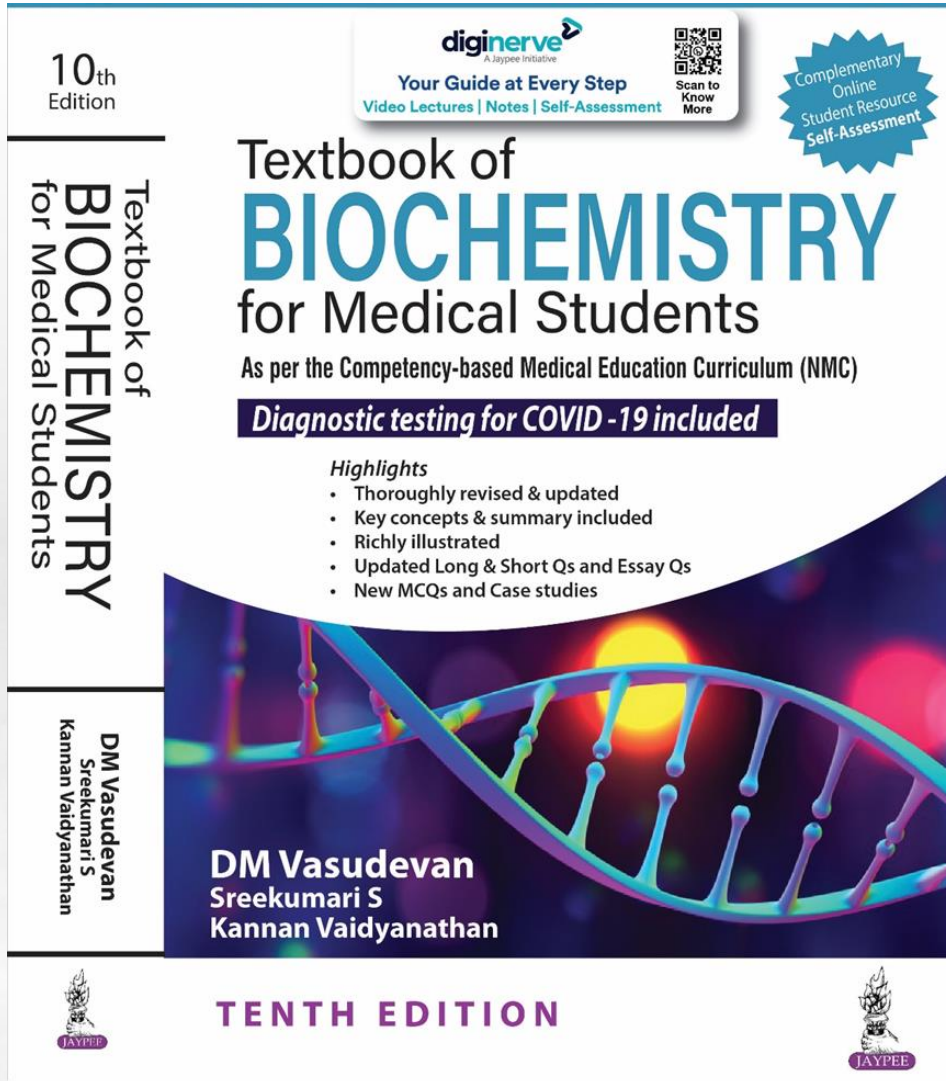


Chapter 24A:

Acid Base Balance and pH Basic Principles

Textbook of
BIOCHEMISTRY
for Medical Students
By DM Vasudevan, *et al.*

TENTH EDITION

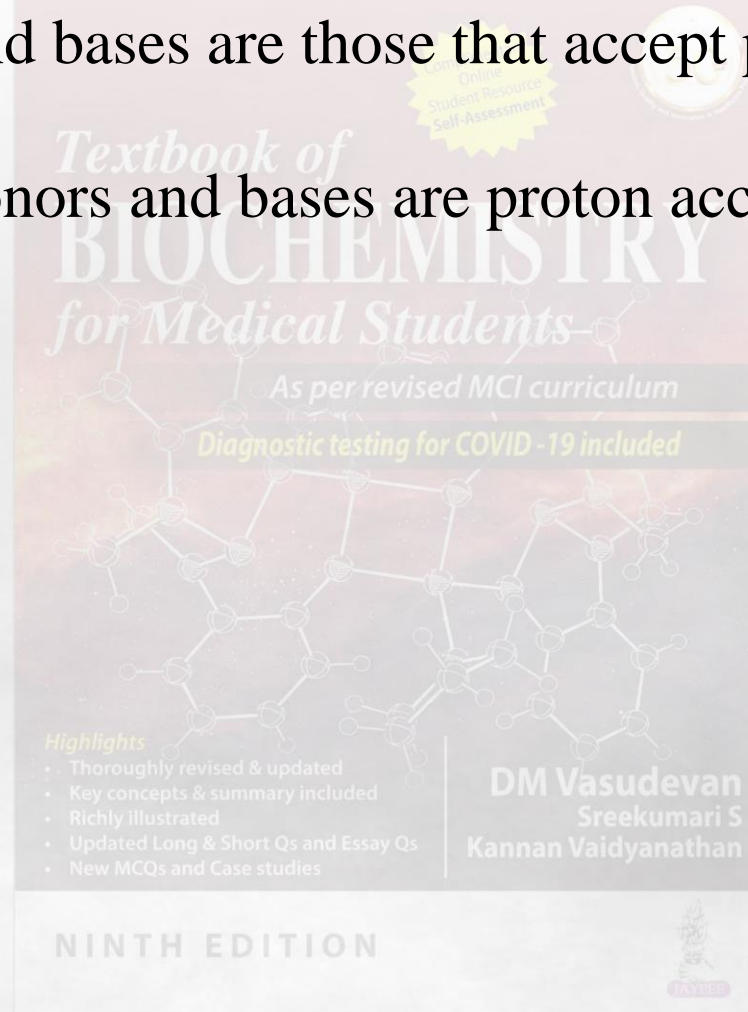


Acids and Bases Definition



According to Bronsted, acids are substances that are capable of donating protons and bases are those that accept protons.

Acids are proton donors and bases are proton acceptors.



Examples of Acids and Bases



Acids	Bases
$HA \leftrightarrow H^+ + A^-$	$NH_3 + H^+ \leftrightarrow NH_4^+$
$HCl \leftrightarrow H^+ + Cl^-$	$HCO_3^- + H^+ \leftrightarrow H_2CO_3$
$H_2CO_3 \leftrightarrow H^+ + HCO_3^-$	

Highlights

- Thoroughly revised & updated
- Key concepts & summary included
- Richly illustrated
- Updated Long & Short Qs and Essay Qs
- New MCQs and Case studies

DM Vasudevan
Sreekumari S
Kannan Vaidyanathan

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Terms Explained



Term	Definition and explanations
pH	Negative logarithm of hydrogen ion concentration. Normal value 7.4 (range 7.38 -7.42)
Acids	Proton donors; pH <7
Bases	Proton acceptors; pH > 7
Strong acids	Acids which ionize completely; e.g. HCl
Weak acids	Acids which ionize incompletely e.g. H ₂ CO ₃
pK value	pH at which the acid is half ionized; Salt : Acid = 1:1
Alkali reserve	Bicarbonate available to neutralise acids; Normal 24 mmol/L (range 22-26 mmol/L)
Buffers	Solutions minimise changes in pH

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Weak and Strong Acids the Extent of Dissociation

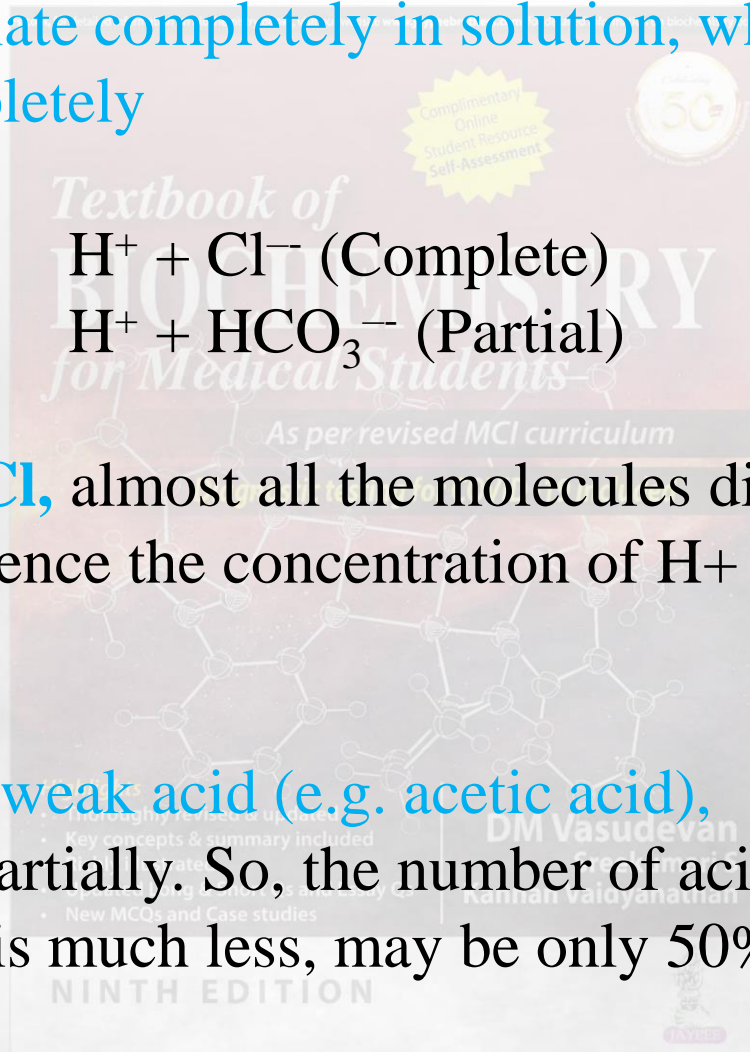


Strong acids dissociate completely in solution, while weak acids ionize incompletely



In a solution of HCl, almost all the molecules dissociate and exist as H^+ and Cl^- ions. Hence the concentration of H^+ is very high and it is a strong acid.

But in the case of a weak acid (e.g. acetic acid), it will ionize only partially. So, the number of acid molecules existing in the ionized state is much less, may be only 50%.



Dissociation Constant



Dissociation of an acid is a freely reversible reaction. At equilibrium the ratio between dissociated and undissociated particle is a constant.

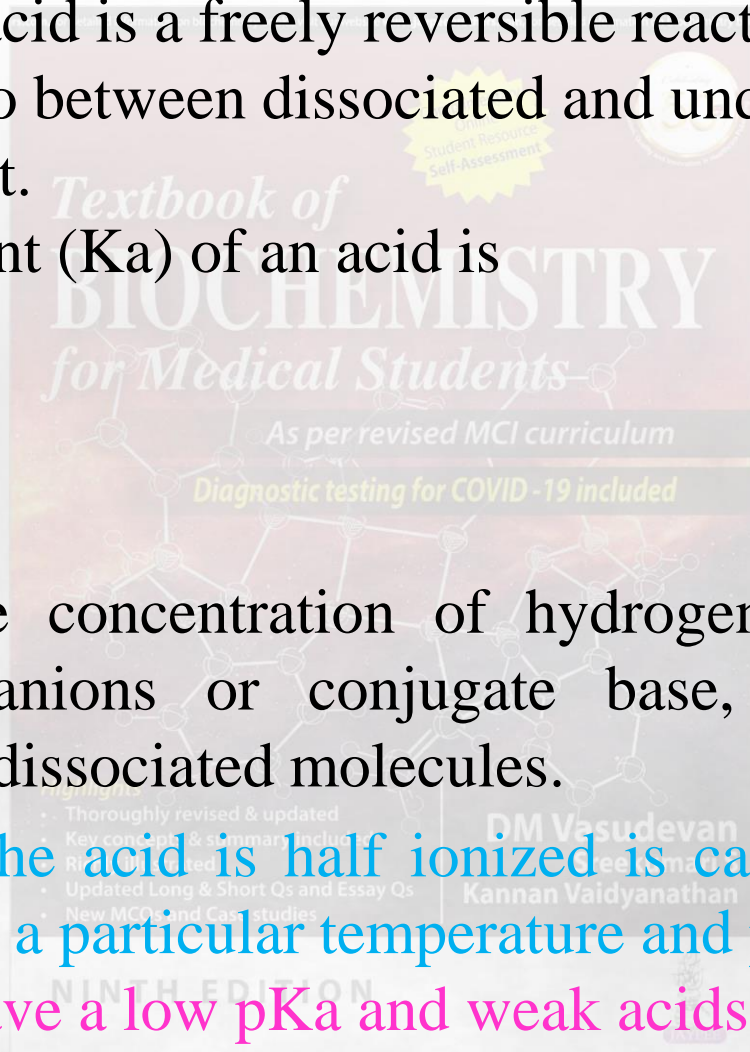
Dissociation constant (K_a) of an acid is

$$K_a = \frac{[H^+][A^-]}{[HA]}$$

where $[H^+]$ is the concentration of hydrogen ions, $[A^-]$ = the concentration of anions or conjugate base, and $[HA]$ is the concentration of undissociated molecules.

