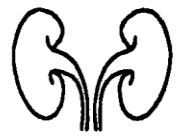




$$CO_2 + H_2O \rightleftharpoons H_2CO_3^- \rightleftharpoons H^+ + HCO_3^-$$

Lungs



Kidneys



# The Bicarbonate Buffer System

1. $H_2CO_3$ forms in the body by the reaction of $CO_2$ & $H_2O$

$$CO_2 + H_2O \xrightleftharpoons{\text{Carbonic anhydrase}} H_2CO_3$$

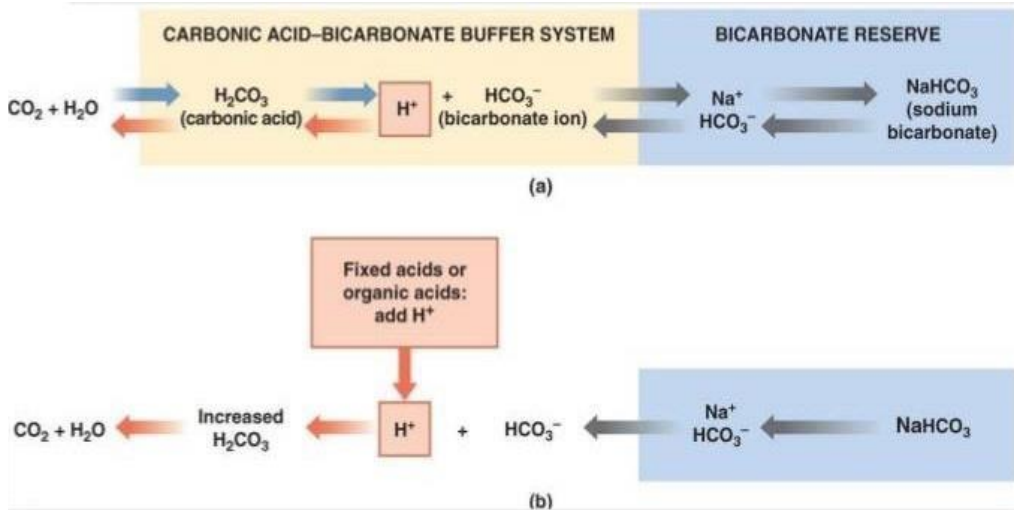
2. H2CO3 ionizes weakly to form small amounts of H+ & HCO3-

$$H_2CO_3 \rightleftharpoons H^+ \,\&\, HCO_3^-$$

3. The second component is NaHCO3 which dissociates to form Na+ & HCO3-

$$NaHCO_3 \rightleftharpoons Na^+ \,\&\, HCO_3^-$$

4. Putting all together:



ONLY IN FEMALES' SLIDES

# The Bicarbonate Buffer System Henderson-Hasselbalch Equation

NOT important

- What is the HHE?

It is an equation that enables the calculation of pH of a solution.

- What is it?

$$pH = pK + log \frac{HCO3^-}{0.03 \; X \; PCo_2}$$

$\Longrightarrow$

K = dissociation constant, pK = 6.1
Solubility of CO2 = 0.03

- How was it derived?

1- H2CO3 and its dissociated ions are always in equilibrium⟶ the products of the eaction on one side of the equation are proportional to the product on the other side.

$$H_2CO_3 \rightleftharpoons H^+ + HCO_3^- \Longrightarrow [H_2CO_3] \; \alpha \; [H^+] \; X \; [HCO_3^-]$$

2- Since H2CO3 is a weak acid, it will not dissociate completely and the concentration of its products will depend on its dissociation constant (K).

$$K \; X \; [H_2CO_3] = [H^+] \; X \; [HCO_3^-]$$

3- Based on the previous equation, [H+] can be expressed as follows:

$$[H^+] = K \; X \; \frac{[H_2CO_3]}{[HCO_3^-]}$$

# The Bicarbonate Buffer System Henderson-Hasselbalch Equation

NOT important

4- Because H2CO3 can rapidly dissociate into CO2 and H2O. And since CO2 is much easier to measure it can replace H2CO3 in the equation:

$$[H^+] = K_X \frac{[Co_2]}{[HCO_3^-]}$$ ⟹ This is Henderson's equation(1908) ⟹ It means that: ↑ [CO2] →↑ [H+], ↑ [HCO3-] → ↓ [H+]

5- In 1909, Sorensen created the pH scale to express [H+ ]

$$pH = -\log[H^+]$$

6- In 1916, Hasselbalch decided to merge Henderson's equation with Sorensen's pH scale creating what we now know as the **"Henderson-Hasselbalch equation".**

$$[H^+] = K_X \frac{[Co_2]}{[HCO_3^-]}$$ ⟹ $$-\log[H^+] = -\log\left(K_X \frac{[CO2]}{[HCO3^-]}\right)$$ ⟹ $$pH = pK + \log\frac{[HCO3]}{[Co2]}$$ ⟹ This is Henderson-Hasselbach equation (1908)

7-Since it is much easier to measure PCO2 rather than dissolved [CO2] and because dissolved CO2 is proportional to PCO2 multiplied by the solubility of CO2 (0.03 mmol/mmHg) → [CO2] was replaced by PCO2 X 0.03

$$pH = pK + \log\frac{[HCO3]}{0.03\,X\ PCO_2}$$

ONLY IN MALES' SLIDES

# The Bicarbonate Buffer System Henderson-Hasselbalch Equation

- At 37°C :

$$pH = 6.1 + \log_{10} \frac{[HCO_3^-]}{0.03 \times Pco_2\ (mM)}$$

- As pH and Pco2 can both easily be measured, possible to estimate [HCO3-]

(normally ~ 24 mEq/L in arterial blood) → Can estimate [acid or base] required to correct imbalance.

- To maintain pH of 7.4 :

HCO3- : H2CO3 = 20:1 [ if ratio changes, so too will pH ]. "Because our bodies produce a lot od acids, we need a lot of base in order to maintain balance" .

- When enough [H+] added to halve [HCO3-], pH would drop to 6.0

BUT, H2CO3 ⇔ H2O + CO2 → ventilation↑ and CO2 is removed.

- Buffering means that pH only drops to ~ 7.2.

# Chemical Buffering System

## 2-Phosphate Buffering System

- Phosphate buffer system is not important as extracellular fluid buffer ( concentration too low ) . " it is an intracellular anion"
- However, major intracellular buffer and important in renal tubular fluid.
- Main components are : $HPO_4^{2-}$ (Hydrogen phosphate) and $H_2PO_4^-$(dihydrogen phosphate)

**ONLY IN MALES' SLIDES**

$$H^+ + HPO_4^{2-} \leftrightarrow H_2PO_4^-$$

(Strong acid converted to weak acid ∴ less effect on pH)

$$OH^- + H_2PO_4^- \leftrightarrow H_2O + HPO_4^{2-}$$

(Strong base converted to weak base ∴ less effect on pH)

# Chemical Buffering System

## 3-Protein buffers

- Contributes to buffering inside cells.E.g. Hb.

**ONLY IN MALES' SLIDES**

- Proteins among most plentiful buffers in body, particularly highly concentrated intracellularly.
- ~ 60 - 70% of total chemical buffering of body fluids is located intracellularly, mostly due to intracellular proteins.
- Carboxyl and amino groups on plasma proteins are effective buffer.

$$RCOOH \leftrightarrow RCOO^- + H^+$$
$$RNH_3^+ \leftrightarrow RNH_2 + H^+$$

- Most important non-bicarbonate buffering proteins are titratable groups on HAEMOGLOBIN (Hb also important for buffering CO2).

$$CO_2 + H_2O \leftrightarrow H_2CO_3 \leftrightarrow H^+ + HCO_3^- \rightarrow H^+ + Hb^- \leftrightarrow HHb$$

- "Deoxygenated Hb is a better buffer than oxygenated Hb."
- pH of cells changes in proportion to pH of extracellular fluid.
- CO2 can rapidly traverse cell membrane.

# Chemical Buffering System

## 4-Bone:

- Probably involved in providing a degree of buffering (by ionic exchange) in most acid-base disorders.
- However, important source of buffer in Chronic metabolic acidosis (i.e. renal tubular acidosis & uremic acidosis).
- CaCO3 (base) "Calcium carbonate" is most important buffer released from bone during metabolic acidosis.
- Results in major depletion of skeletal mineral content (e.g. Chronic metabolic acidosis that occurs with renal tubule acidosis (RTA) can lead to development of Rickets / osteomalacia).

# Summary of Body's Buffering Systems

**ONLY IN FEMALES' SLIDES**

- Buffer systems do not work independently in body fluids but actually work together.
- A change in the balance in one buffer system, changes the balance of the other systems.
- Buffers do not reverse the pH change, they only limit it.
- Buffers do not correct changes in [H+] or [HCO3-], they only limit the effect of change on body pH until their concentration is properly adjusted by either the lungs or the kidney.

**ONLY IN MALES' SLIDES**

Control of (H+)-Buffers

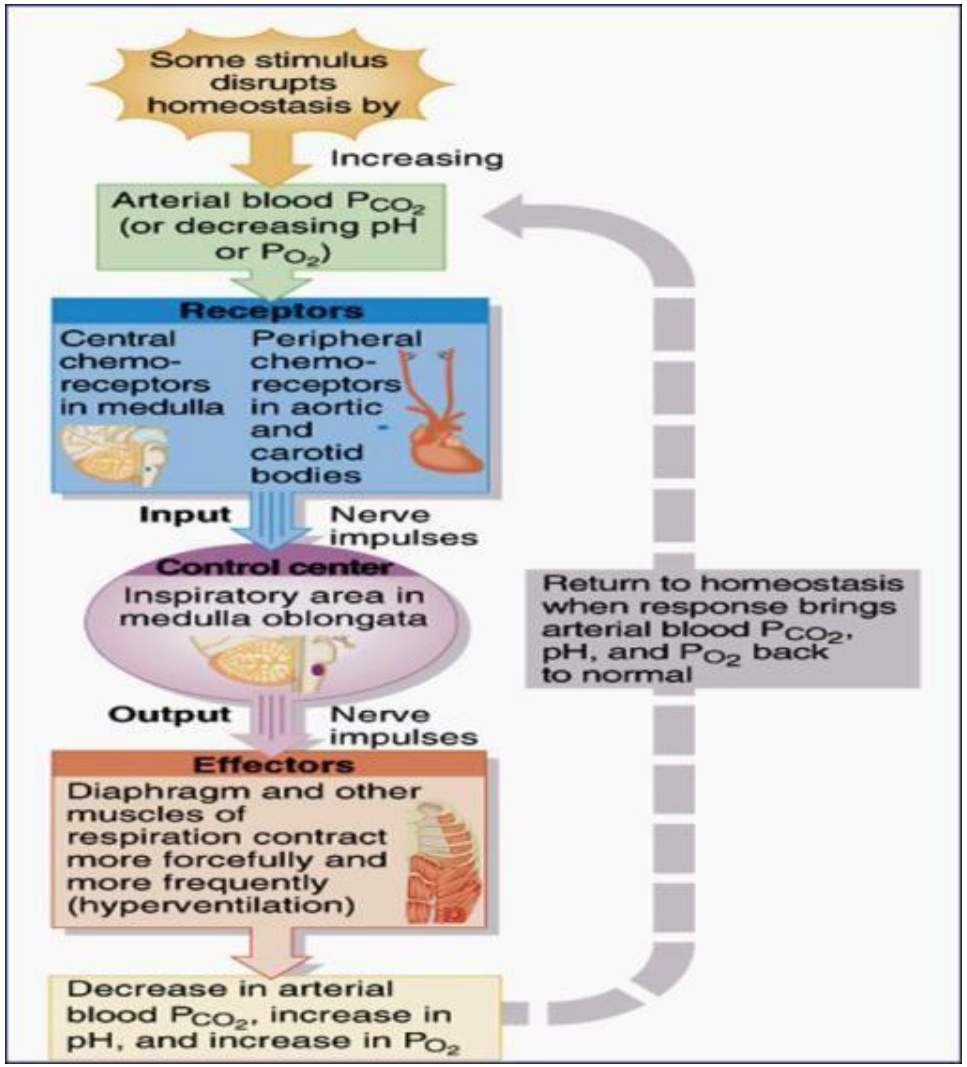
Remember that all of these buffer systems work in tandem*, NOT in isolation.
Buffers can only limit changes in pH, they cannot reverse them.
Once arterial pH has deviated from normal value, can only be returned to normal by respiratory or renal compensation.