The pH at which the acid is half ionized is called pKa of an acid which is constant at a particular temperature and pressure.

Strong acids will have a low pKa and weak acids have a higher pKa.



Acidity of a Solution and pH



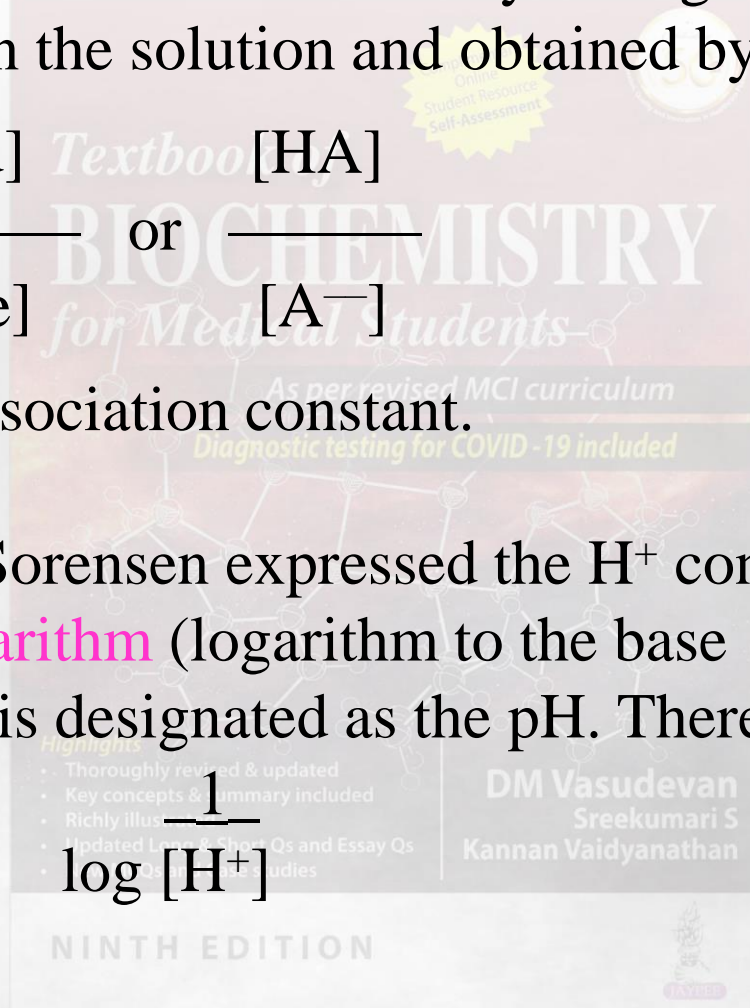
The acidity of a solution is measured by noting the hydrogen ion concentration in the solution and obtained by the equation.

$$[H^+] = K_a \frac{[\text{acid}]}{[\text{base}]} \quad \text{or} \quad \frac{[HA]}{[A^-]}$$

where K_a is the dissociation constant.

To make it easier, Sorensen expressed the H^+ concentration as the **negative of the logarithm** (logarithm to the base 10) of hydrogen ion concentration, and is designated as the pH. Therefore,

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



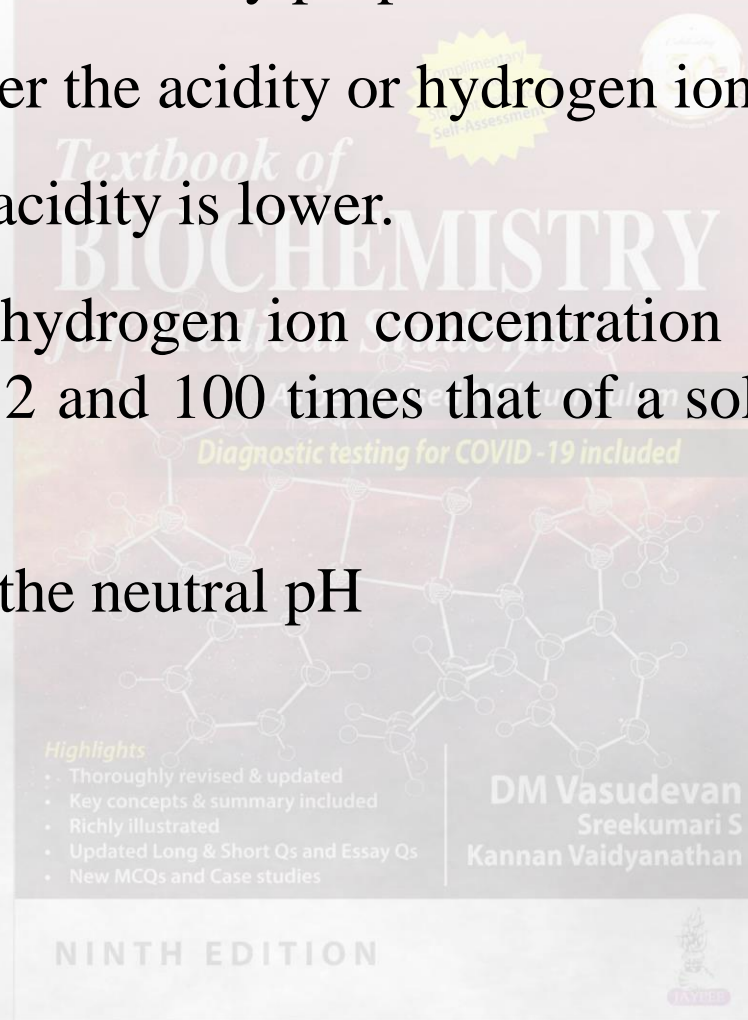
Thus the pH value is inversely proportional to the acidity.

Lower the pH, higher the acidity or hydrogen ion concentration.

Higher the pH, the acidity is lower.

At a pH of 1, the hydrogen ion concentration is 10 times that of a solution with a pH 2 and 100 times that of a solution with a pH of 3 and so on.

The pH 7 indicates the neutral pH



Relation Between Hydrogen Ions, Hydroxyl Ions and pH of Aqueous Solutions



Ionic product of water = $[H^+][OH^-] = 10^{-14}$

$[OH^-]$ mol/liter	$[H^+]$ mol/liter	log $[H^+]$	$-\log[H^+]$ =pH	pOH	Inference
1×10^{-13}	1×10^{-1}	-1	1	13	Strong acid
1×10^{-10}	1×10^{-4}	-4	4	10	Acid
1×10^{-7}	1×10^{-7}	-7	7	7	Neutral
1×10^{-4}	1×10^{-10}	-10	10	4	Alkali
1×10^{-1}	1×10^{-13}	-13	13	1	Strong alkali

The Effect of Salt upon the Dissociation



The relationship between pH, pKa, concentration of acid and conjugate base (or salt) is expressed by the

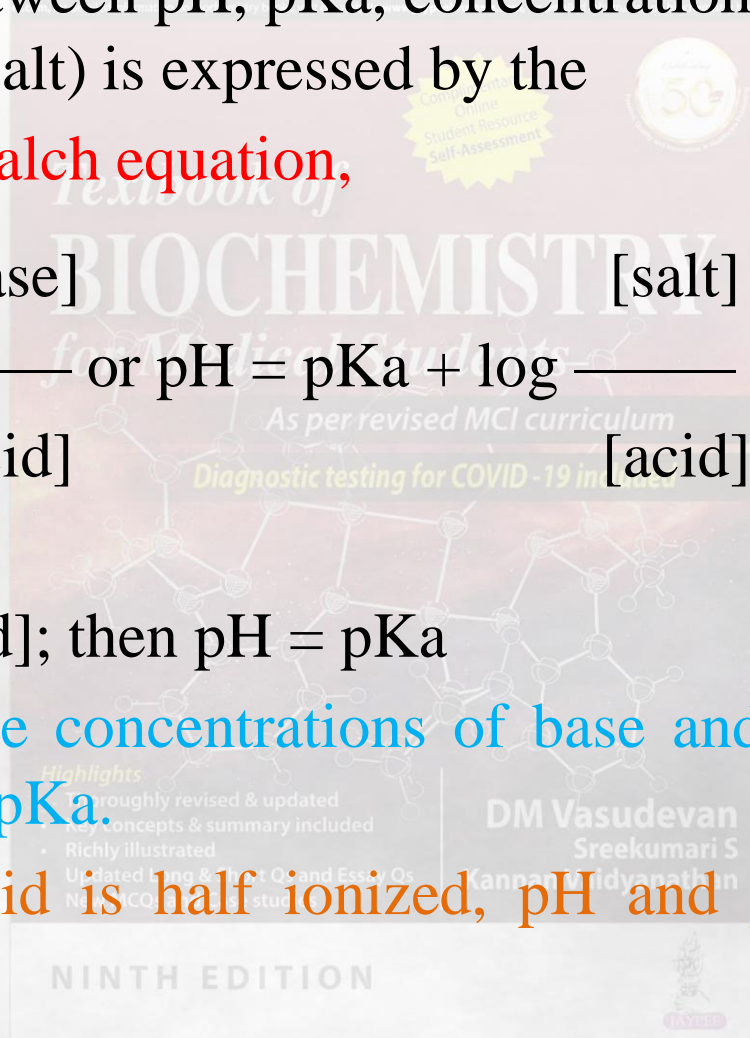
Henderson-Hasselbalch equation,

$$\text{pH} = \text{pKa} + \log \frac{[\text{base}]}{[\text{acid}]} \quad \text{or} \quad \text{pH} = \text{pKa} + \log \frac{[\text{salt}]}{[\text{acid}]}$$

When [base] = [acid]; then pH = pKa

Therefore, when the concentrations of base and acid are the same, then pH is equal to pKa.

Thus, when the acid is half ionized, pH and pKa have the same values.



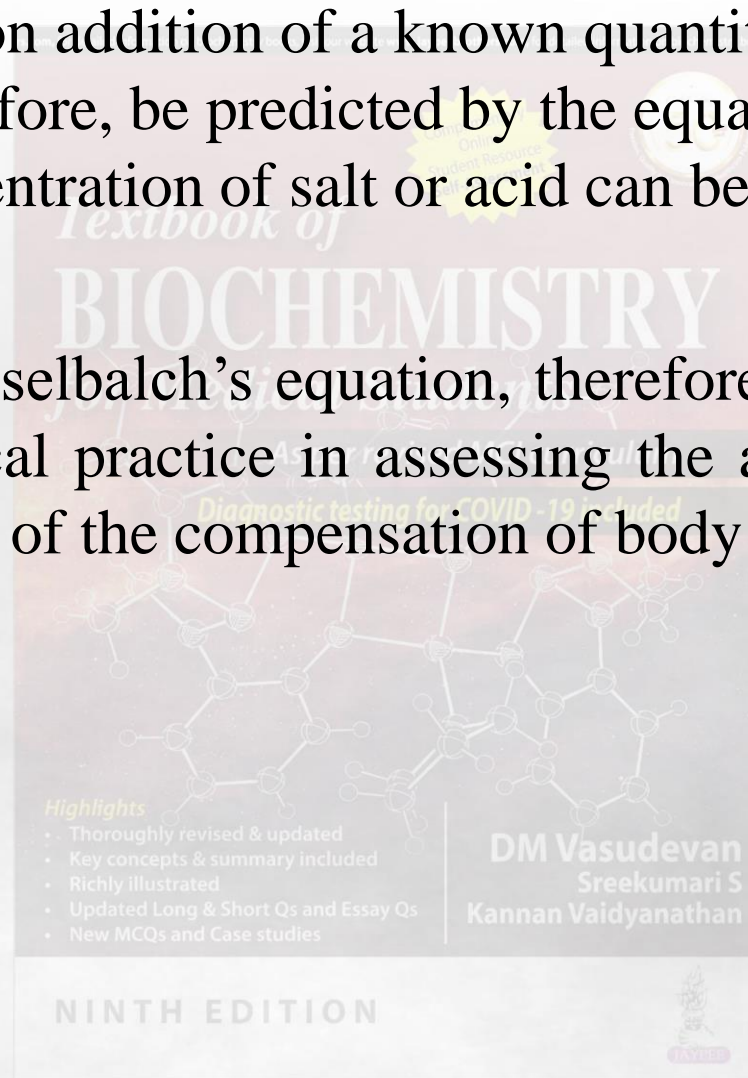
Clinical Application of Henderson-Hasselbalch's Equation



The pH of a buffer on addition of a known quantity of acid and alkali can, therefore, be predicted by the equation.