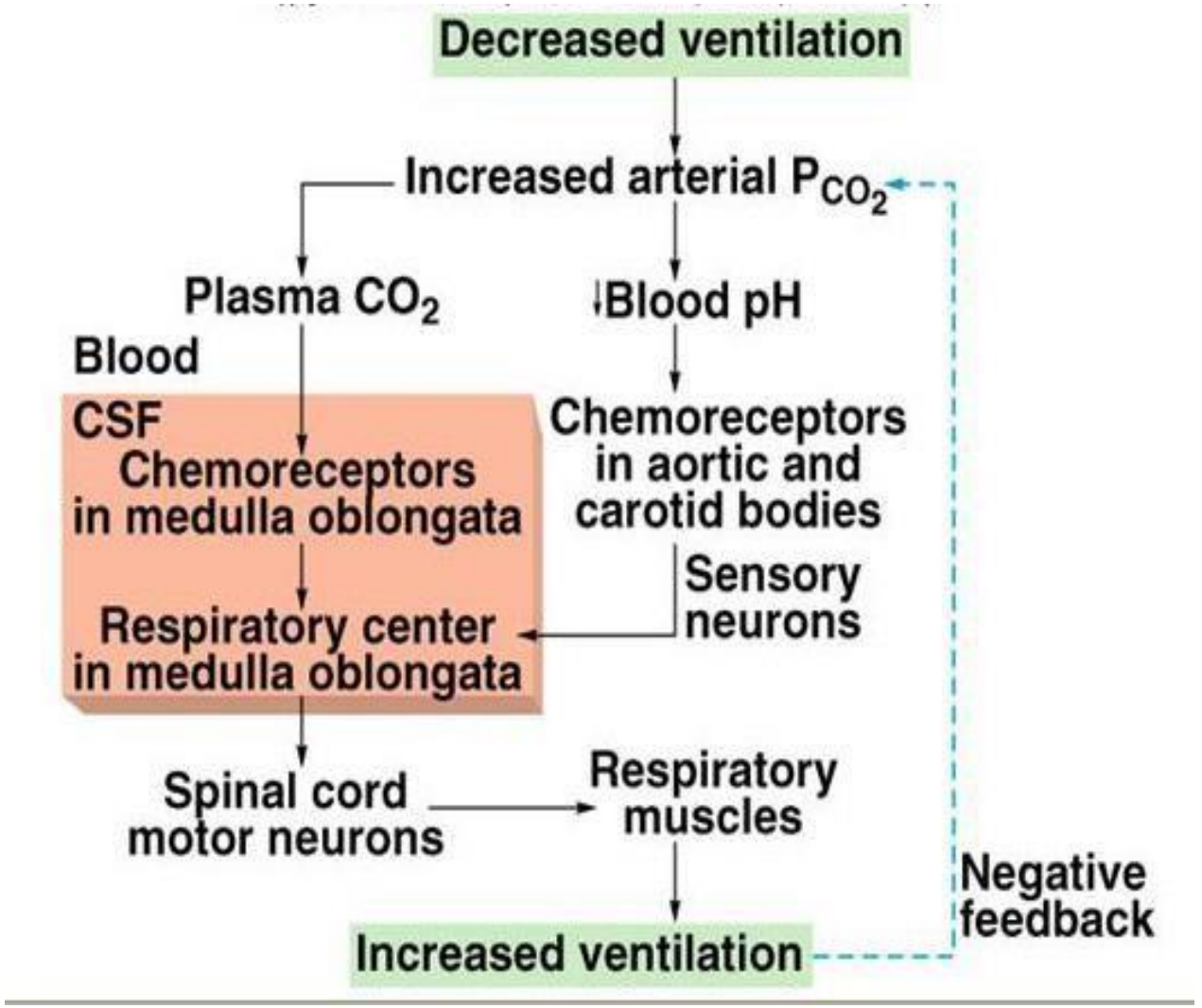
*Tandem : one after the other

# Respiratory Regulation of Acid-Base Balance

| Respiratory system |
| --- |
| 2nd line of defence against acid-base disturbances in thebody. |
| Pulmonary expiration of CO2 normally BALANCES metabolic formation of CO2 . |
| HOW? By modulating CO2 excretion |
| Changes in alveolar ventilation can alter<br>**plasma Pco2**<br>↑ $[H^+]$ → ↑ ventilation, ↓Pco2 , ↑pH<br>↓ $[H^+]$ → ↓ ventilation, ↑ Pco2 , ↓ pH<br>Normally, PCO2 = 40 mmHg (35-45 mmHg) |
| Changes in [H+ ] also alters **ALVEOLAR VENTILATION.** |
| **POWERFUL**<br>(1-2 x better than extracellular chemical buffers), but cannot fully rectify disturbances outside respiratory system, i.e. with fixed acids like lactic acid. |
| Acts relatively **RAPIDLY** to stop [H+ ] changing too much until renal buffering kicks in but **DOES NOT** eliminate H+ (or HCO3 - ) from body. |
| Abnormalities of respiration can alter bodily [H+ ] resulting in;<br>-**RESPIRATORY ACIDOSIS** -<br>Or<br>- **RESPIRATORY ALKALOSIS.** |

# Respiratory Regulation of CO2



# Renal regulation of Acid-Base Balance

**ONLY IN MALES' SLIDES**

| Renal system |
| --- |
| **MOST EFFECTIVE** regulator of pH but much **SLOWER** (i.e. max. activity after 5-6 days) than other processes. |
| Responsible for **ELIMINATING** the 80 -100 mEq of fixed **ACIDS** generated each day. |
| Normally, must also **PREVENT** renal **LOSS** of freely – filterable **HCO3** - in order to preserve this primary buffer system. |
| **BOTH PROCESSES** are dependent on both H+ filtration / secretion into renal tubules and secretion / reabsorption of plasma [HCO3 - ]. |
| Kidneys also responsible for **COMPENSATORY CHANGES** in [HCO3 - ] during respiratory acid-base disorders. |

IF KIDNEYS FAIL, pH BALANCE WILL FAIL

**ONLY IN FEMALES' SLIDES**

- 3rd line of defence against acid-base disturbances and the most powerful.
- It regulates by excreting either an acidic or basic urine.

## HOW?

1. Secreting H+
2. Reabsorbing HCO3-
3. Generating "new" bicarbonate ions.

ONLY IN MALES' SLIDES

# Renal regulation of Acid-Base

- Overall mechanism straightforward:

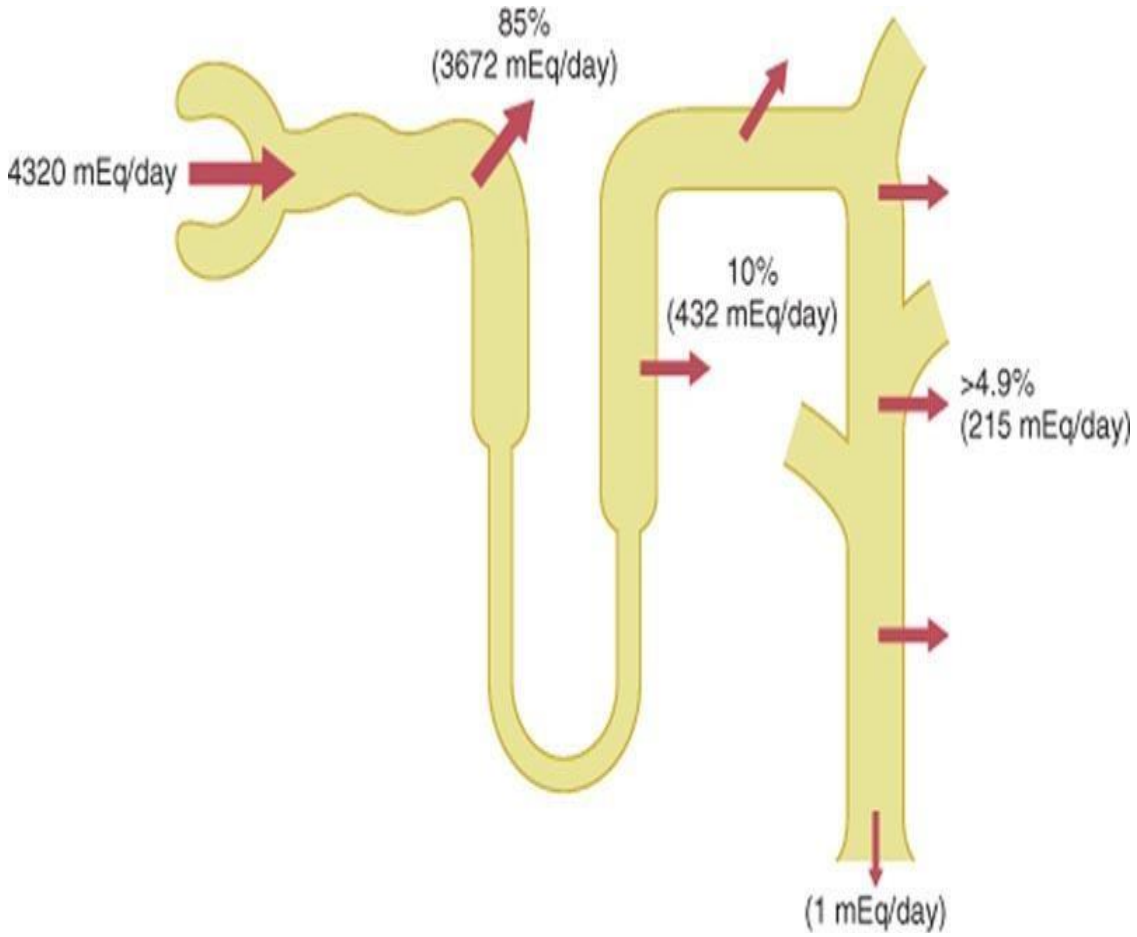
- large [HCO3 - ] continuously filtered into tubules
- large [H+ ] secreted into tubules

if more H+ secreted than HCO3 – filtered
= a net loss of acid → ↑pH

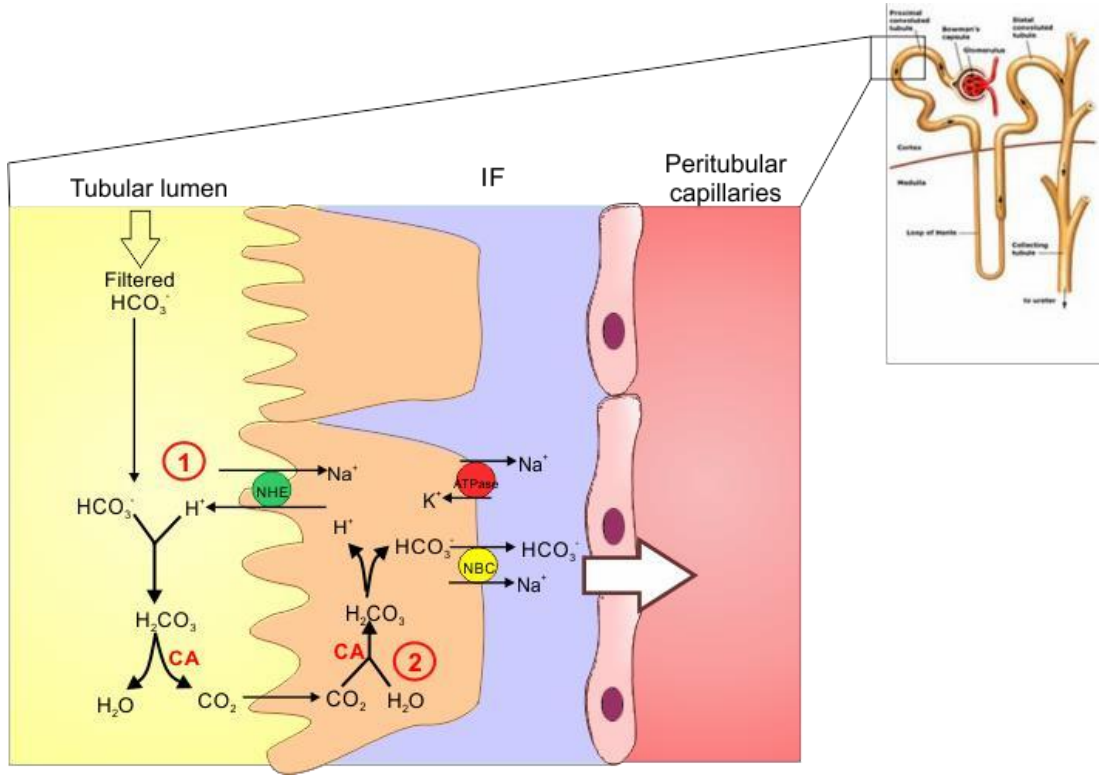
if more HCO3 - filtered than H+ secreted
= a net loss of base → ↓pH

# Cont.

## Overview HCO3- Reabsorption by the Renal Tubules




## How is HCO3- Reabsorbed by the tubules?



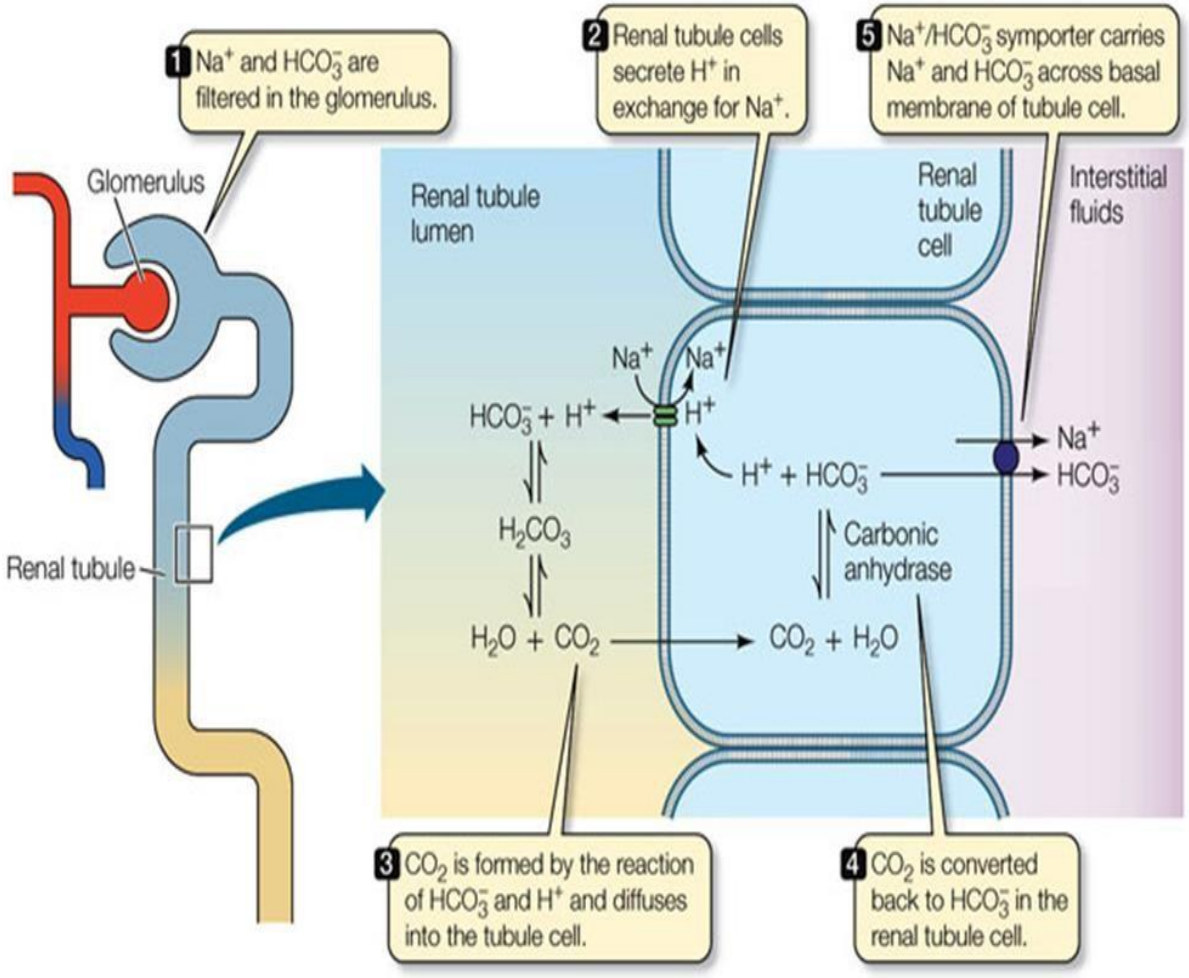

## What happens at the PCT?

The PCT reabsorbs, "reclaims", 80- 90% of the filtered HCO3-

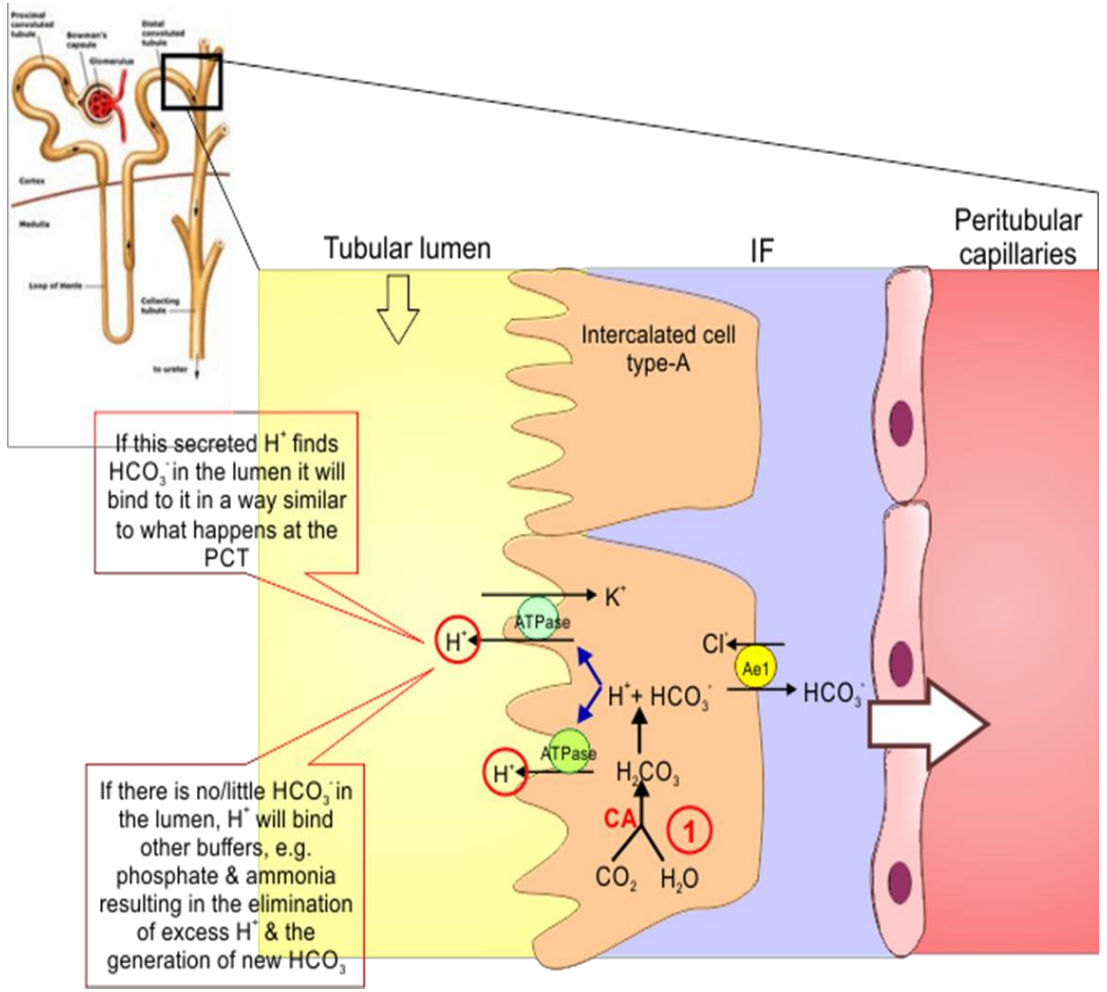
HCO3-reabsorption is linked to H+ secretion.

# Cont.

## HCO3- Reabsorption by the PCT




## What happens at the DCT & CT?



# What happens at the late DCT & CT?

- The filtrate arriving at the DCT & CT is low in $HCO_3^-$.

- The distal segments of the nephron are characterised by the presence of "intercalated cells" capable of ***actively secreting $H^+$*** through $H^+$-ATPase and $H^+$-$K^+$ ATPase present on their apical membrane (***Type-A intercalated cells***).

- Only a limited number of $H^+$ can be excreted in its free form in urine.

- Lowest possible urine pH=4.5 → ≈ 0.04 mmol/L of free $H^+$.

ONLY IN FEMALES' SLIDES

# Non-Bicarbonate Buffers in the Tubular Lumen?

- The extra H+ secreted will need to be buffered in the tubular lumen

2 main non-bicarbonate buffers in the tubule

**Filtered**

**Phosphate buffer system**

$H_2PO_4^- \leftrightarrow HPO_4^{-2} + H^+$

*(acid) (base)*

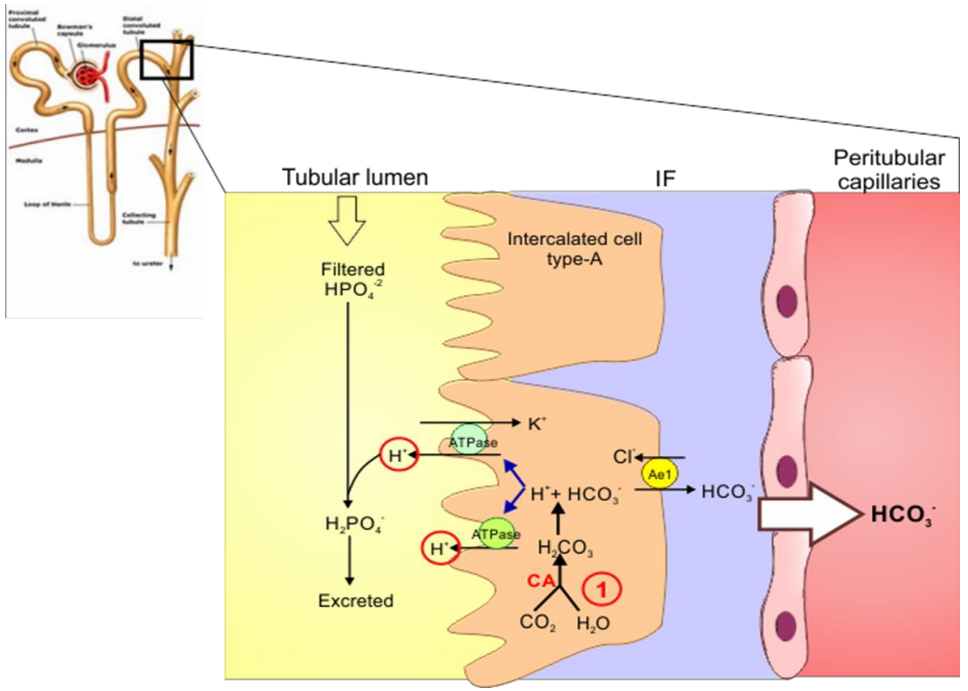
**Synthesized**

**Ammonia buffer system**

$NH_4^+ \leftrightarrow NH_3 + H^+$

*(acid) (base)*

Excretion of H+ and Generation of New HCO3



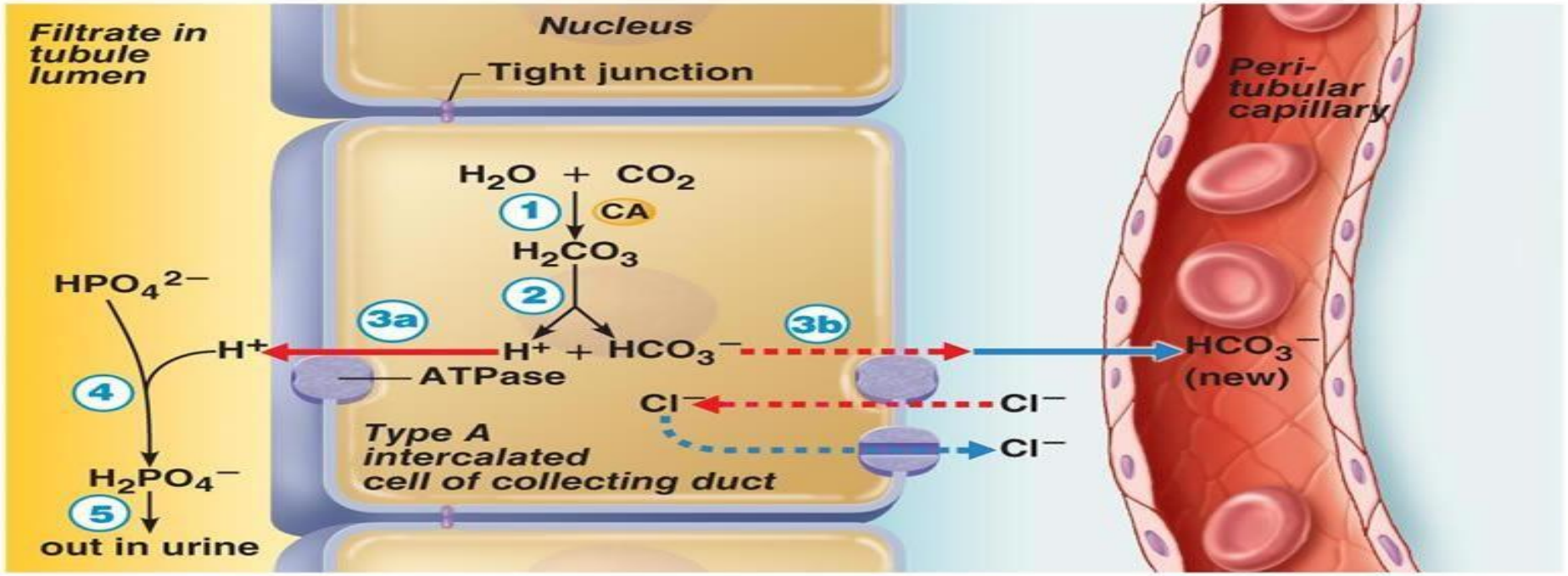

Excretion of $H^+$ as phosphate is capable of handling a limited amount of $H^+$ and will not be enough to rid the body of its daily acid load nor if there is unusually high acid production.