Moreover, the concentration of salt or acid can be found out by measuring the pH.

The Henderson-Hasselbalch's equation, therefore, has great practical application in clinical practice in assessing the acid-base status, and predicting the limits of the compensation of body buffers.



Buffers



Buffers are solutions which can resist changes in pH when acid or alkali is added.

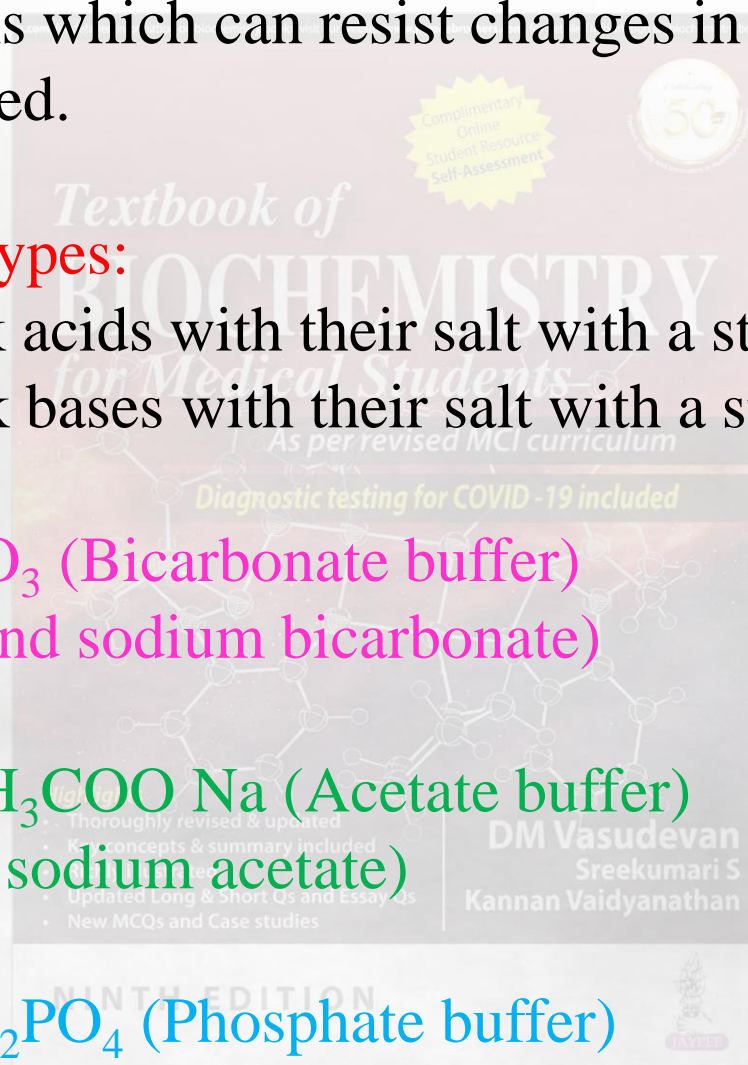
Buffers are of two types:

- a. Mixtures of weak acids with their salt with a strong base or
- b. Mixtures of weak bases with their salt with a strong acid.

i. $\text{H}_2\text{CO}_3 / \text{NaHCO}_3$ (Bicarbonate buffer)
(carbonic acid and sodium bicarbonate)

ii. $\text{CH}_3\text{COOH} / \text{CH}_3\text{COO Na}$ (Acetate buffer)
(acetic acid and sodium acetate)

iii. $\text{Na}_2\text{HPO}_4 / \text{NaH}_2\text{PO}_4$ (Phosphate buffer)



Factors Affecting pH of a Buffer

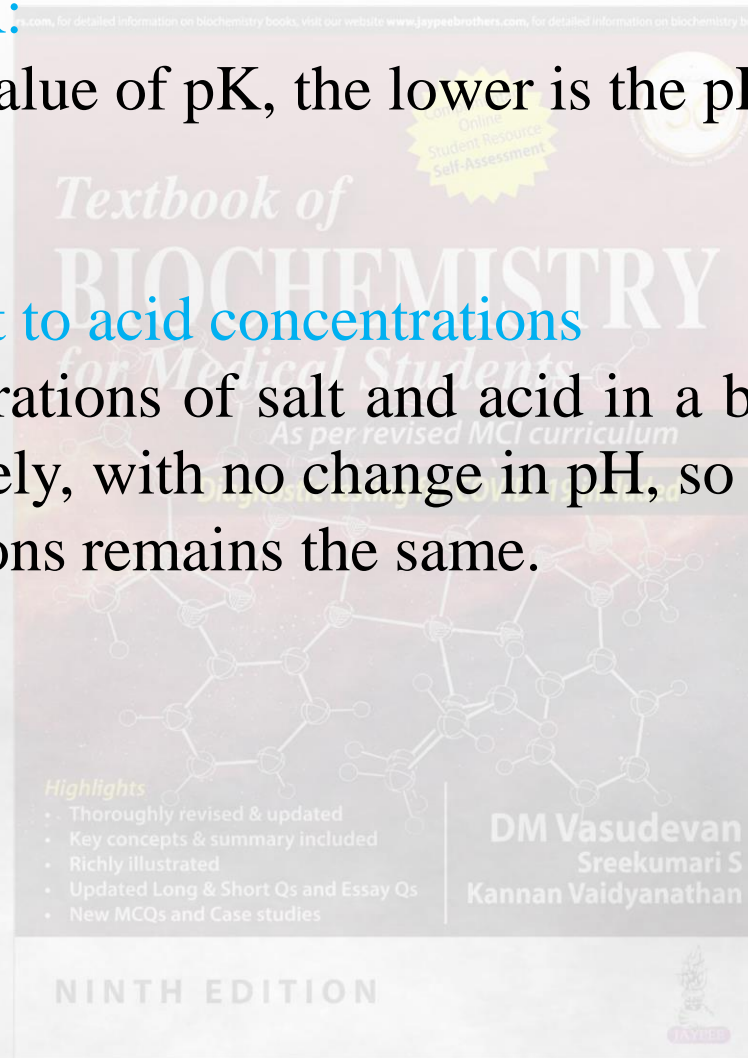


a. The value of pK :

The lower the value of pK , the lower is the pH of the solution.

b. The ratio of salt to acid concentrations

Actual concentrations of salt and acid in a buffer solution may be varying widely, with no change in pH, so long as the ratio of the concentrations remains the same.



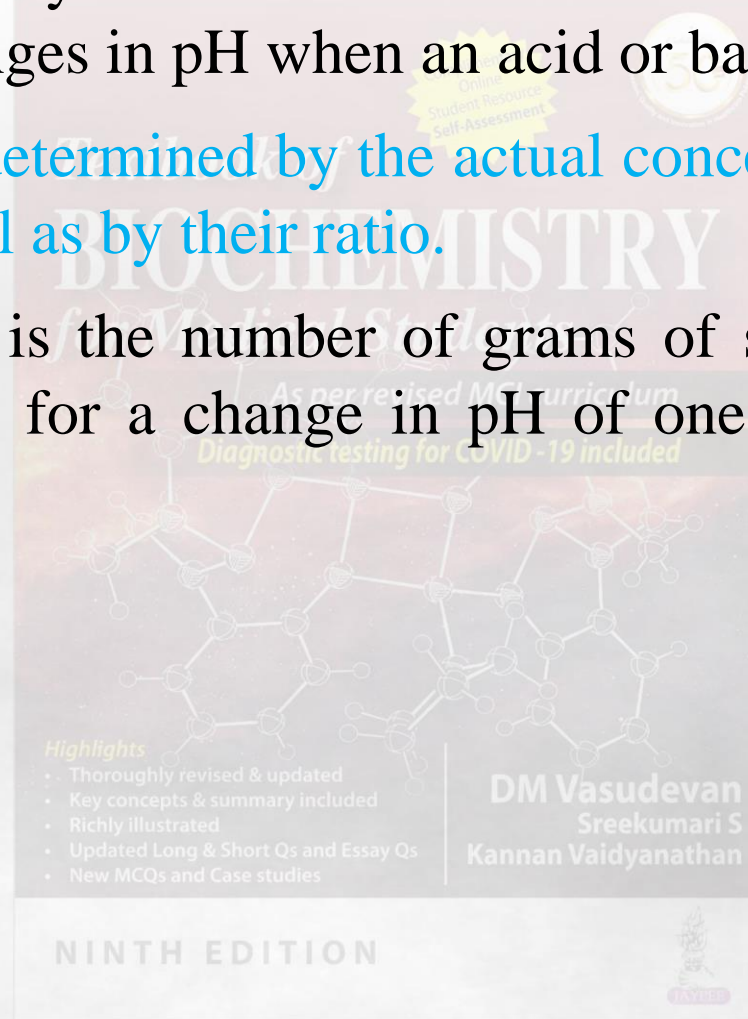
Factors Affecting Buffer Capacity



The buffering capacity of a buffer is defined as the ability of the buffer to resist changes in pH when an acid or base is added.

Buffer capacity is determined by the actual concentrations of salt and acid present, as well as by their ratio.

Buffering capacity is the number of grams of strong acid or alkali which is necessary for a change in pH of one unit of one liter of buffer solution.



How do Buffers Act?



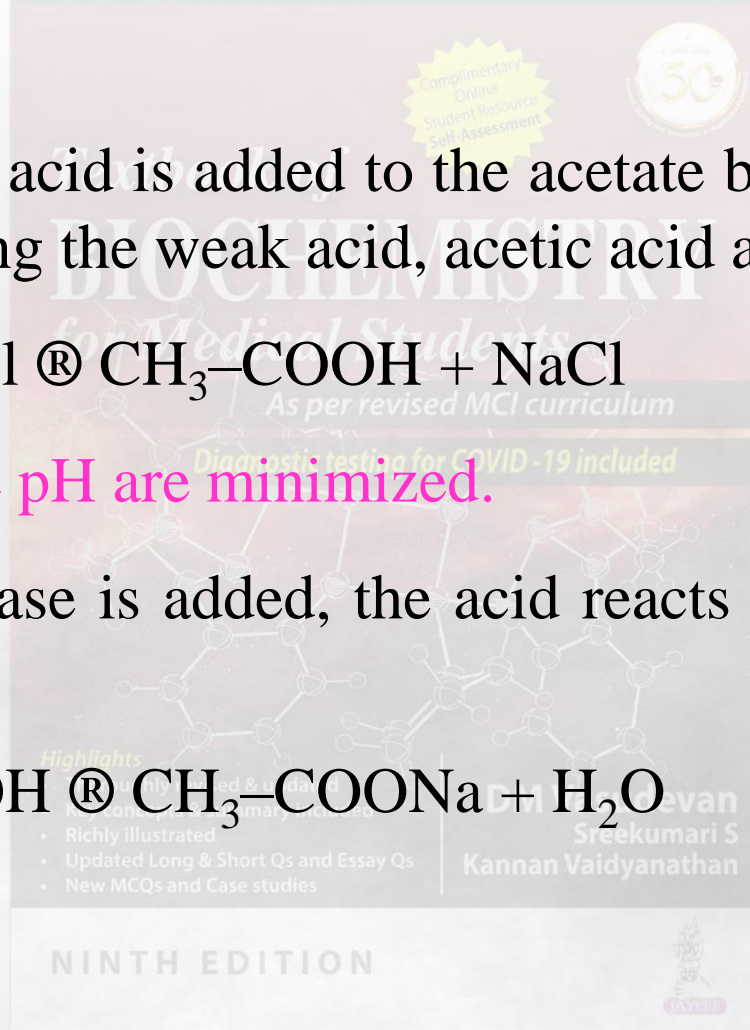
Buffer solutions consist of mixtures of a weak acid or base and its salt.

When hydrochloric acid is added to the acetate buffer, the salt reacts with the acid forming the weak acid, acetic acid and its salt.



Thus changes in the pH are minimized.

Similarly when a base is added, the acid reacts with it forming salt and water.



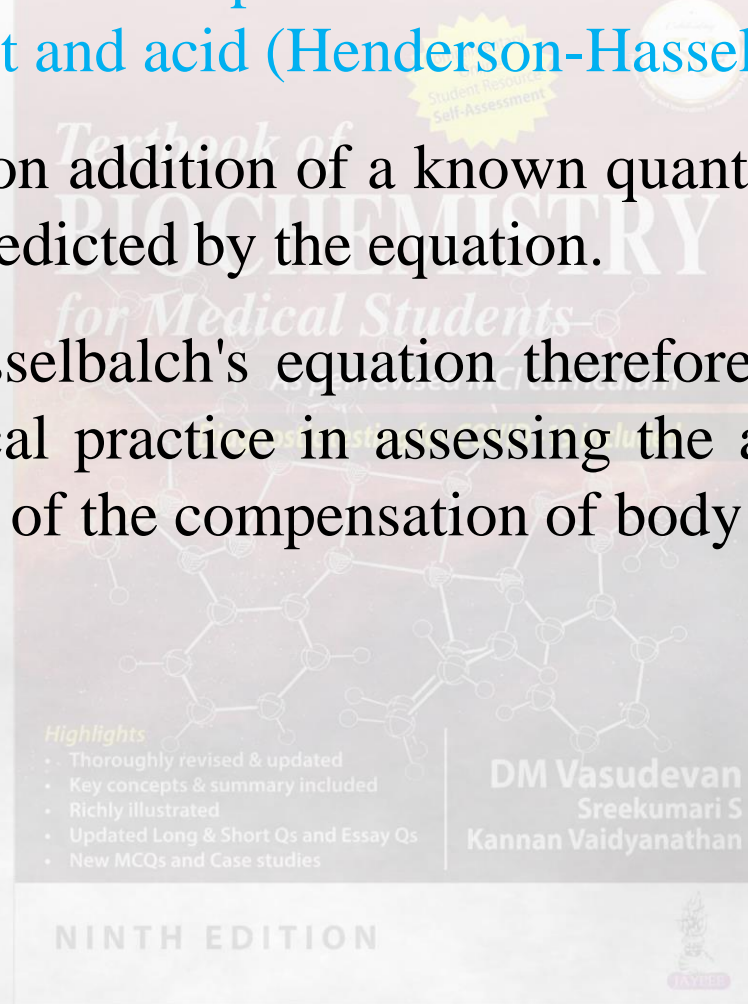
The Buffer Capacity is Determined by the Absolute Concentration of the Salt and Acid



But the pH of the buffer is dependent on the relative proportion of the salt and acid (Henderson-Hasselbalch's equation).

The pH of a buffer on addition of a known quantity of acid and alkali can, therefore, be predicted by the equation.

The Henderson-Hasselbalch's equation therefore, has great practical application in clinical practice in assessing the acid-base status, and predicting the limits of the compensation of body buffers.



Effective Range of a Buffer

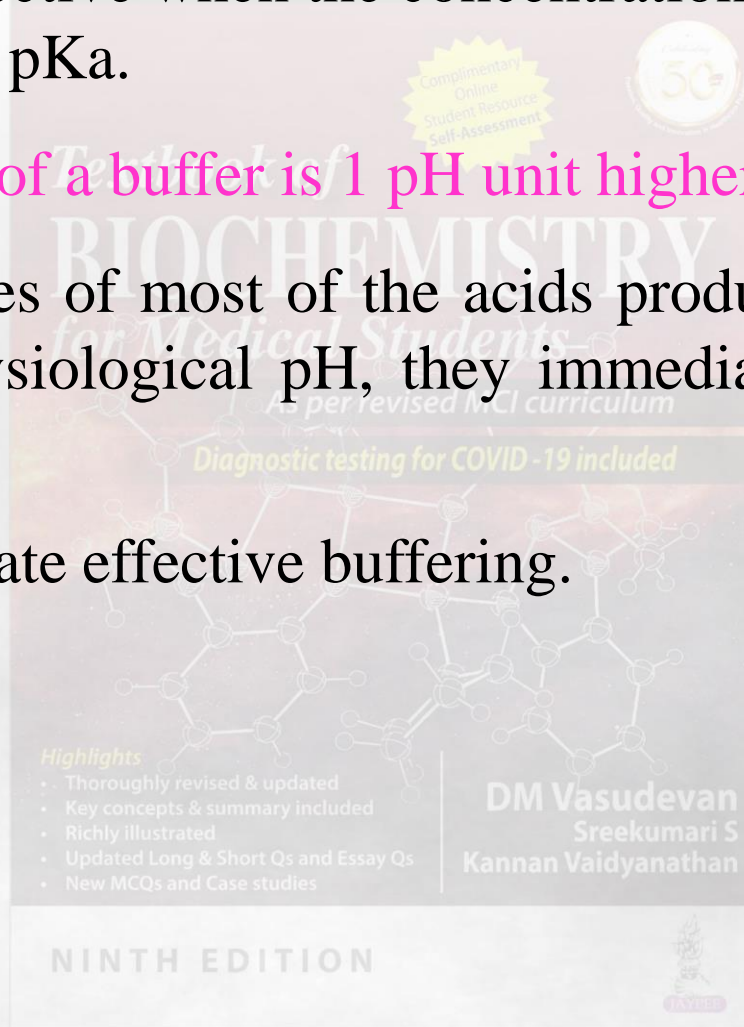


A buffer is most effective when the concentrations of salt and acid are equal or when $\text{pH} = \text{pK}_a$.

The effective range of a buffer is 1 pH unit higher or lower than pK_a .

Since the pK_a values of most of the acids produced in the body are well below the physiological pH, they immediately ionize and add H^+ to the medium.

This would necessitate effective buffering.



Acid-base Balance

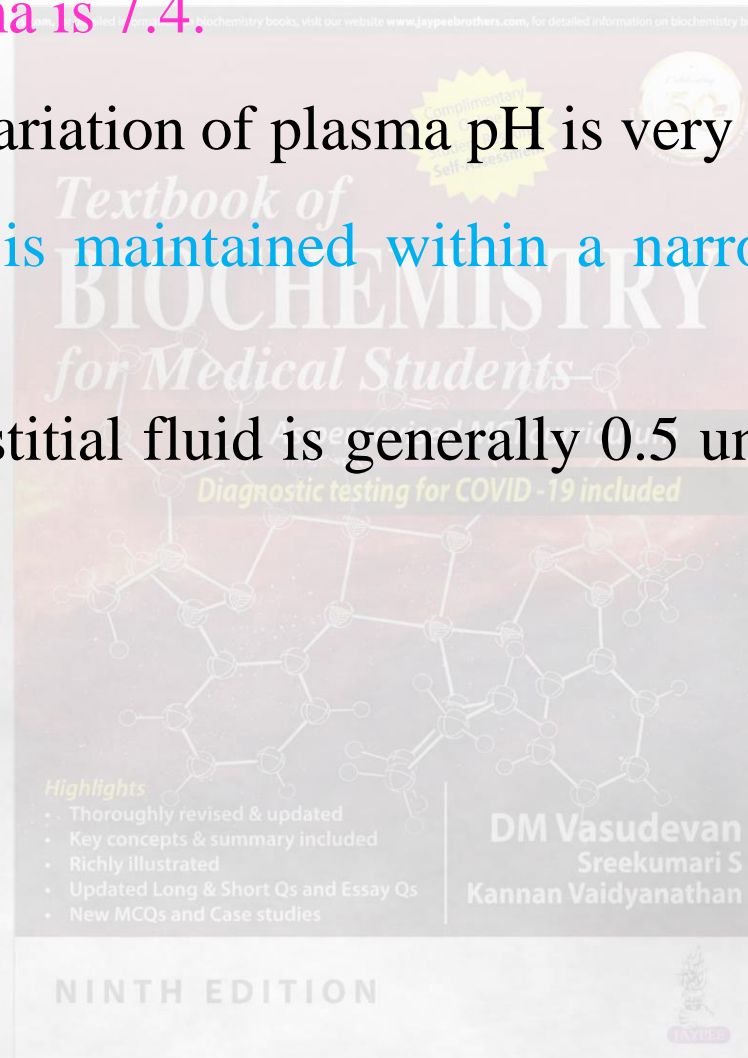


Normal pH of plasma is 7.4.

In normal life, the variation of plasma pH is very small.

The pH of plasma is maintained within a narrow range of 7.38 to 7.42.

The pH of the interstitial fluid is generally 0.5 units below that of the plasma.



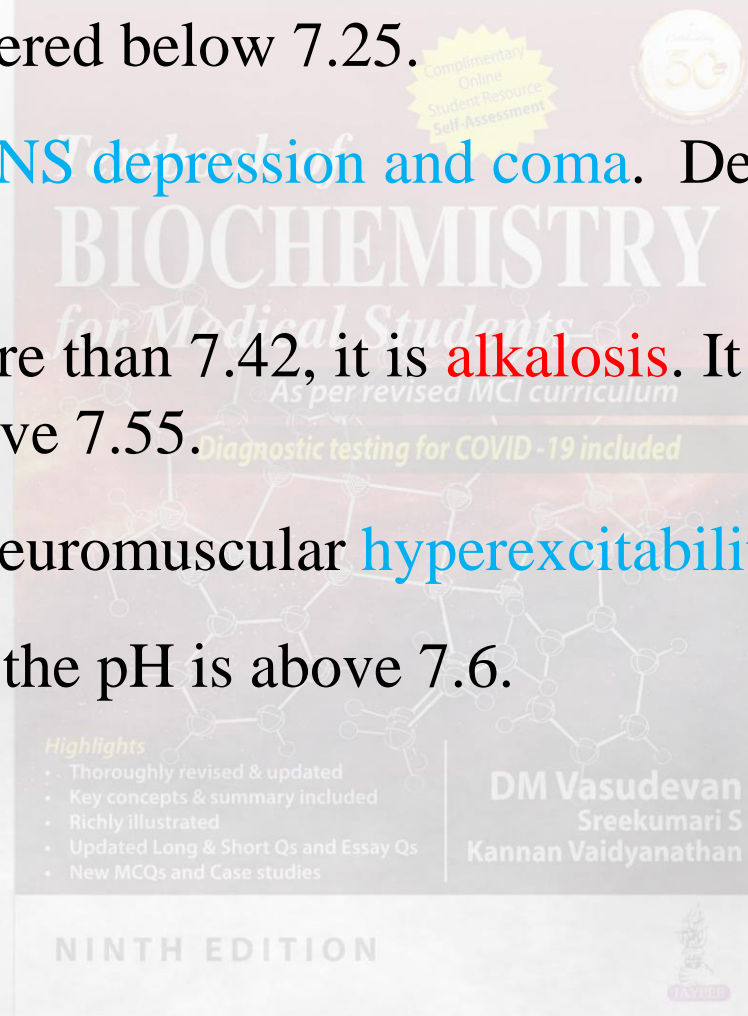
If the pH is below 7.38, it is called **acidosis**. Life is threatened when the pH is lowered below 7.25.

Acidosis leads to **CNS depression and coma**. Death occurs when pH is below 7.0.

When the pH is more than 7.42, it is **alkalosis**. It is very dangerous if pH is increased above 7.55.

Alkalosis induces neuromuscular **hyperexcitability** and tetany.

Death occurs when the pH is above 7.6.



Volatile and Fixed Acids

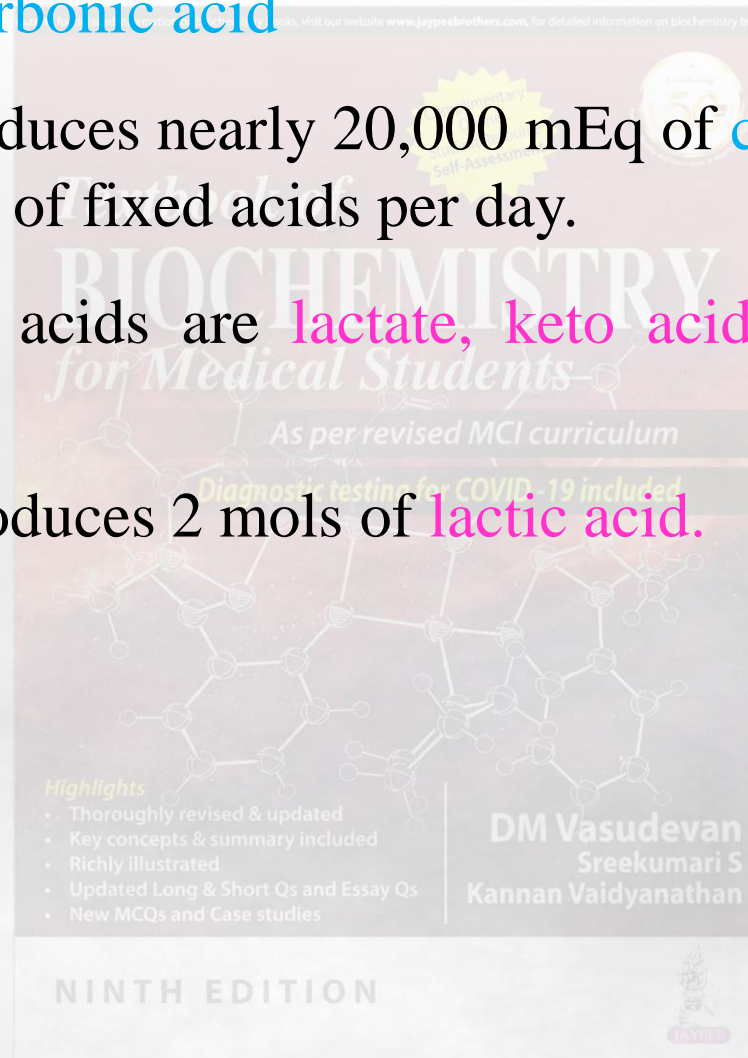


Volatile acids are **carbonic acid**

The metabolism produces nearly 20,000 mEq of **carbonic acid** and 60-80 mEq of fixed acids per day.