# Cont.

(1) $CO_2$ combines with water within the type A intercalated cell, forming $H_2CO_3$.

(2) $H_2CO_3$ is quickly split, forming $H^+$ and bicarbonate ion ($HCO_3^-$).

(3a) $H^+$ is secreted into the filtrate by a $H^+$ ATPase pump.




(3b) For each $H^+$ secreted, a $HCO_3^-$ enters the peritubular capillary blood via an antiport carrier in a $HCO_3^-$-$Cl^-$ exchange process.

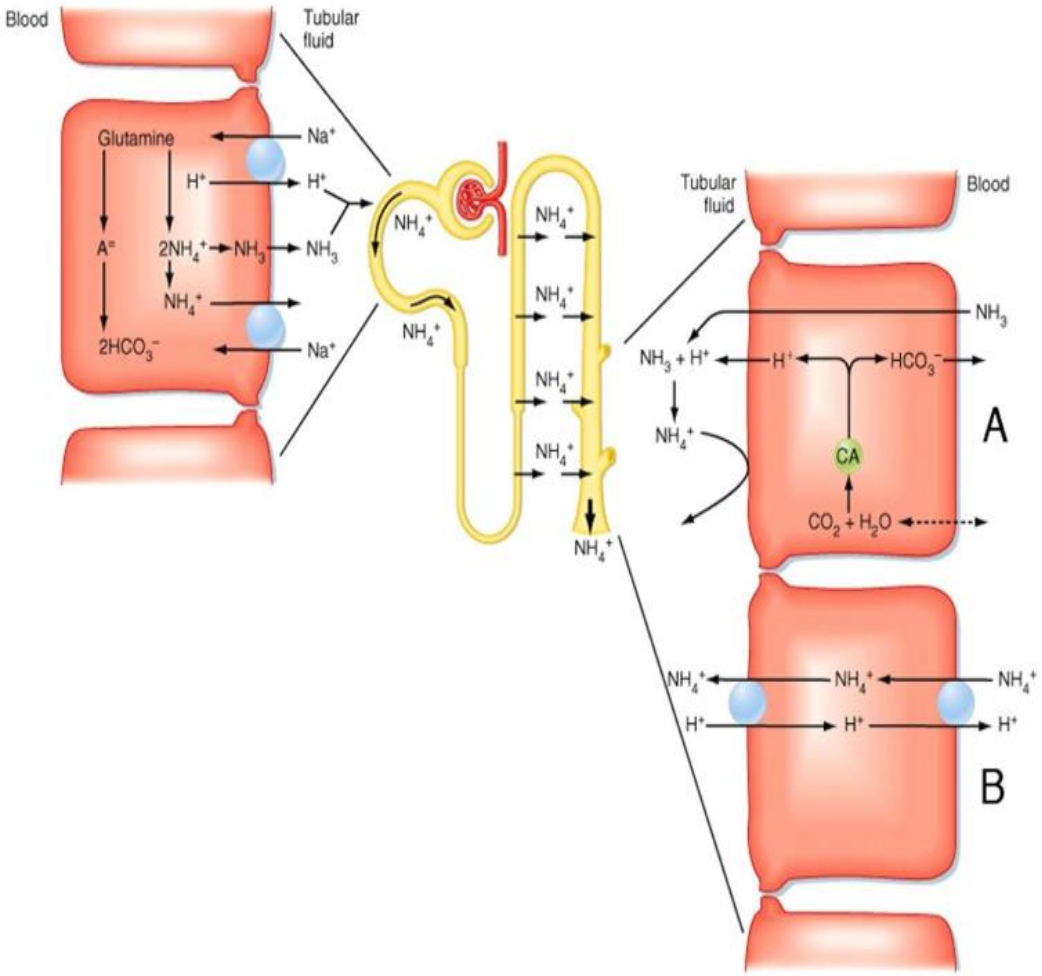
(4) Secreted $H^+$ combines with $HPO_4^{2-}$ in the tubular filtrate, forming $H_2PO_4^-$.

(5) The $H_2PO_4^-$ is excreted in the urine.

# Excretion of H+ and Generation of New HCO3 (The Ammonia Buffer System)

**ONLY IN FEMALES' SLIDES**

- Renal tubular cells, especially PCT, are capable of generating ammonium (NH4+) "ammoniagenesis" which is then excreted in urine carrying with it H+.
- The rate of ammoniagenesis can be modified according to the needs of the body.
- Quantitatively, the ammonia buffer system is more important than the phosphate buffer system for H+ excretion in urine.
- It is the most important system in case of acidosis.



ONLY IN MALES' SLIDES

# H+ / HCO3- Control by the Kidney:

## Renal H+ secretion

- H+ enters filtrate by FILTRATION through glomeruli and SECRETION into tubules.
- Most H+ secretion (80%) occurs across wall of PCT via Na+ /H+ antiporter (& H+ - ATPase in type A cells of DCT).
- This H+ secretion enables HCO3 - reabsorption.
- The primary factor regulating H+ secretion is systemic acid-base balance

a) ACIDOSIS stimulates H+ secretion

b) ALKALOSIS reduces H+ secretion

## Bicarbonate handling

- HCO3 - FREELY FILTERABLE at glomeruli (3 mM/min) and undergoes significant (> 99%) reabsorption in PCT, aLoH & cortical collecting ducts (CCDs).
- Mechanisms of HCO3 - reabsorption at PCT (& aLoH) and CCD are similar but not identical (will look at CCD cells in acid-base practical)
- Renal HCO3 - reabsorption is an ACTIVE process - BUT dependent on tubular secretion of H+ , NO apical transporter or pump for HCO3 - .

ONLY IN FEMALES' SLIDES

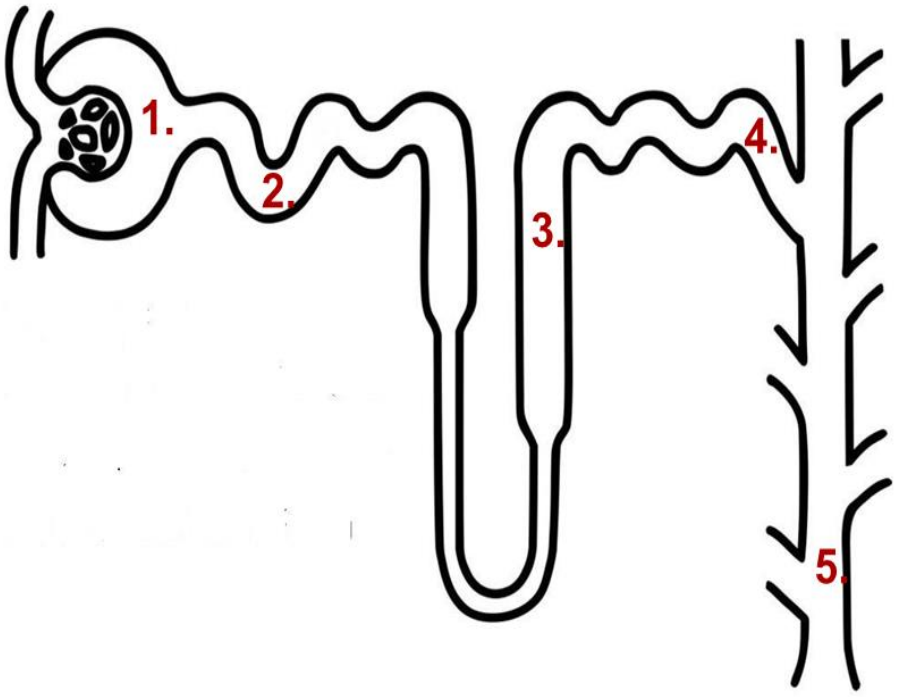
# The Overall Scheme of Renal Excretion of Acids & Bases

**To excrete acid**:

1. Freely filter $HCO_3^-$
2. Reabsorb the majority of filtered $HCO_3^-$
3. Reabsorb some additional $HCO_3^-$
4. Secrete $H^+$ (titrate filtered bases, i.e. $HPO_4^{-2}$) and secrete $NH_4^+$
5. Excrete acidic urine containing $NH_4^+$

**To excrete base**:

1. Freely filter $HCO_3^-$
2. Reabsorb the majority of filtered $HCO_3^-$
3. Reabsorb some additional $HCO_3^-$
4. Secrete some $HCO_3^-$
5. Excrete alkaline urine containing $HCO_3^-$



MEDICINE
KING SAUD UNIVERSITY

Physiology Team 436

Revised by
شوق الأحمري & طراد الوكيل

# Acid Base Disorders

Renal Block

This work is based on MALES' slides and contains Dr. Maha's notes and cases but NOT all of the females' slides since she did not go through them all.

Red: very important.
Green: Doctor's notes.
Pink: formulas.
Yellow: numbers.
Gray: notes and explanation.

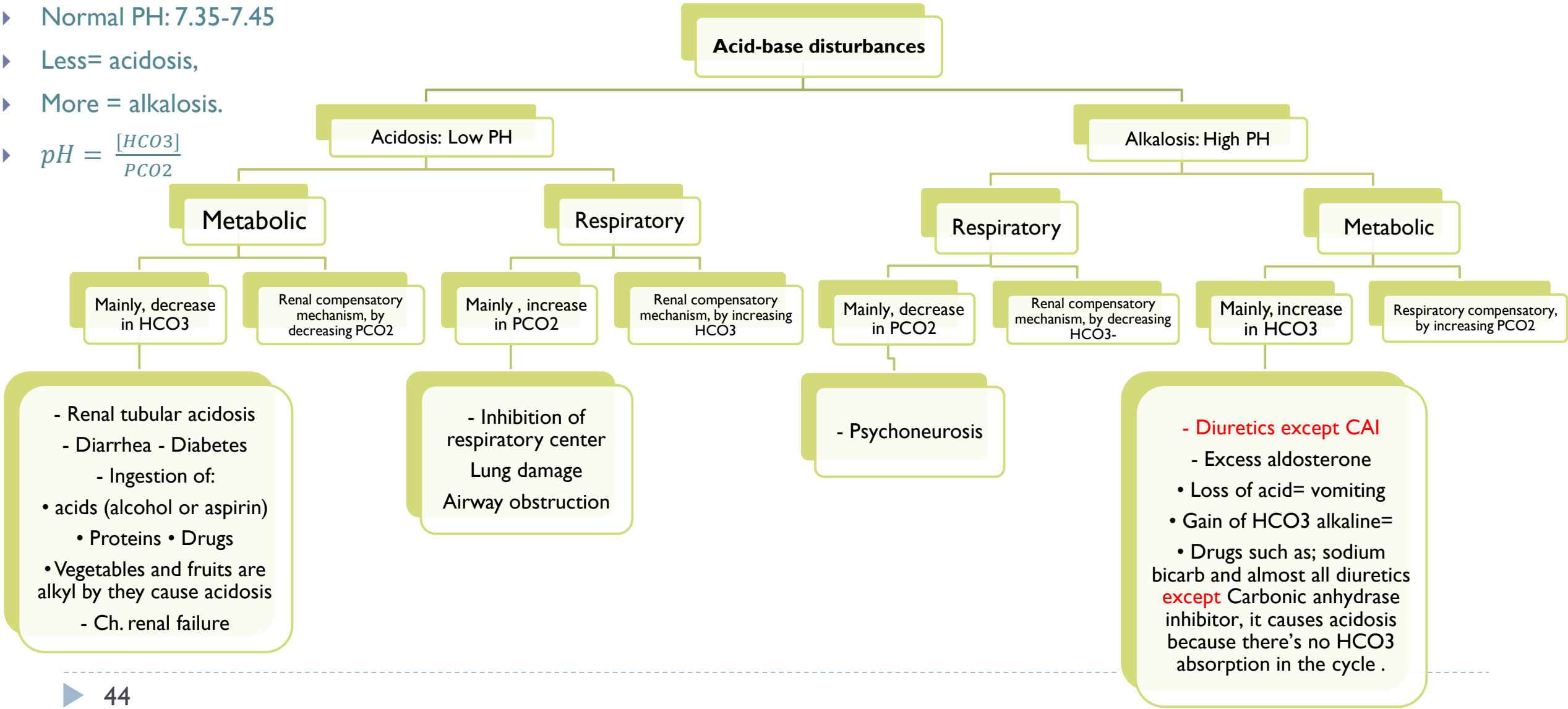
**Physiology Team 436 – Renal Block Lecture 11**



For further understanding please check our "Extra Notes" file which contains extra explanation from reference books.