Nonvolatile (fixed) acids are **lactate, keto acids, sulfuric acid and phosphoric acid**.

1 mol of glucose produces 2 mols of **lactic acid**.



Volatile and Fixed Acids



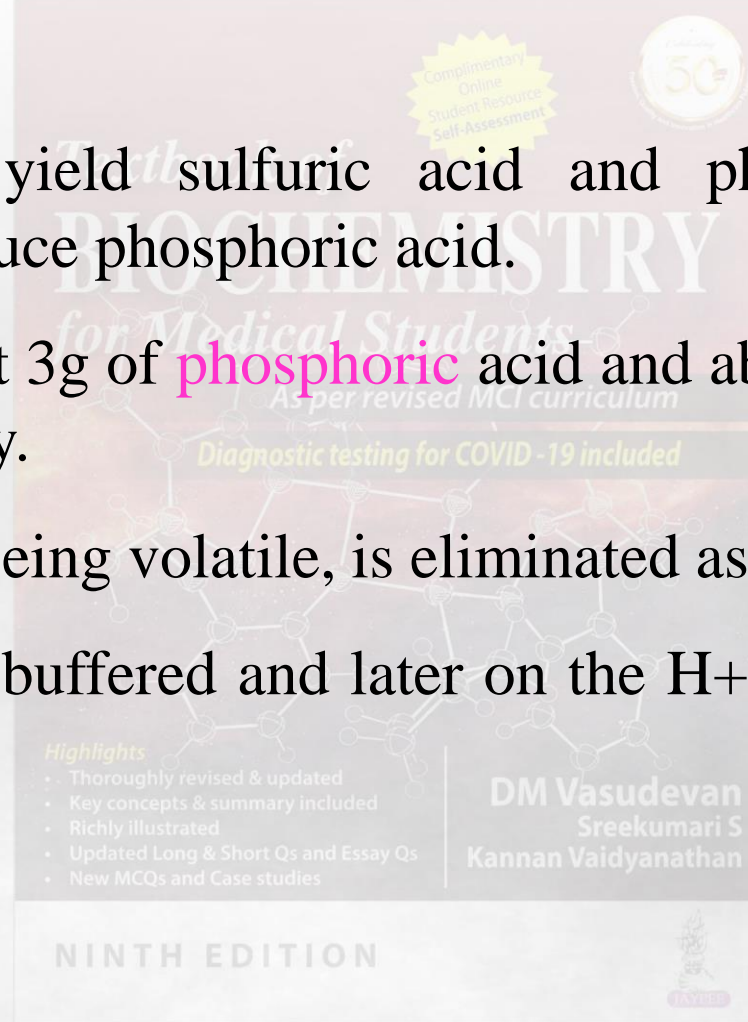
The dietary protein content decides the amount of sulfuric and phosphoric acids.

The sulfoproteins yield sulfuric acid and phosphoproteins and nucleoproteins produce phosphoric acid.

On an average about 3g of phosphoric acid and about 3g sulfuric acid are produced per day.

The carbonic acid, being volatile, is eliminated as CO₂ by the lungs.

The fixed acids are buffered and later on the H⁺ are excreted by the kidney.



Normal pH

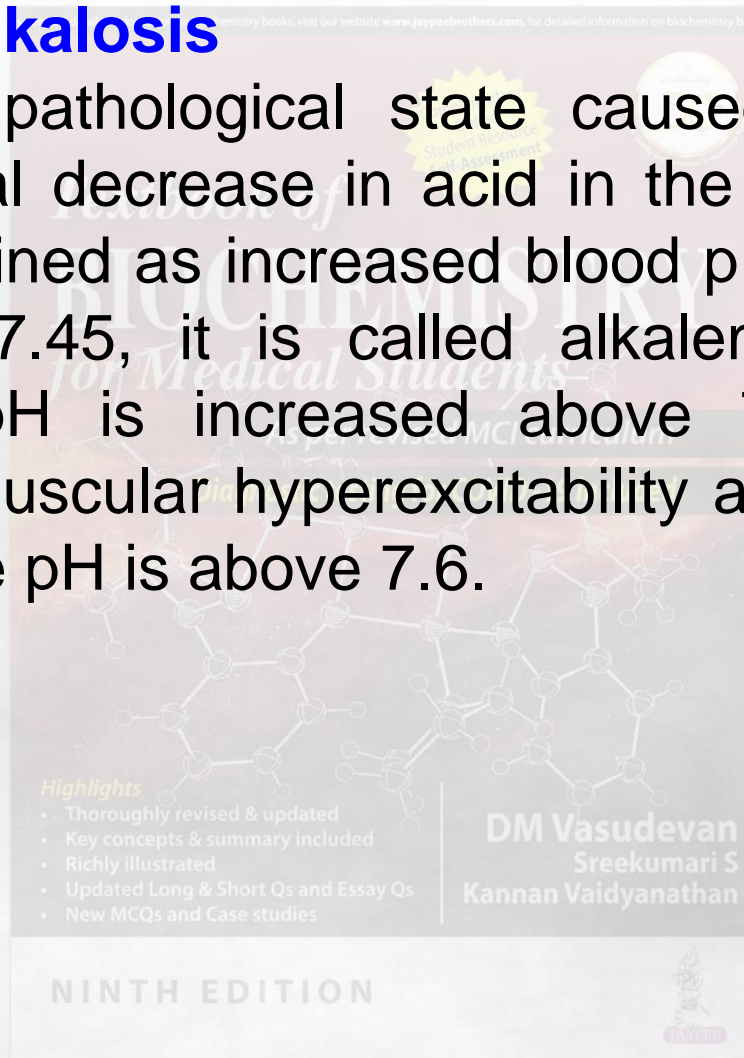
The **pH of plasma is 7.4**. The pH of plasma is maintained within a **narrow range of 7.38 to 7.42**; but for clinical purposes, the range used is 7.35 to 7.45. The pH of the interstitial fluid is generally 0.5 units below that of the plasma. The intracellular pH is lower than the extracellular pH.

Acidemia or Acidosis

Acidosis is a pathological state caused by a disease having abnormal increase in acid in the body. Whereas acidemia is defined as decreased blood pH. If the pH is below 7.35, it is called acidemia. Life is threatened when the pH is lowered below 7.25. Death occurs when pH is below 7.0.

Alkalemia or Alkalosis

Alkalosis is a pathological state caused by a disease having abnormal decrease in acid in the body. Whereas alkalemia is defined as increased blood pH. When the pH is more than 7.45, it is called alkalemia. It is very dangerous if pH is increased above 7.55. Alkalosis induces neuromuscular hyperexcitability and tetany. Death occurs when the pH is above 7.6.



Mechanisms of Regulation of pH



- First line of defense: Blood buffers
- Second line : Respiratory regulation
- Third line of defense: Renal regulation



1. Buffers of the Body Fluids



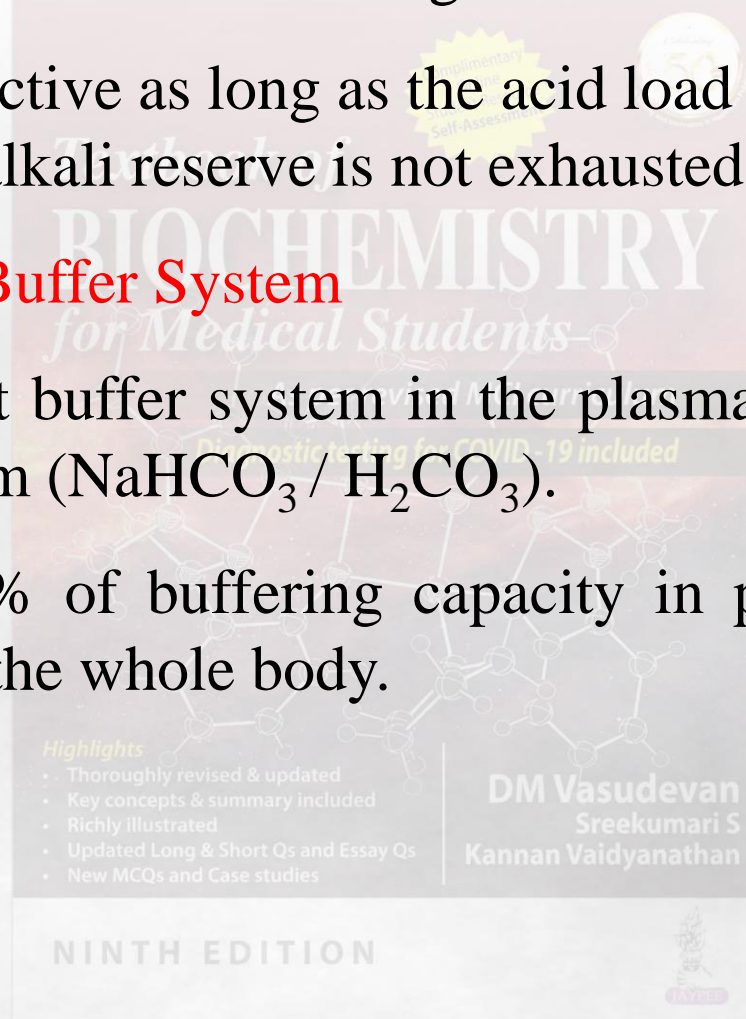
Buffers are the **first line of defense** against acid load.

The buffers are effective as long as the acid load is not excessive, and the alkali reserve is not exhausted.

1-A) Bicarbonate Buffer System

The most important buffer system in the plasma is the bicarbonate-carbonic acid system ($\text{NaHCO}_3 / \text{H}_2\text{CO}_3$).

It accounts for 65% of buffering capacity in plasma and 40% of buffering action in the whole body.

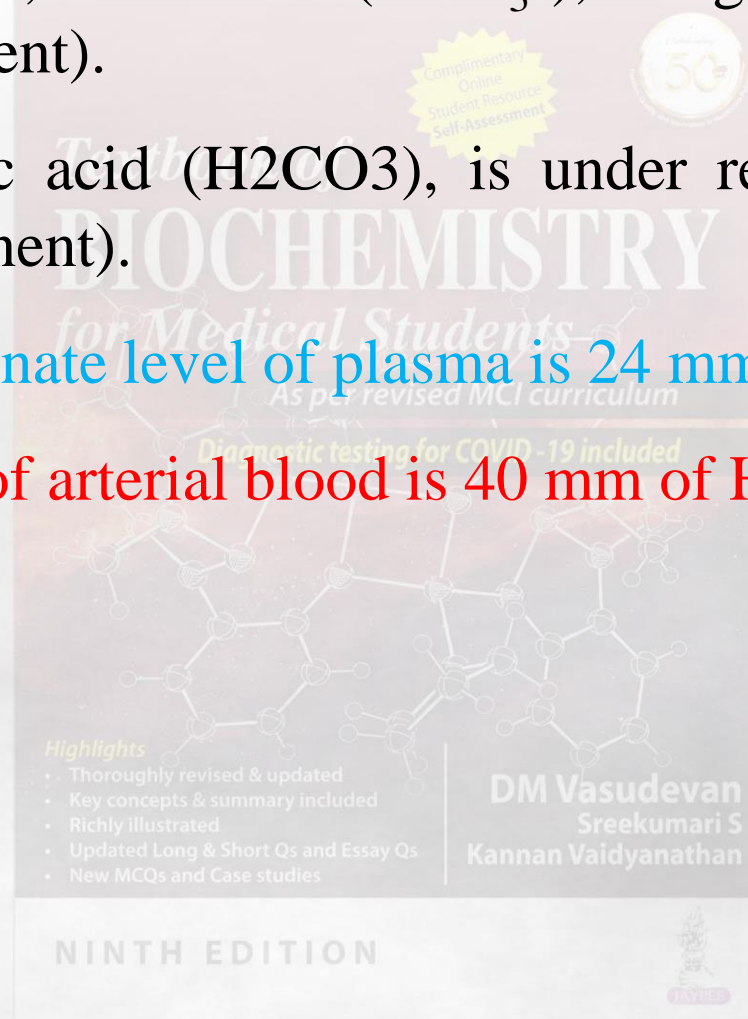


The base constituent, bicarbonate (HCO_3^-), is regulated by the kidney (metabolic component).