# Overview (Important)

- Normal PH: 7.35-7.45
- Less= acidosis,
- More = alkalosis.
- $pH = \frac{[HCO3]}{PCO2}$



# Disturbances of Acid-Base Balance

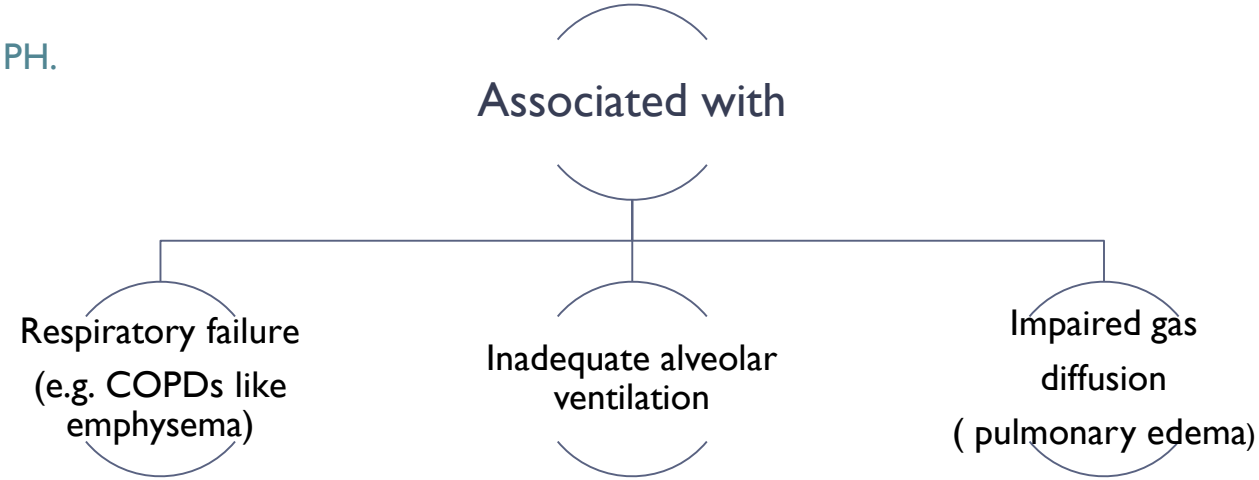
- Acid-base disturbances may be either **RESPIRATORY** or **METABOLIC**.


- **Linda corner:**
  - Disturbances of acid-base balance are among the most common conditions in all of clinical medicine. Acid- base disorders are characterized by an abnormal concentration of H+ in blood, reflected as abnormal pH. Acidemia is an increase in H+ concentration in blood (decrease in pH) and is caused by a pathophysiologic process called acidosis. Alkalemia, on the other hand, is a decrease in H+ concentration in blood (increase in pH) and is caused by a pathophysiologic process called alkalosis.
  - Disturbances of acid-base balance are described as either metabolic or respiratory, depending on whether the primary disturbance is in $HCO_3^-$ or $CO_2$. There are four simple acid-base disorders, where simple means that only one acid-base disorder is present. When there is more than one acid-base disorder present, the condi- tion is called a mixed acid-base disorder.

# Respiratory Acidosis

Hypoventilation = less co2 excretion= increase PCO2, decrease PH.

Kidney messes with HCO3 to increase PH, so: increase HCO3.



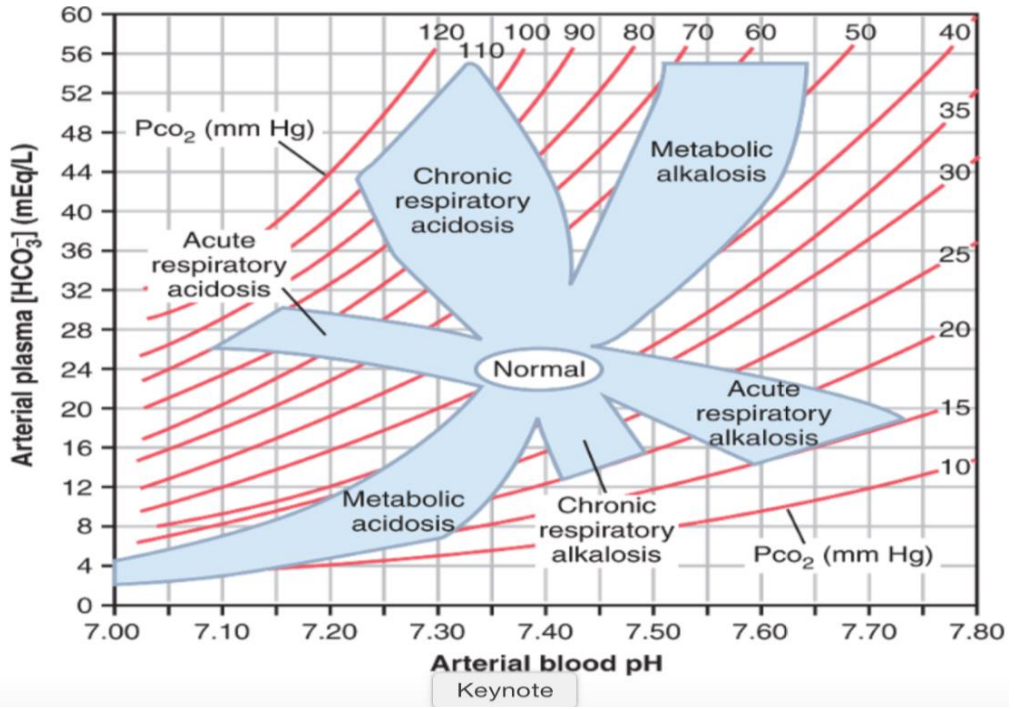

- Characterized by ↑ Pco2 *(hypercapnia)* and ↓ plasma pH.
- **Initial response is :**
- Increased conversion of CO2 to H+ and HCO3 **Increase** in ECF [H+] and plasma [HCO3-].
- INCREASED → Renal **SECRETION** OF H+
  INCREASED → **ABSORPTION** OF $HCO_3$ **[ COMPENSATORY MECHANISM ]**

- Alveolar ventilation : عملية التنفس وهي أخذ الأكسجين وطرح ثاني أكسيد الكربون
- the kidney act as a compensatory mechanism to correct respiratory acidosis caused by the lung

# Respiratory Acidosis

ممكن يجي عليها سؤال مهمة

**Respiratory Acidosis**
↓ plasma pH
↑ Pco2
↑ plasma [HCO3-]




- the diagram shows the acid base disorders. The shaded area show the range of values usually seen for each of the disorders.

- the reason why the HCO3 is high in respiratory acidosis is because it compensate the disorder. In other words, respiratory acidosis is compensated by metabolic alkalosis. Why?? The Henderson-hasselbalch equation can be used to understand why the increased HCO3 conc. is a compensatory response. It's used to normalize the ratio of HCO3/CO2 . Because when CO2 increase the ration will decrease leading to low ph. But when HCO3 also increase the PH will be normal

# Respiratory Alkalosis

- **Characterised by :** Reduced plasma $Pco_2$ *(hypocapnia)* and elevated pH↓ $Pco_2$ & ↑ plasma pH
- **Causes:** (results from ↑ ventilation and ↓ Pco2 ) increased gas exchange mainly due to HYPERVENTILATION *(Anxiety / fear/High altitude).*
- **Reduction** in **$Pco_2$** shifts buffering reaction to *the left* **Decrease** in ECF [$H^+$] and plasma [$HCO_3^-$]
- COMPENSATORY MECHANISM : **Decreased** : *↓ Renal **Secretion** of $H^+$ *↓ **Absorption** of $HCO_3^-$ *(still an excess of $HCO_3^-$ relative to $H^+$).*

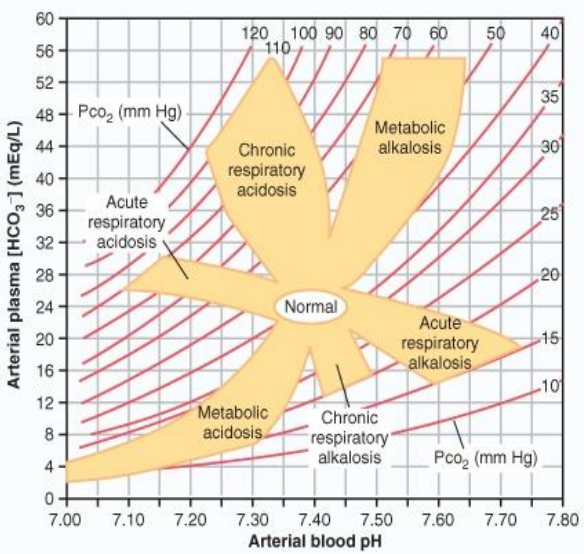
- in high altitude, the O2 is less, so to compensate this loss hyperventilation is triggered leading to depletion of Co2. Thus respiratory alkalosis may occur.
- HCO3 buffering reaction is shifted to left to decrease the amount of HCO3 and increase extracellular amount of Co2
- In alkalosis, the loss of $HCO_3^-$ helps return the plasma pH toward normal.

- Hyperventilation results in= increase PH, PCO2 decrease.
- If someone is going through a panic attack, we give him a paper bag so he can re-inhale CO2 and doesn't't go through alkalosis

# Davenport Diagram Acid-base alterations

**Respiratory Alkalosis**

↑ plasma pH
↓ Pco2
↓ plasma [HCO3-]




renal compensation for respiratory alkalosis consist of decrease excretion of H+ and decreased synthesis of new HCO3. Therefore decrease its reabsorption. And again the ration of HCO3/Co2 in the equation will be normalized

From the diagram :

- PH : 7.6 "HIGH"
- HCO3- : 20 "low"
- Pco2 : 20 "low"

# Metabolic Acidosis & Alkalosis

- Metabolic acidosis by Ingestion of:
Proteins
Drug
Vegetables and fruits are alkyl by they cause acidosis

- In shock: lactate accumulate > acidosis
- Diabetics: miss an insulin dose > hyperventilating (kussmaul breathing) > they go through acidosis and by breathing the compensate it

Loss of HCO3:
• From GIT or kidney:
Renal tubular acidosis

- Metabolic acidosis and alkalosis *( Non-$Co_2$)* includes **all situations** other than those in which primary problem is respiratory.
- By definition, metabolic acidosis and alkalosis *cannot* be due to excess retention or loss of CO2

Does arterial Pco2 remain unchanged in these cases? **NO!**

- ↑ **[H+]** in acidosis will reflexely *stimulate ventilation to lower Pco2.* Conversely, *ventilation will be inhibited in alkalosis* to restore [H+].
- **Remember, plasma Pco2** changes during metabolic acidosis / alkalosis are **a result of, not cause of**, compensatory reflex responses to non-respiratory abnormalities.

- Metabolic alkalosis:
• Loss of acid= vomiting
• Gain of HCO3= drugs such as; sodium bicarb and almost all diuretics except Carbonic anhydrase inhibitor, it causes acidosis because there's no HCO3 absorption in the cycle

الرئة ليست السبب في تغير الهيدروجين لكنها تساعد في تعديل هذا التغيير عن طريق التحكم بالتنفس.

# Metabolic Acidosis

**Characterized by :**

DECREASED [HCO3-] (<25mM) and pH.