Acid part, carbonic acid (H_2CO_3), is under respiratory regulation (respiratory component).

The normal bicarbonate level of plasma is 24 mmol/liter.

The normal pCO_2 of arterial blood is 40 mm of Hg.



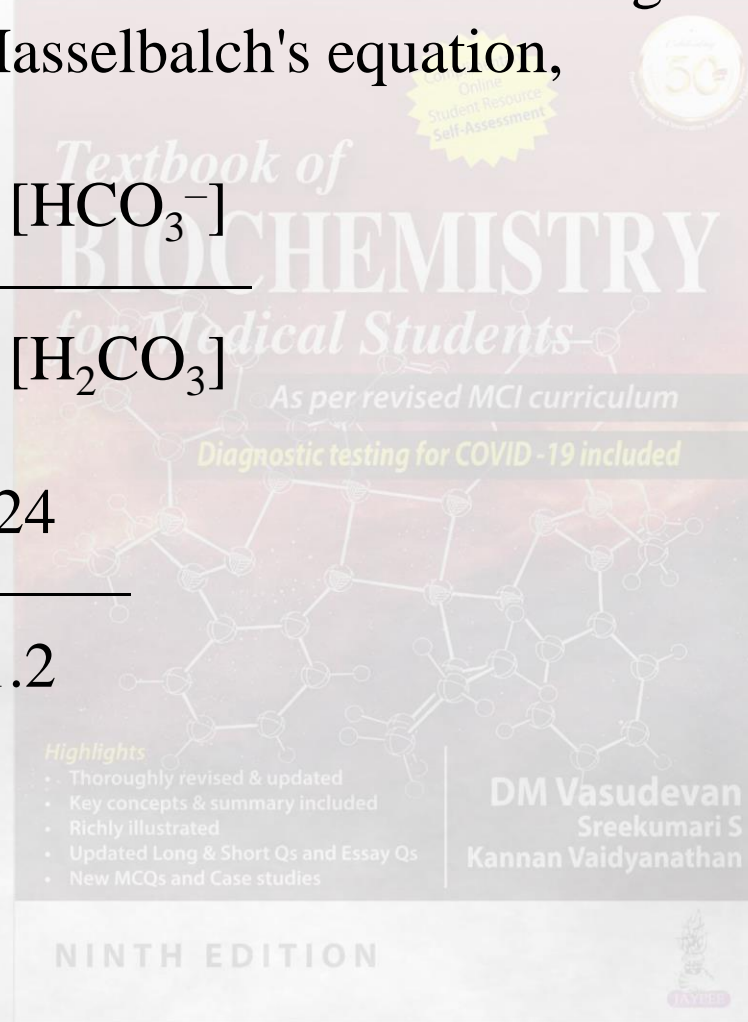
The pKa for carbonic acid is 6.1. Substituting these values in the Henderson–Hasselbalch's equation,

$$\text{pH} = \text{pKa} + \log \frac{[\text{HCO}_3^-]}{[\text{H}_2\text{CO}_3]}$$

$$7.4 = 6.1 + \log \frac{24}{1.2}$$

$$= 6.1 + \log 20$$

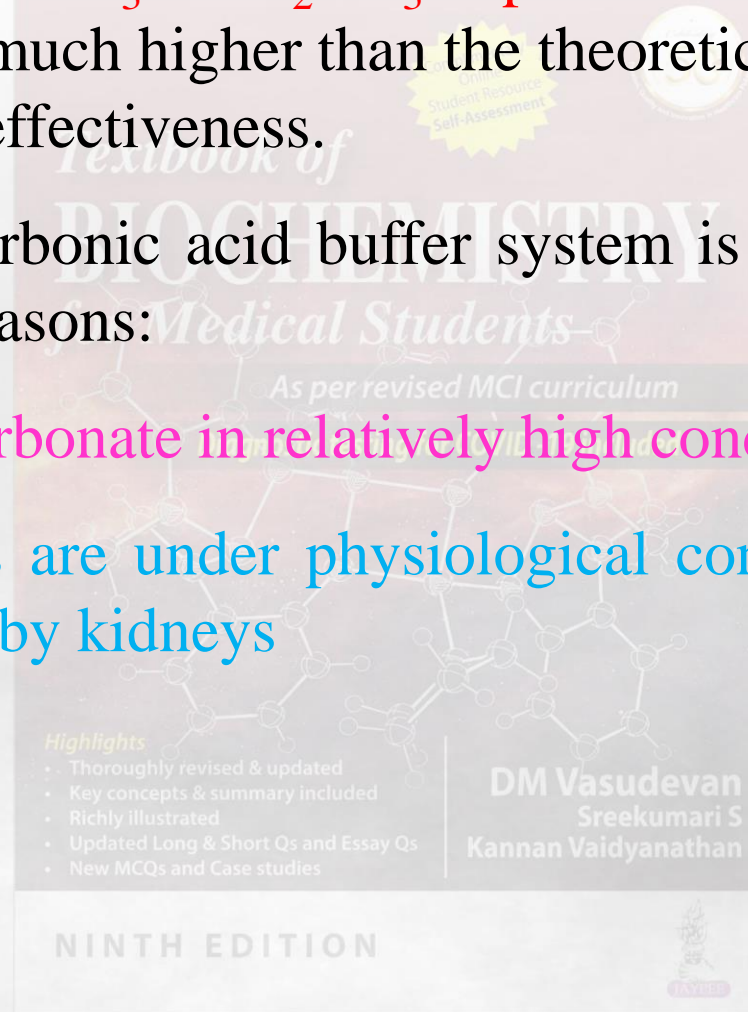
$$= 6.1 + 1.3$$



Hence, the ratio of HCO_3^- to H_2CO_3 at pH 7.4 is 20 under normal conditions. This is much higher than the theoretical value of 1 which ensures maximum effectiveness.

The bicarbonate carbonic acid buffer system is the most important for the following reasons:

- Presence of bicarbonate in relatively high concentrations.
- The components are under physiological control, CO_2 by lungs and bicarbonate by kidneys



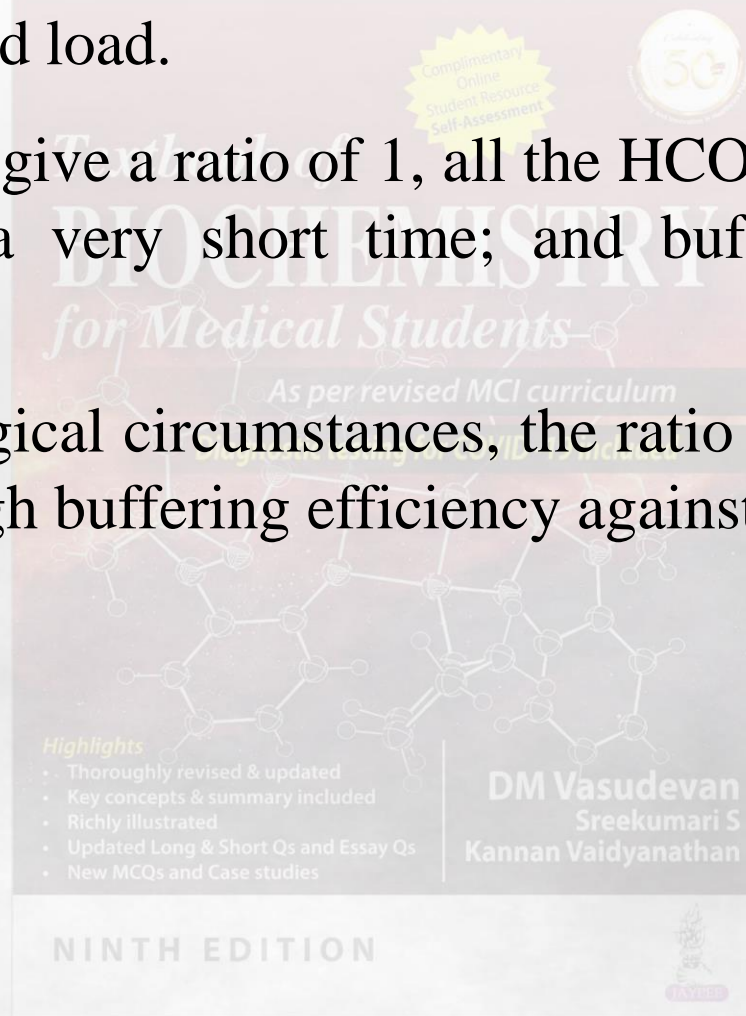
Alkali Reserve



Bicarbonate represents the alkali reserve. It has to be sufficiently high to meet the acid load.

If it was too low to give a ratio of 1, all the HCO_3^- would have been exhausted within a very short time; and buffering will not be effective.

So, under physiological circumstances, the ratio of 20 (a high alkali reserve) ensures high buffering efficiency against acids.



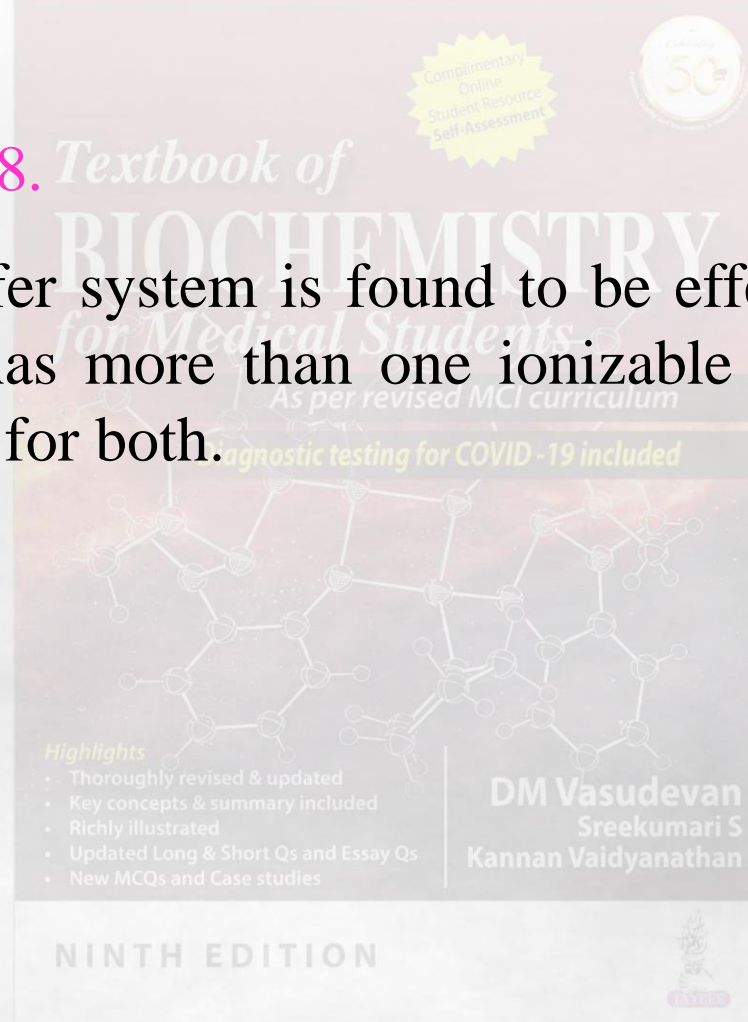
1-B) Phosphate Buffer System

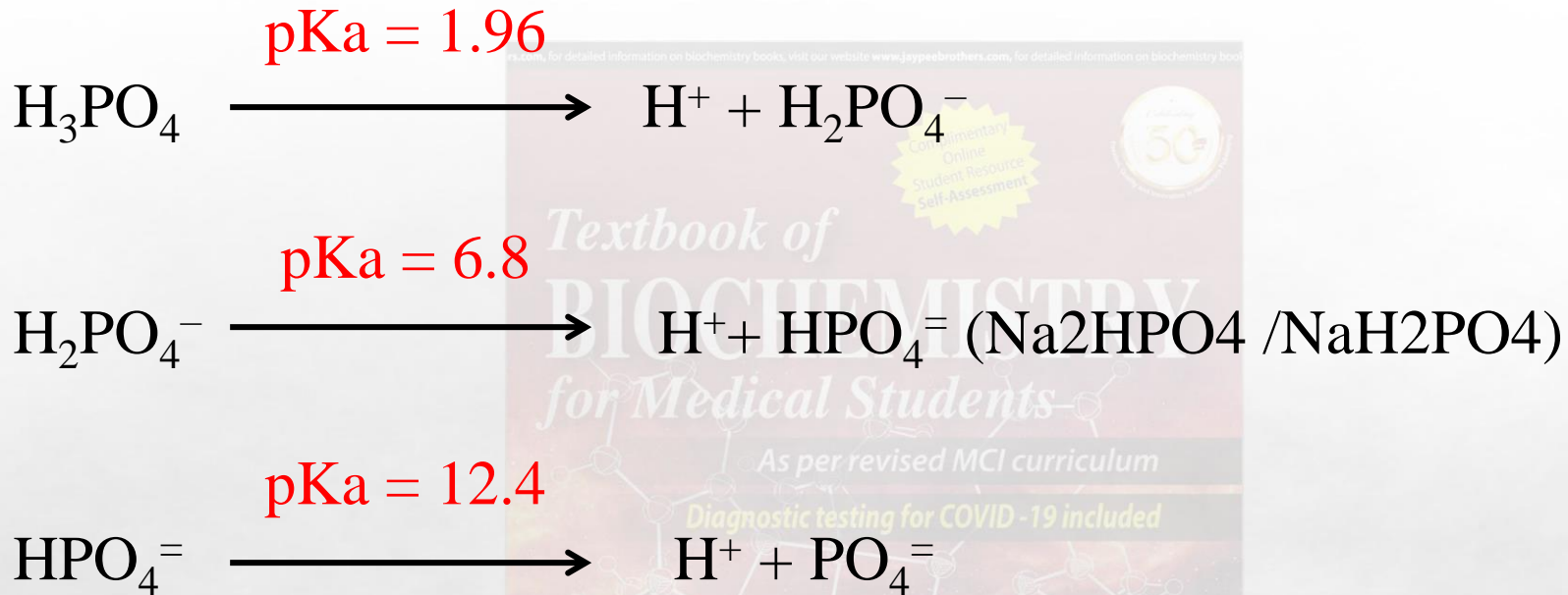


It is mainly an **intracellular** buffer. Its concentration in plasma is very low.

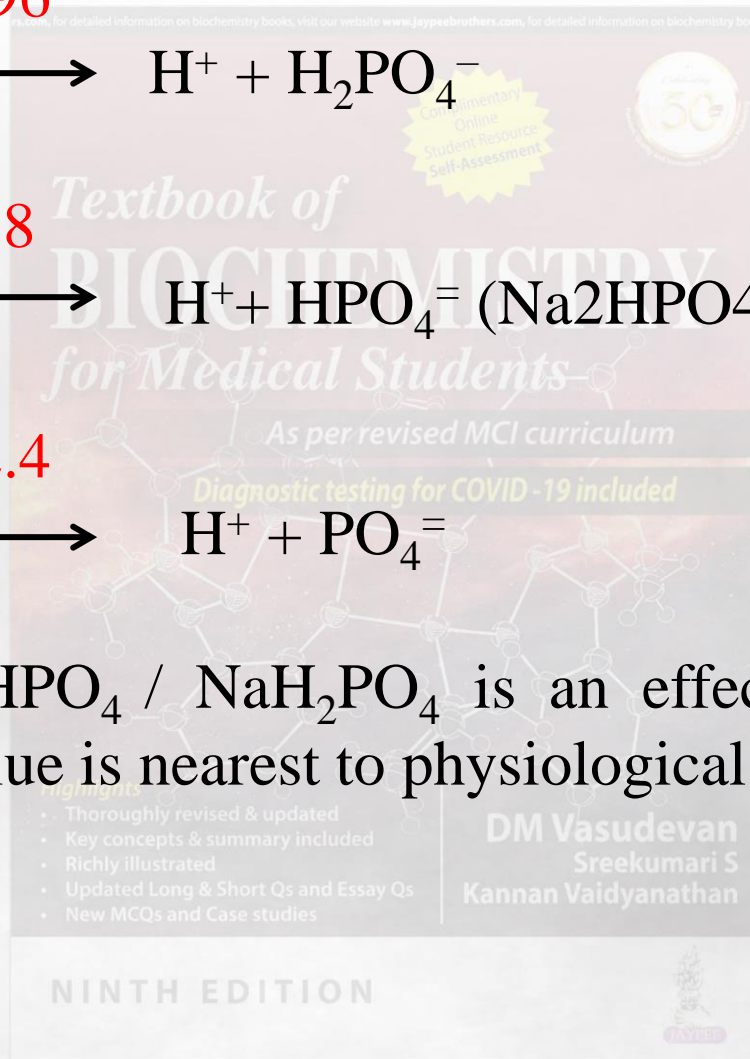
The pKa value is 6.8.

The phosphate buffer system is found to be effective at a wide pH range, because it has more than one ionizable group and the pKa values are different for both.





In the body, $\text{Na}_2\text{HPO}_4 / \text{NaH}_2\text{PO}_4$ is an effective buffer system, because its pKa value is nearest to physiological pH.



1-C) Protein Buffer System



Buffering capacity of protein depends on the pKa value of ionizable side chains.

The most effective group is **histidine imidazole group** with a pKa value of 6.1.

Hemoglobin buffer



Buffer Systems of the Body



Extracellular fluid	Intracellular fluid	Erythrocyte fluid
NaHCO_3 H_2CO_3 (bicarbonate)	K_2HPO_4 KH_2PO_4 (phosphate)	$\text{K}+\text{Hb}$ $\text{H}+\text{Hb}$ (hemoglobin)
Na_2HPO_4 NaH_2PO_4 (phosphate)	$\text{K}+\text{Protein}$ $\text{H}^+\text{Protein}$ (protein buffer)	K_2HPO_4 KH_2PO_4 (phosphate)
$\text{Na} + \text{Albumin}$ $\text{H}^+\text{Albumin}$	KHCO_3 H_2CO_3	KHCO_3 H_2CO_3

- Richly illustrated
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- New MCQs and Case studies

Sreekumari S
Kannan Vaidyanathan

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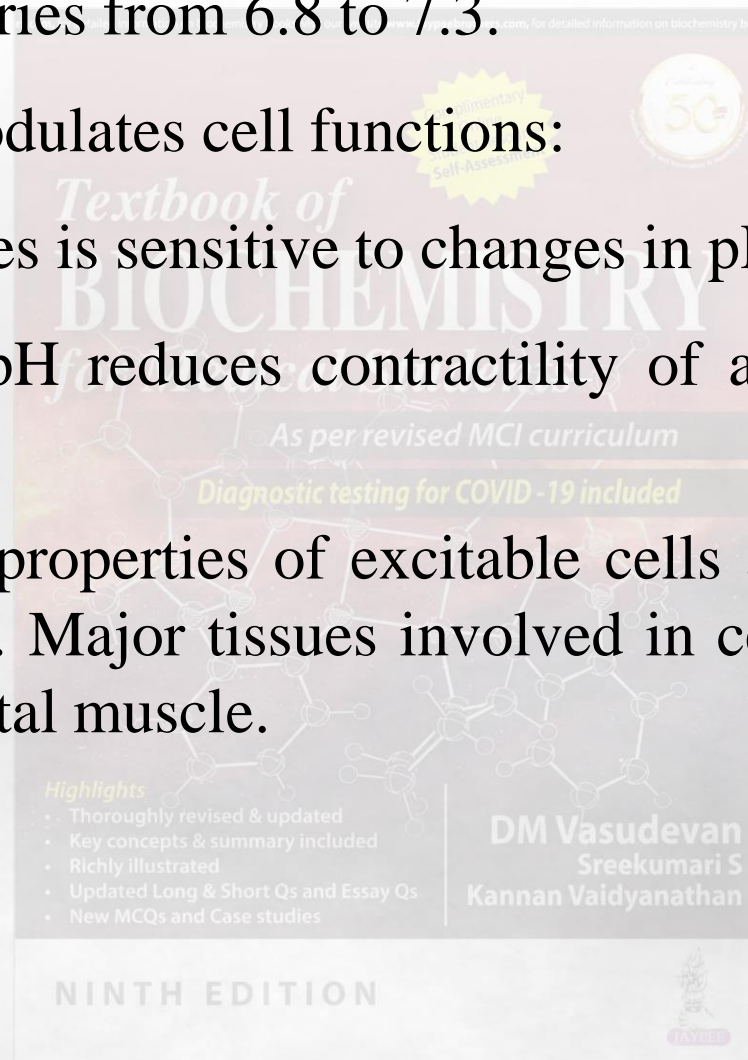
Cellular Buffers



Cytoplasmic pH varies from 6.8 to 7.3.

Intracellular pH modulates cell functions:

1. Several enzymes is sensitive to changes in pH.
2. Reduction in pH reduces contractility of actin and myosin in muscles.
3. The electrical properties of excitable cells are also affected by changes in pH. Major tissues involved in cellular buffering are bone and skeletal muscle.

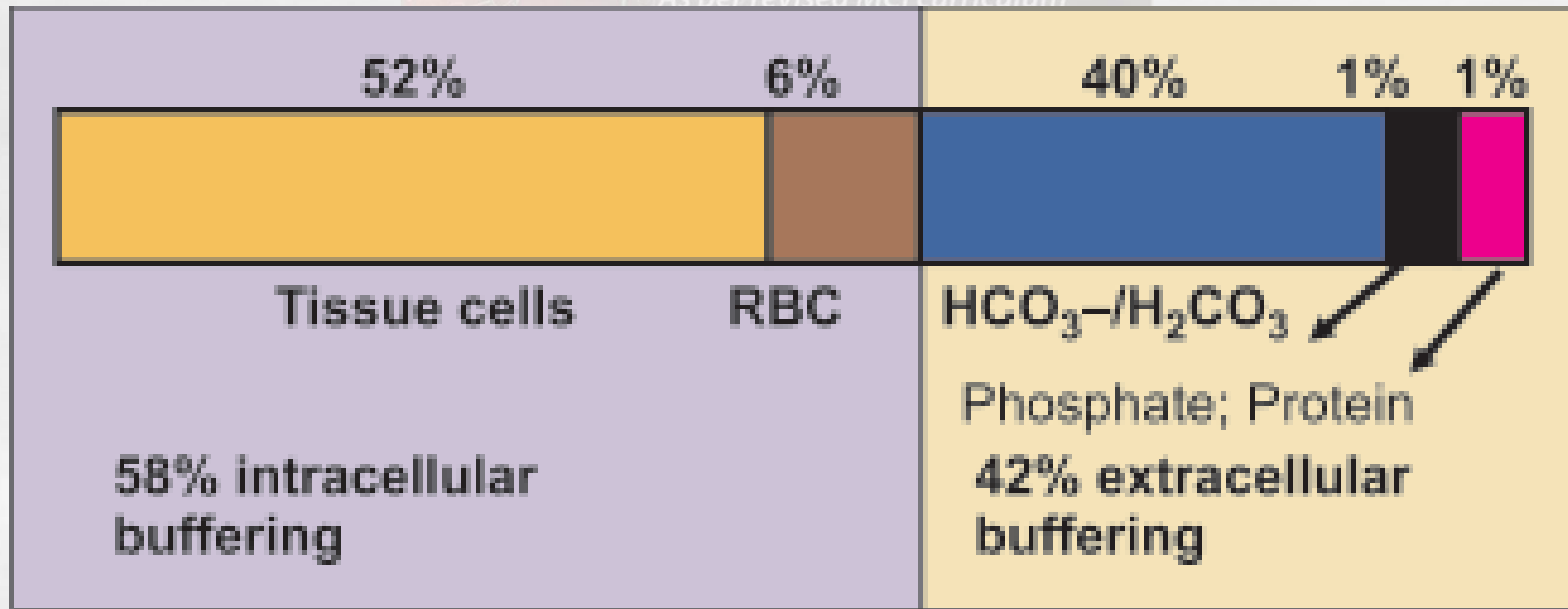


Relative Capacity of Buffer Systems



In the body, 52% buffer activity is in tissue cells and 6% in RBCs; 43% is by extracellular buffers.

In plasma and extracellular space, about 40% buffering action is by bicarbonate system; 1% by proteins and 1% by phosphate buffer system.