- Reduction in pH + ↑ in ECF H+ concentration

**Caused by :**

Increased acid production

Impaired acid excretion

**Heavy exercise** stimulates anaerobic metabolism, producing lactic acid.

**Tissue hypoxia** (produces lactic acid)

**High protein diet** : protein catabolism produces phosphoric acid and sulphuric acid.

Ingested substances such as methanol, aspirin (acetylsalicylic acid), ethylene glycol

**Metabolic Acidosis**

**Can occur in response to:**

**'Causes**

**Addition of fixed acids** (e.g. diabetic ketoacidosis ).

**Alterations in renal function** (**inability to excrete $H^+$** ).

**High fat diet** fat catabolism produces fatty acids.

**Severe diarrhoea** loss of bicarbonate from intestines

ONLY IN FEMALES' SLIDES

# Major Causes of Metabolic Acidosis

**Increased acid production**

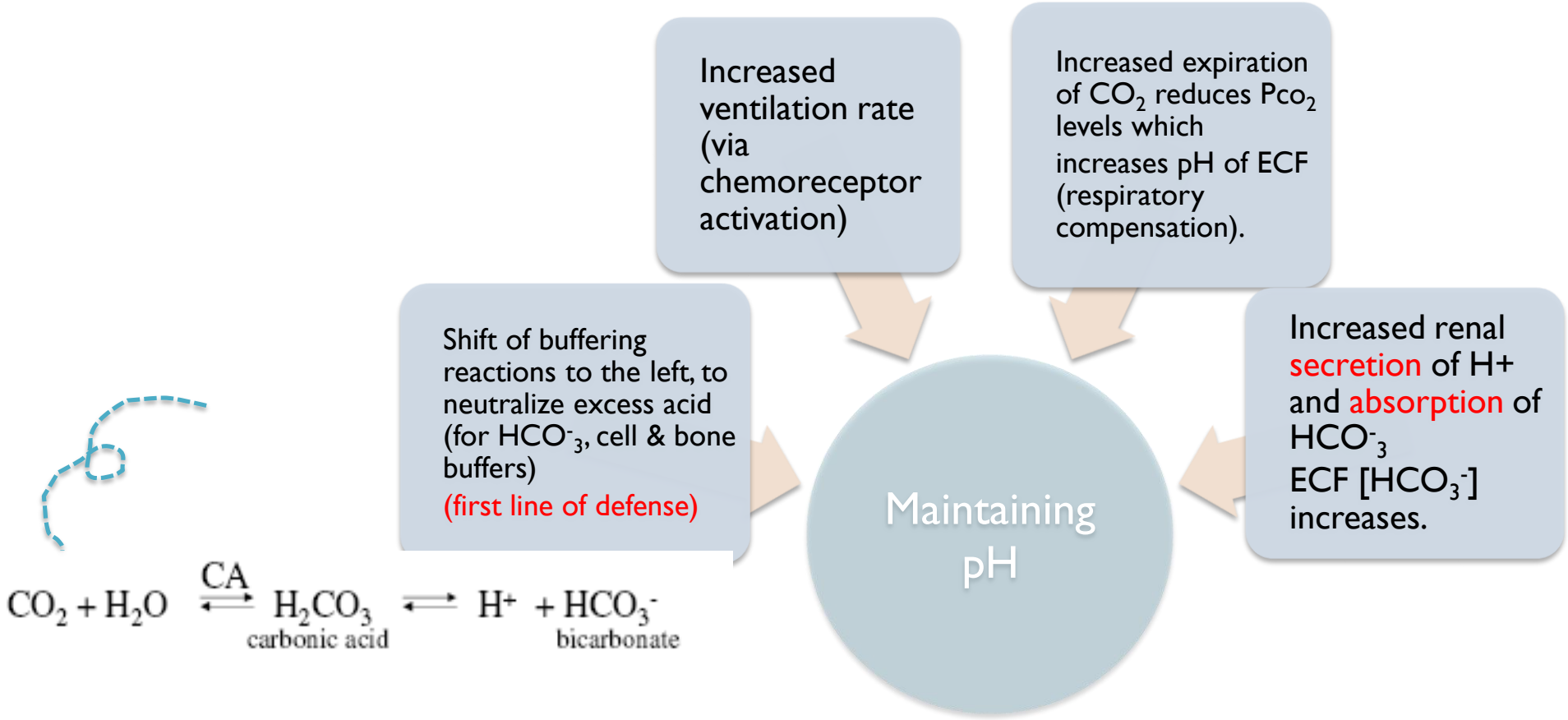
A. **Lactic acidosis**
B. **Ketoacidosis, most often due to uncontrolled diabetes mellitus**
C. **Ingestions**
    1. Aspirin
    2. Ethylene glycol, a component of antifreeze and solvents
    3. Methanol (wood alcohol), a component of shellac and de-icing solutions
D. **Loss of bicarbonate**
    1. Gastrointestinal—diarrhea, pancreatic, biliary or intestinal fistulas, ureterosigmoidostomy
    2. Renal—type 2 (proximal) renal tubular acidosis

**Decreased acid excretion**

A. **Renal failure—decreased $NH_4^+$ excretion**
B. **Type 1 (distal) renal tubular acidosis**
C. **Type 4 renal tubular acidosis (hypoaldosteronism)**

# Metabolic Acidosis

- Metabolic acidosis is characterized by decreased $HCO^-_3$ (<25mM) and pH.






shift to left to decrease H+ and accumulate Co2 to get rid of it in lungs HCO3 will be decreased. After the correction of the kidney it will increase.

on onset of metabolic acidosis, the

# Metabolic Acidosis

For cells: there is uptake of excess H+ & loss of ICF K+ (and Na+) to ECF to maintain Electroneutrality.

So, metabolic acidosis is often associated with increased plasma K+ relative to that expected from state of potassiumbalance.

Hyperkalemia can develop even though body K+ stores are diminished.

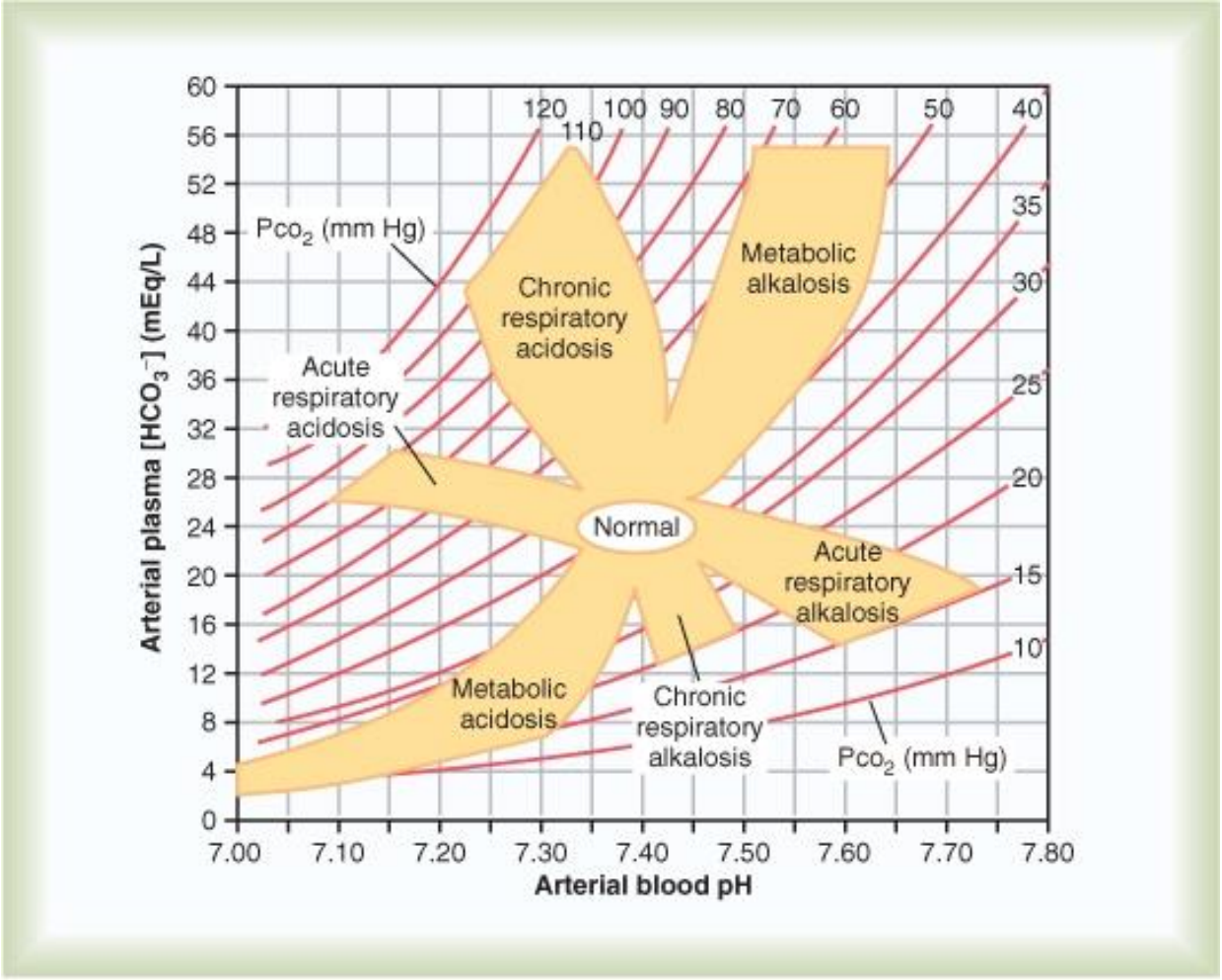
"Cation shift" is reversed with correction of acidosis.

- **Linda corner:**

In ICF, the excess fixed H+ is buffered by organic phosphates and proteins. To utilize these intracel- lular buffers, H+ first must enter the cells. H+ can enter the cells with an organic anion such as ketoan- ion, lactate, or formate, or it can enter the cells in exchange for K+. When the H+ is exchanged for K+, **hyperkalemia** occurs.

# Davenport Diagram Acid-base Alterations

**Metabolic Acidosis**
**↓ plasma pH,**
**↓ plasma [$HCO_3^-$]**
**↓ $Pco_2$,**



# Metabolic Alkalosis

Rare, occur in response to:

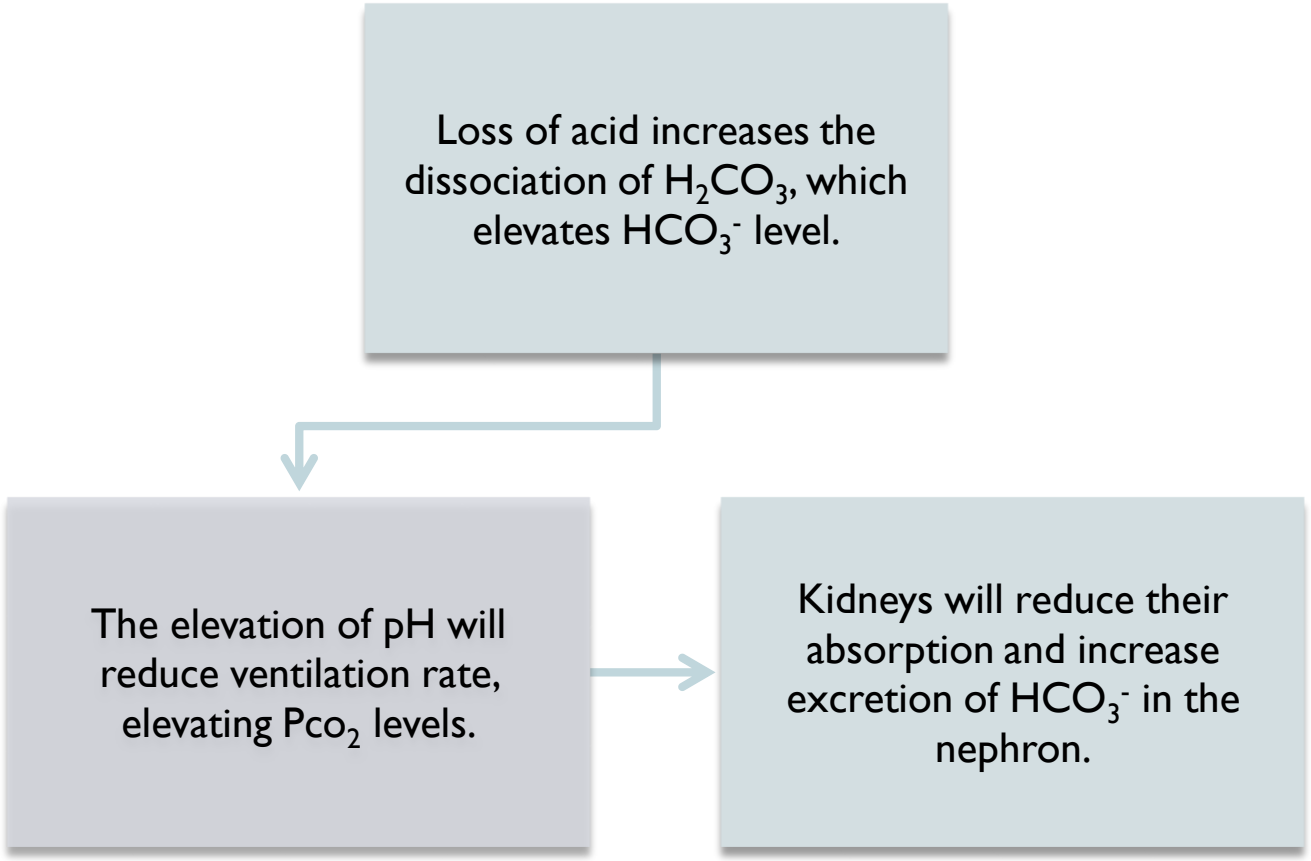
- **Excessive vomiting:** (loss of HCl from stomach which will cause retention of duodenal $HCO_3$ into circulation)
- Excessive ingestion of bicarbonate antacids paired with renal failure.
- Volume contraction via diuretic therapy will increase plasma $HCO_3$
- Excess aldosterone will stimulate collecting duct $H^+$-ATPase to excrete $H^+$
- **Alterations in renal function** "increased excretion of titratable acid..(E.g. thiazide & loop diuretics) will increase Na+ reabsorption..Which eventually increase excretion of H+"

- **Linda corner:**

Loss of fixed acid; The classic example of metabolic alkalosis is vomiting, in which HCl is lost from the stomach. The gastric parietal cells produce H+ and HCO3− from CO2 and H2O. The H+ is secreted with Cl− into the lumen of the stomach to aid in digestion, and the HCO3− enters the blood. In normal persons, the secreted H+ moves from the stomach to the small intestine, where a low pH triggers the
the HCO3 added to blood by the parietal cells is later removed from blood in the pancreatic secretions. However, when vomiting occurs, H+ is lost from the stomach and never reaches the small intestine. HCO3− secretion from the pancreas, therefore, is not stimulated, and the HCO3− remains in the blood, resulting in an increase in HCO3− concentration.

# Metabolic Alkalosis

- Characterized by elevated plasma **$HCO_3$ & PH**




- **Guyton corner :** increased HCO3− concentration in the extracellular fluid increases the filtered load of HCO3−, which, in turn, causes excess HCO3− over H+ secreted in the renal tubular fluid. The excess HCO3− in the tubular fluid fails to be reabsorbed because there is no H+ to react with, and it is excreted in the urine.
- In metabolic alkalosis, the primary compensations are decreased ventilation, which raises Pco2, and increased renal HCO3− excretion, which helps−compensate for the initial rise in extracellular fluid HCO3 concentration.

ONLY IN FEMALES' SLIDES

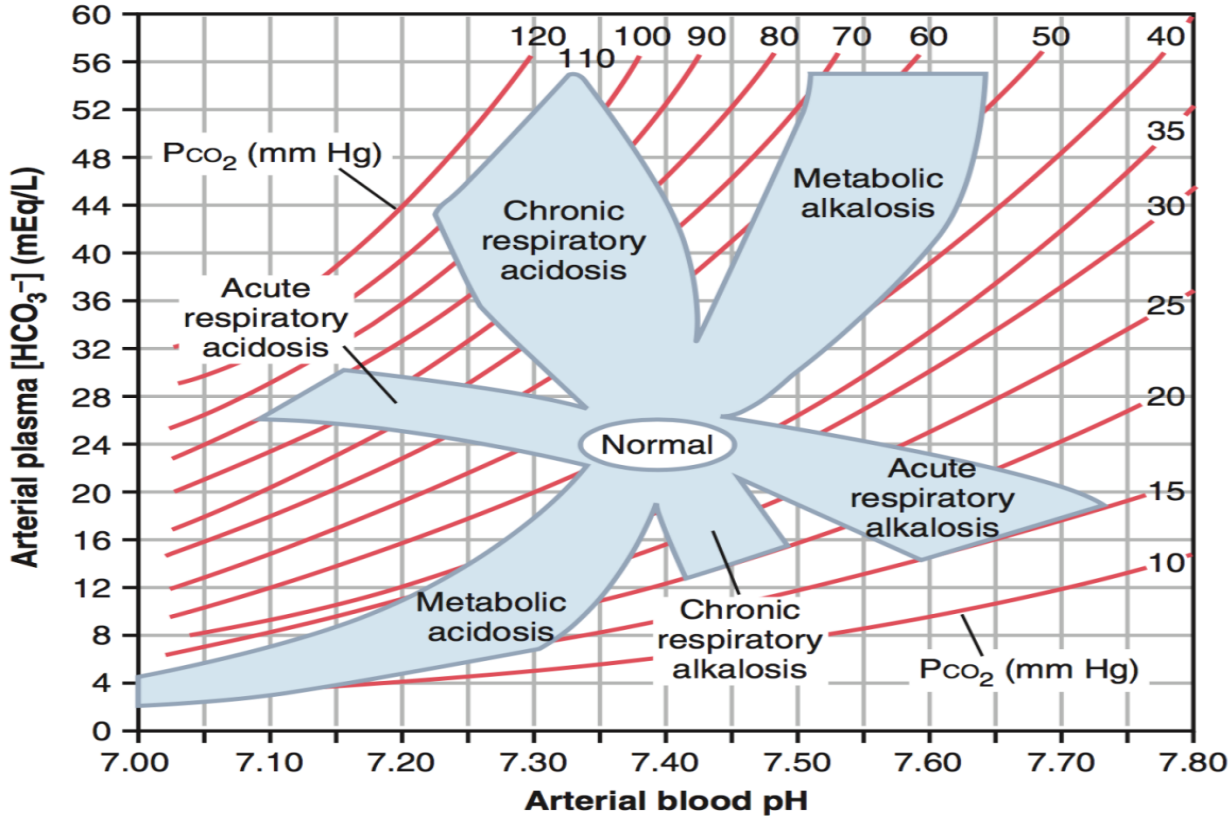
# Major Causes of Metabolic Alkalosis

I. **Hydrogen loss**
   A. **Gastrointestinal loss**
      1. Removal of gastric secretions due to vomiting or nasogastric suction
      2. Antacids in advanced renal failure
   B. **Urinary loss**
      1. Loop or thiazide-type diuretics
      2. Primary mineralocorticoid excess (hyperaldosteronism)
      3. Posthypercapnic alkalosis
      4. Hypercalcemia and milk alkali syndrome
   C. **Movement of $H^+$ into the cells**
      1. Hypokalemia

II. **Administration of bicarbonate or an organic ion that can be metabolized to bicarbonate, such as citrate in blood transfusions**

III. **Contraction alkalosis**
   A. **Loop or thiazide-type diuretics in edematous patients**
   B. **Vomiting or nasogastric suction in achlorhydria**
   C. **Sweat losses in cystic fibrosis**

# Davenport Diagram




- pCo2 is low due to the respiratory compensation
- PCo2 is high due to respiratory compensation by accumulating it

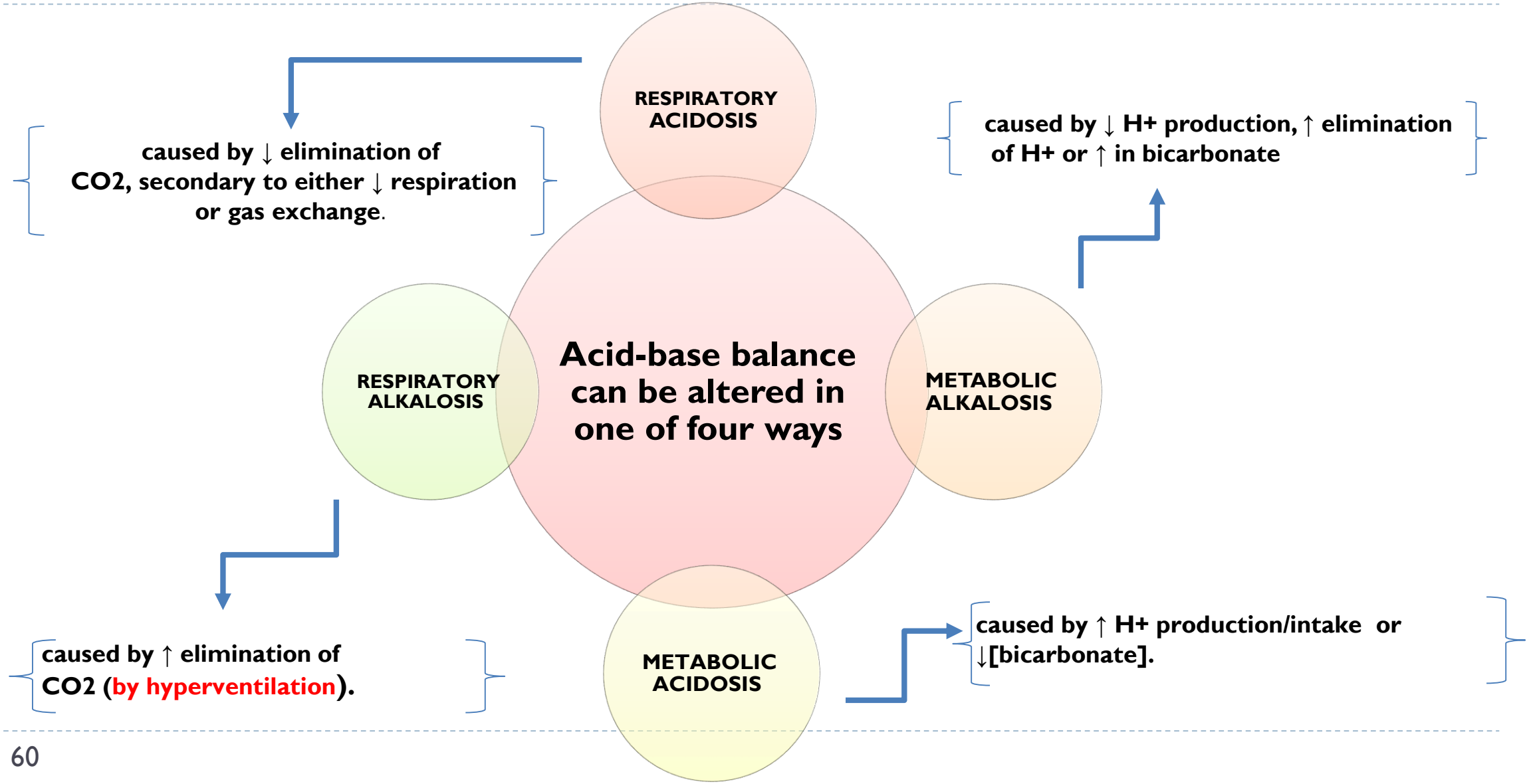
**In Metabolic Acidosis:**

↓ plasma pH
↓ $Pco_2$
↓ plasma $[HCO_3^-]$

**In Metabolic Alkalosis:**

↑ plasma pH
↑ plasma $[HCO_3^-]$
↑ $Pco_2$

# Acid-Base Imbalances (Summary)



# Acid / Base Disorders Summary

Table 28-3. The Four Major Acid-Base Disorders

| Disorder | Proximate Causes | Clinical Causes | Changes in Arterial Acid-Base Parameters |
|---|---|---|---|
| Respiratory acidosis | Increased $P_{CO_2}$ | Decreased alveolar ventilation (e.g., drug overdose)<br>↓ Lung diffusing capacity (e.g., pulmonary edema)<br>Ventilation-perfusion mismatch | pH ↓<br>[$HCO_3^-$] ↑<br>$P_{CO_2}$ ↑ |
| Respiratory alkalosis | Decreased $P_{CO_2}$ | Increased alveolar ventilation caused by:<br>Hypoxia (e.g., acclimatization to high altitude)<br>Anxiety<br>Aspirin intoxication | pH ↑<br>[$HCO_3^-$] ↓<br>$P_{CO_2}$ ↓ |
| Metabolic acidosis | Addition of acids other than $CO_2$ or $H_2CO_3$<br>Removal of alkali (fixed $P_{CO_2}$) | ↓Urinary secretion of $H^+$ (e.g., renal failure)<br>Ketoacidosis (e.g., diabetes mellitus)<br>Lactic acidosis (e.g., shock)<br>$HCO_3^-$ loss (e.g., severe diarrhea) | pH ↓<br>[$HCO_3^-$] ↓<br>$P_{CO_2}$: no change |
| Metabolic alkalosis | Addition of alkali<br>Removal of acids other than $CO_2$ or $H_2CO_3$ (fixed $P_{CO_2}$) | $HCO_3^-$ load (e.g., $NaHCO_3$ therapy)<br>Loss of $H^+$ (e.g., severe vomiting) | pH ↑<br>[$HCO_3^-$] ↑<br>$P_{CO_2}$: no change |

| | pH | $H^+$ | $Pco_2$ | $HCO_3^-$ |
|---|---|---|---|---|
| Normal | 7.4 | 40 mEq/L | 40 mm Hg | 24 mEq/L |
| Respiratory acidosis | ↓ | ↑ | ↑↑ | ↑ |
| Respiratory alkalosis | ↑ | ↓ | ↓↓ | ↓ |
| Metabolic acidosis | ↓ | ↑ | ↓ | ↓↓ |
| Metabolic alkalosis | ↑ | ↓ | ↑ | ↑↑ |

# Primary and compensatory changes in different acid-base disorders

**Table 5.2 Primary and Compensatory Changes in Different Acid–Base Disorders**

| Disorder | Primary Change | Compensatory Response |
|---|---|---|
| Metabolic acidosis | Fall in plasma bicarbonate concentration | Reduction in $PCO_2$ averaging 1.2 mm Hg per 1-mEq/L reduction in plasma bicarbonate concentration |
| Metabolic alkalosis | Rise in plasma bicarbonate concentration | Elevation in $PCO_2$ averaging 0.6–0.7 mm Hg per 1-mEq/L rise in plasma bicarbonate concentration |
| Respiratory acidosis | Elevation in $PCO_2$ | **Acute**: Rise in plasma bicarbonate concentration averaging 1 mEq/L per 10 mm Hg elevation in $PCO_2$<br>**Chronic**: Increase in plasma bicarbonate concentration averaging 3.5 mEq/L per 10 mm Hg rise in $PCO_2$ |
| Respiratory alkalosis | Reduction in $PCO_2$ | **Acute**: Fall in plasma bicarbonate concentration averaging 2 mEq/L per 10 mm Hg decline in $PCO_2$<br>**Chronic**: Fall in plasma bicarbonate concentration averaging 4 mEq/L per 10 mm Hg decline in $PCO_2$ |

**Table 7–2** Summary of Acid-Base Disorders

| Disorder | $CO_2 + H_2O$ | ↔ | $H^+$ | + | $HCO_3^-$ | Respiratory Compensation | Renal Compensation or Correction |
|---|---|---|---|---|---|---|---|
| Metabolic Acidosis | ↓ | | ↑ | | **↓** | Hyperventilation | ↑ $HCO_3^-$ reabsorption (correction) |
| Metabolic Alkalosis | ↑ | | ↓ | | **↑** | Hypoventilation | ↑ $HCO_3^-$ excretion (correction) |
| Respiratory Acidosis | **↑** | | ↑ | | ↑ | None | ↑ $HCO_3^-$ reabsorption (compensation) |
| Respiratory Alkalosis | **↓** | | ↓ | | ↓ | None | ↓ $HCO_3^-$ reabsorption (compensation) |

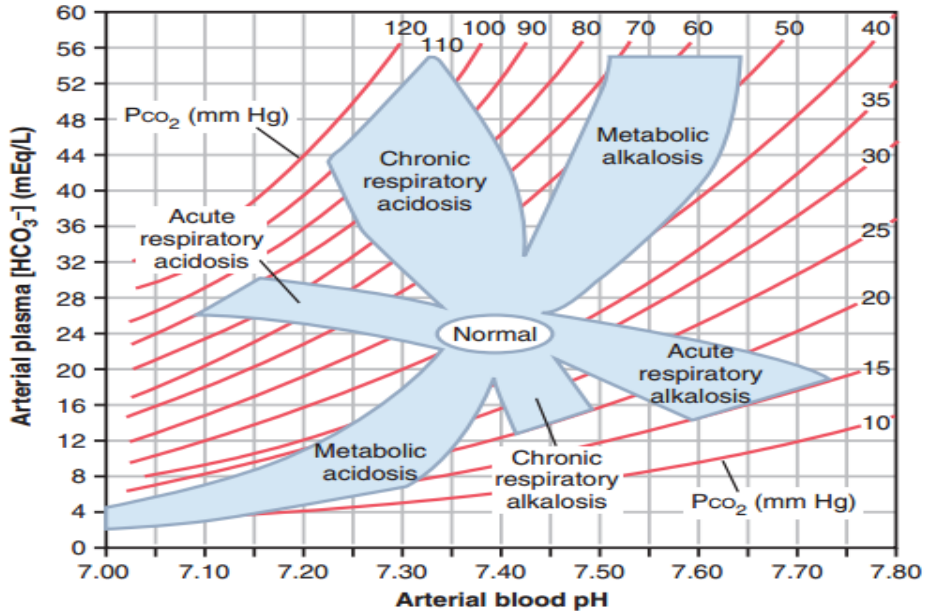
*Bold arrows* indicate initial disturbance.

# Analysis of Acid-Base Disorders

- Analysis aimed at identifying underlying cause of disorder such that appropriate therapy can be initiated.
- In addition to usual history taking and physical findings, sampling of arterial blood can yield valuable information.
- Analysis of blood sample data is straightforward if approached systematically either using the Davenport nomogram or flow diagram.

- **Guyton corner :**

A convenient way to diagnose acid-base disorders is to use an acid-base nomogram, as shown in the Figure. This diagram can be used to determine the type of acidosis or alkalosis, as well as its severity. In this acid-base diagram, pH, HCO3−concentration, and PCO2 values intersect according to the Henderson-Hasselbalch equation. The central open circle shows normal values and the deviations that can still be considered within the normal range. The shaded areas of the diagram show the 95 percent confidence limits for the normal compensations to simple metabolic and respiratory disorders



# How to Analyze an ABG

| | PO2 | pH | PCO2 | HCO3 |
|---|---|---|---|---|
| normal | 80-100mmHg | 7.35_7.45 | 35-45 mmHg | 22-26 mmol/L |
| acidotic | - | <7.35 | >45 | < 22 |
| alkalotic | - | >7.45 | <35 | > 26 |

- **Guyton corner: (Table 31-3)** summarizes the characteristics associated with respiratory and metabolic acidosis, as well as respiratory and metabolic alkalosis, which are discussed in the next section. Note that in respiratory acidosis, there is a reduction in pH, an increase in extracellular fluid H+ concentration, and an increase in PCO2, which is the initial cause of the acidosis. The compensatory response is an increase in plasma HCO3−, caused by the addition of new HCO3−to the extracellular fluid by the kidneys.The rise in HCO3− helps offset the increase in PCO2 , thereby returning the plasma pH toward normal. In metabolic acidosis, there is also a decrease in pH and a rise in extracellular fluid H+ concentration. However, in this case, the primary abnormality is a decrease in plasma HCO3−. The primary compensations include increased ventilation rate, which reduces PCO2, and renal compensation, which, by adding new HCO3−to the extracellular fluid, helps minimize the initial fall in extracellular HCO3−concentration.

**Table 31-3** Characteristics of Primary Acid-Base Disturbances

| | pH | $H^+$ | $P_{CO_2}$ | $HCO_3^-$ |
|---|---|---|---|---|
| Normal | 7.4 | 40 mEq/L | 40 mm Hg | 24 mEq/L |
| Respiratory acidosis | ↓ | ↑ | ↑↑ | ↑ |
| Respiratory alkalosis | ↑ | ↓ | ↓↓ | ↓ |
| Metabolic acidosis | ↓ | ↑ | ↓ | ↓↓ |
| Metabolic alkalosis | ↑ | ↓ | ↑ | ↑↑ |

# Analysis of Acid-Base Disorders

Example :

- pH = 7.35
- [HCO3-] = 16mEq/L
- PCO2 = 30 mm Hg

1)Evaluate pH - acid

2) Metabolic or respiratory source?
[HCO3-] < 24mM = metabolic

3) Analysis of compensatory response.
↓ PCO2 – respiratory compensation

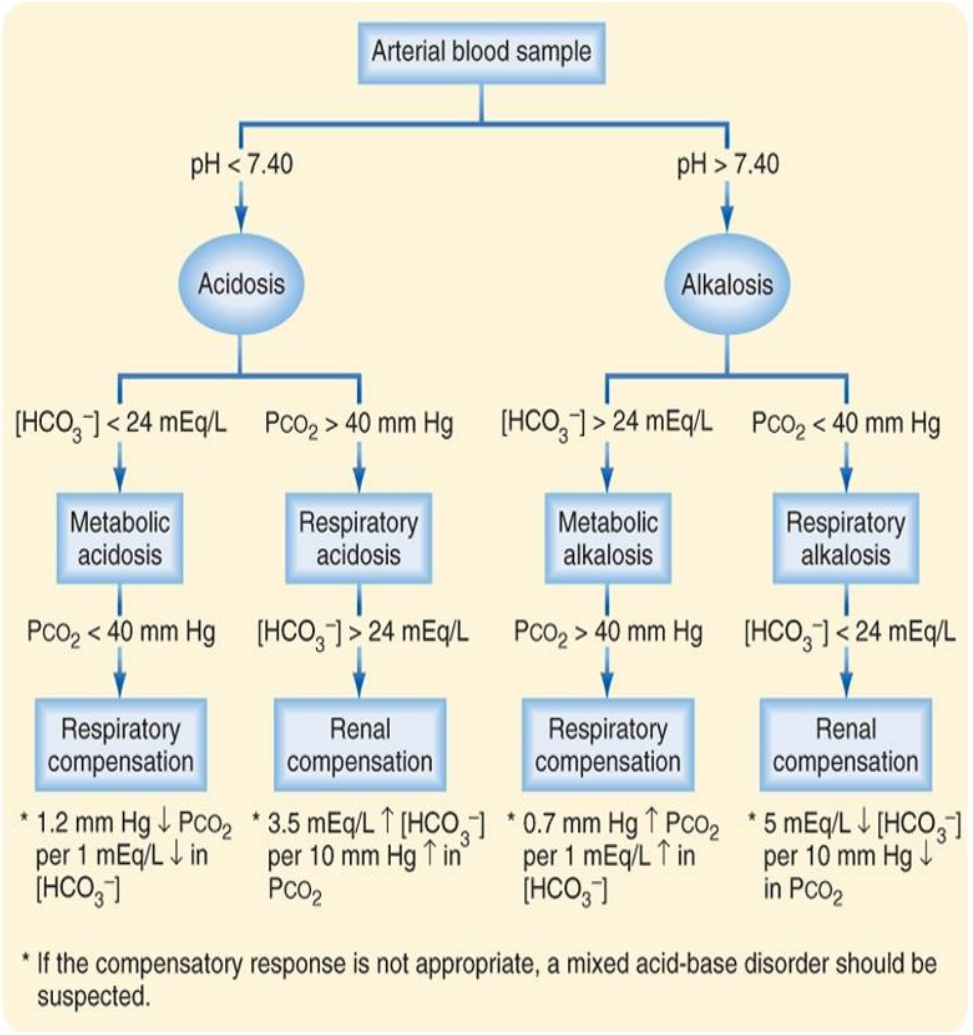
Mixed acid-base disorders can also occur (e.g. emphysema with diarrhea) in which an appropriate compensatory response has not occurred.

الخطوات بسيطة:

١- ناظر ال ph

٢-ناظر pco2 عشان تعرف (ميتابولك ولا ريسبايراتوري)

٣- ناظر hco3 عشان تعرف compensatedولا لا او هيا ميتابولك اسيدوسز ولا الكلوسز






The normal range of pH [7.35-7.45] it depends on the oxygenation in venous blood it is 7.35 (slightly acidic, why?) there are more CO2 in the arterial blood it is 7.4

# Cases:

Normal PCO2= 35-45 mmHg
Normal (HCO3-) = 33-28 mEq/L

- Case 1 :
- A patient known to have COPD presented with 3-day history of fever, SOB, and cough productive of yellowish sputum. His ABGs showed:
- pH = 7.25
- PCO2 = 80 mmHg.
- HCO3- = 34 mEq/L.

Answer:
Acidosis
PCO= matches PH
HCO3= this a result of compensatory mechanism

3 days HISTORY!!
Compensating mechanism is incomplete.

- Case 2:
- A 21 year old man with IDDM presents to ER with mental status changes, nausea, vomiting, abdominal pain and rapid respirations. His ABGs showed:
- pH = 7.2
- PCO2 = 20 mmHg
- HCO3 = 8 mEq/l

Answer:
PH= Acidosis
PCO2= low, matches PH
HCO3 = low, doesn't match PH

Respiratory compensating

- Case 3:
- A 2-year old child who is lethargic and dehydrated has a 3-day history of vomiting. His ABGs showed:
- pH = 7.56
- PCO2 = 44 mmHg
- HCO3- = 37 mEq/l

Answer:
Alkalosis
PCO2= Normal
HCO3- = High

Uncompensated metabolic alkalosis

# Cases:

- Case 4:
- A 20-year old student suffered a panic attack while awaiting an exam. Her ABGs showed:
- pH = 7.6
- PCO2 = 24 mmHg.
- HCO3 = 23 mEq/L.

Answer:
Alkalosis
PCO2= Low
HCO3 = Normal

Uncompensated Respiratory alkalosis

- Case 5:
- A 69 year old patient had a cardiac arrest soon after return to the ward following an operation. Resuscitation was commenced and included intubation and ventilation. Femoral arterial blood gases were collected about five minutes after the arrest. Other results: Anion gap 24, Lactate 12 mmol/l.
- • pH 6.85
- • pCO2 82 mmHg
- • HCO3 14 mmol/l

Ignore anion gap

Answer:
Severe acidosis
PCO2= High, matching
HCO3- = Low, matching

Mixed acid base disorder

# Thank you!

اعمل لترسم بسمة، اعمل لتمسح دمعة، اعمل و أنت تعلم أن الله لا يضيع أجر من أحسن عملا.

## The Physiology 436 Team:

Female Members:
Amal AlQarni
Heba Alnasser
Deena AlNowiser
Reema Alotaibi
Laila Mathkour
Ghada Alskait
Ghada Alhadlaq

Male Members:
Mohammed Alayed
Mohammad Almutlaq
Mohammed Baqais
Mohammad Naser
Abdullah Abuamarah

**Team Leaders:**
Qaiss Almuhaideb
Lulwah Alshiha

**Contact us:**
Physiology436@gmail.com
@Physiology436

Link to Editing File

**Special thanks to Team435's Leaders: Meshal Alhazmy & Khawla Alammari and members!**

References:
- Girls' and boys' slides.
- 435 Team.
- Guyton and Hall Textbook of Medical Physiology (13th Edition).
- Linda (5th Edition).