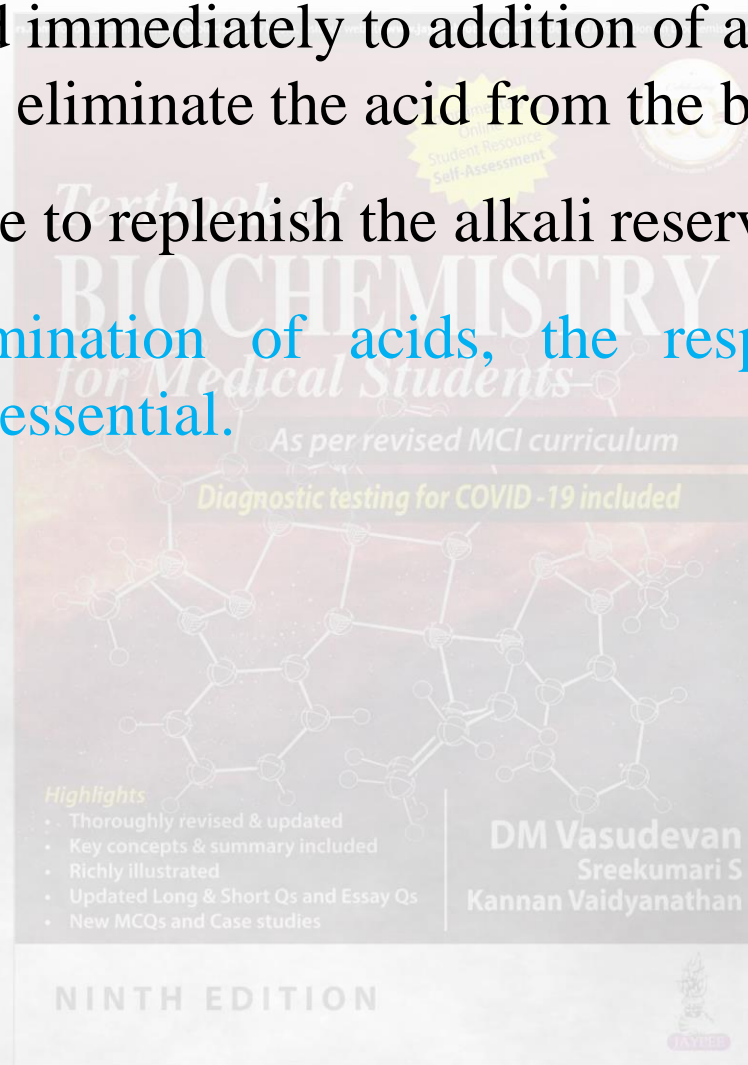
Buffers Act Quickly, But Not Permanently



Buffers can respond immediately to addition of acid or base, but they do not serve to eliminate the acid from the body.

They are also unable to replenish the alkali reserve of the body.

For the final elimination of acids, the respiratory and renal regulations are very essential.



2. Respiratory Regulation of pH

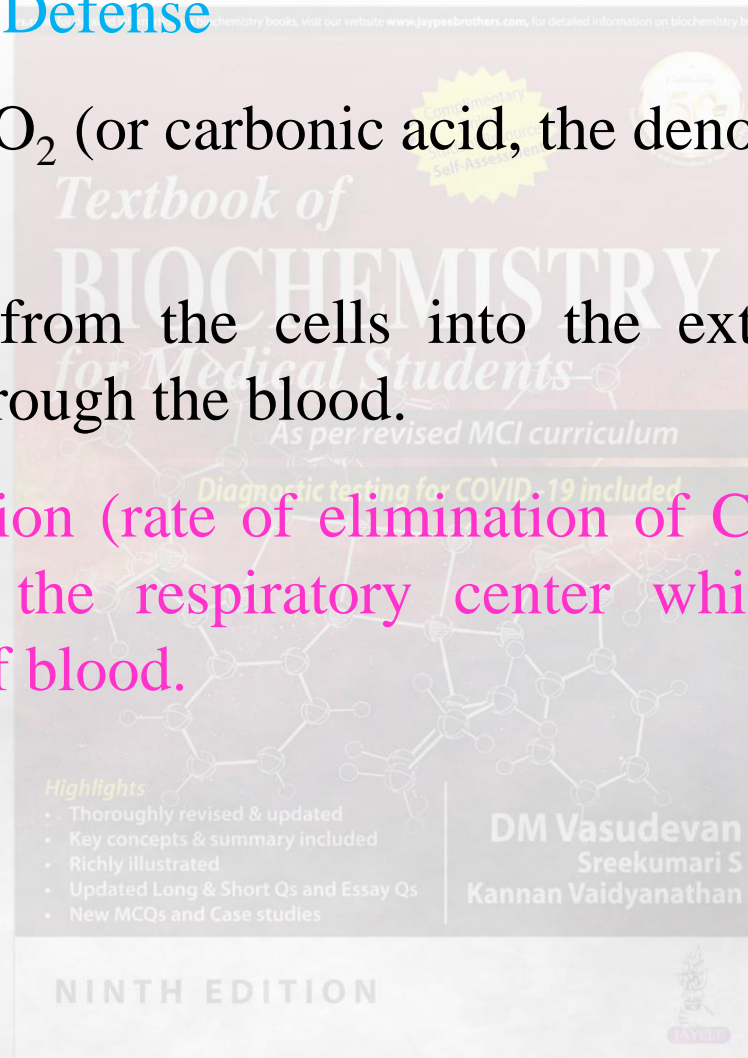


The Second Line of Defense

By changing the $p\text{CO}_2$ (or carbonic acid, the denominator in the equation).

The CO_2 diffuses from the cells into the extracellular fluid and reaches the lungs through the blood.

The rate of respiration (rate of elimination of CO_2) is controlled by chemoreceptors in the respiratory center which are sensitive to changes in the pH of blood.



2. Respiratory Regulation of pH

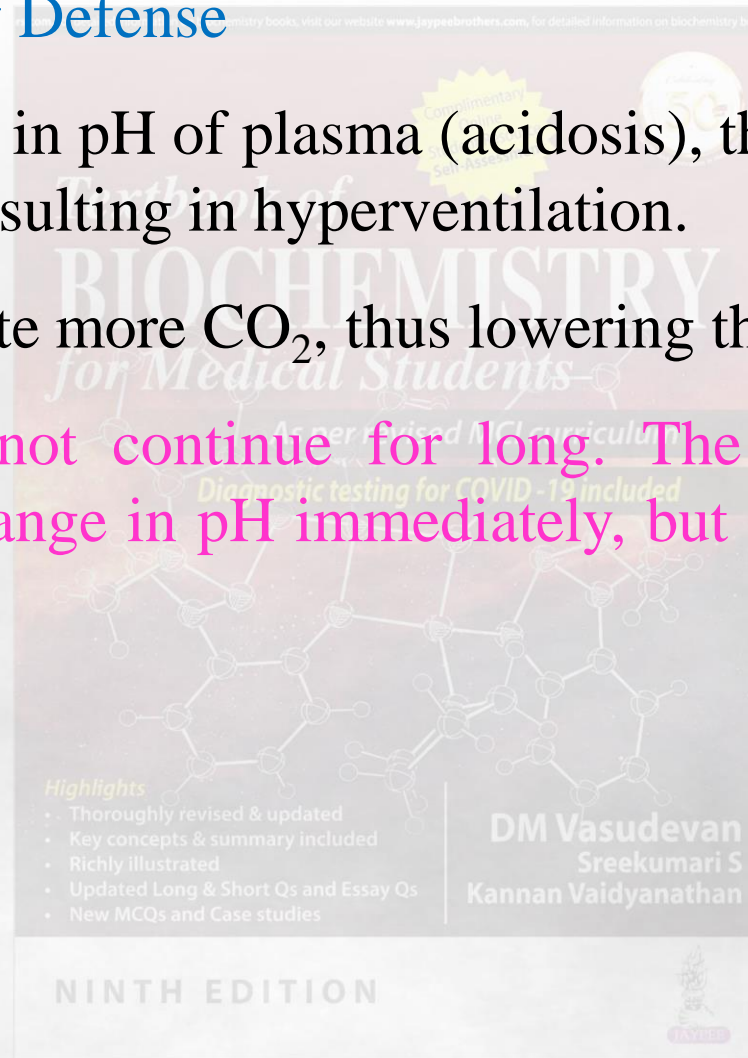


The Second Line of Defense

When there is a fall in pH of plasma (acidosis), the respiratory rate is stimulated resulting in hyperventilation.

This would eliminate more CO_2 , thus lowering the H_2CO_3 level.

However, this cannot continue for long. The respiratory system responds to any change in pH immediately, but it cannot proceed to completion.

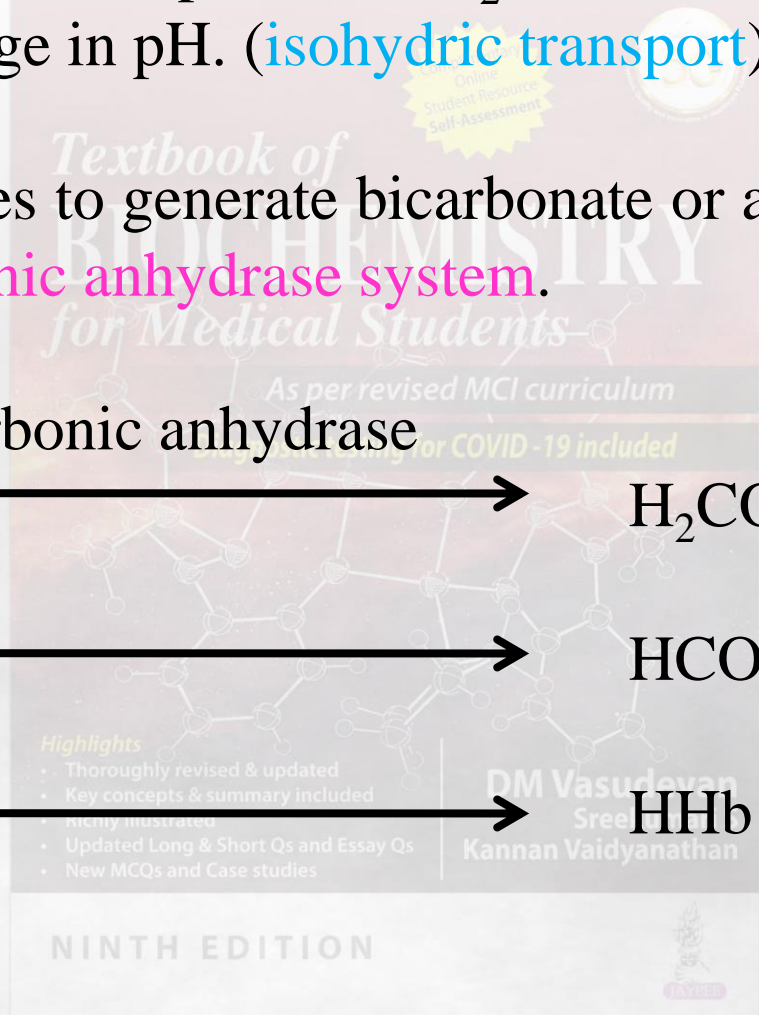
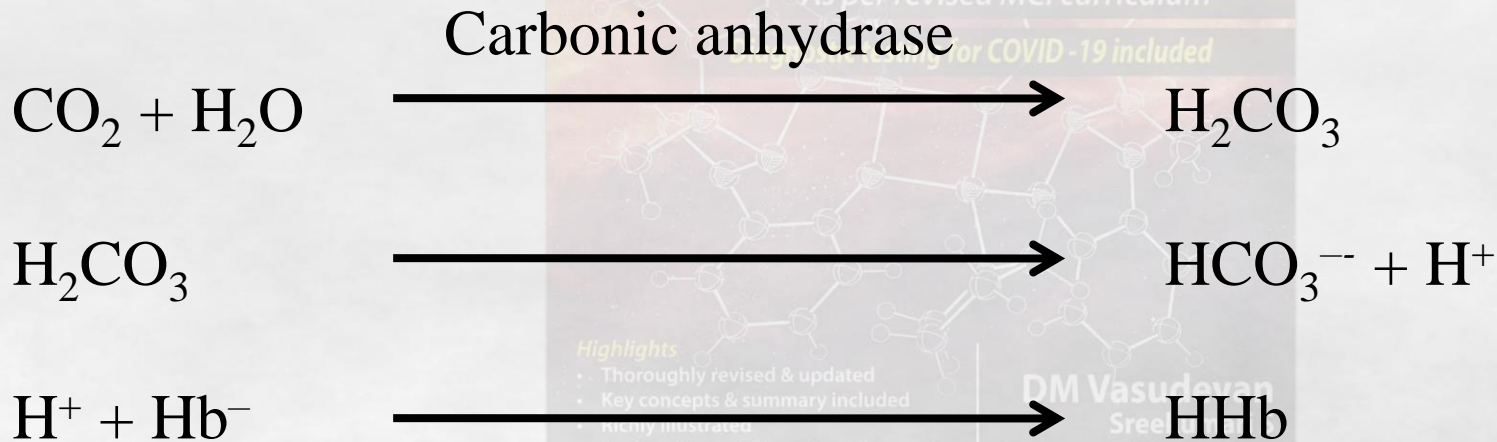


Action of Hemoglobin



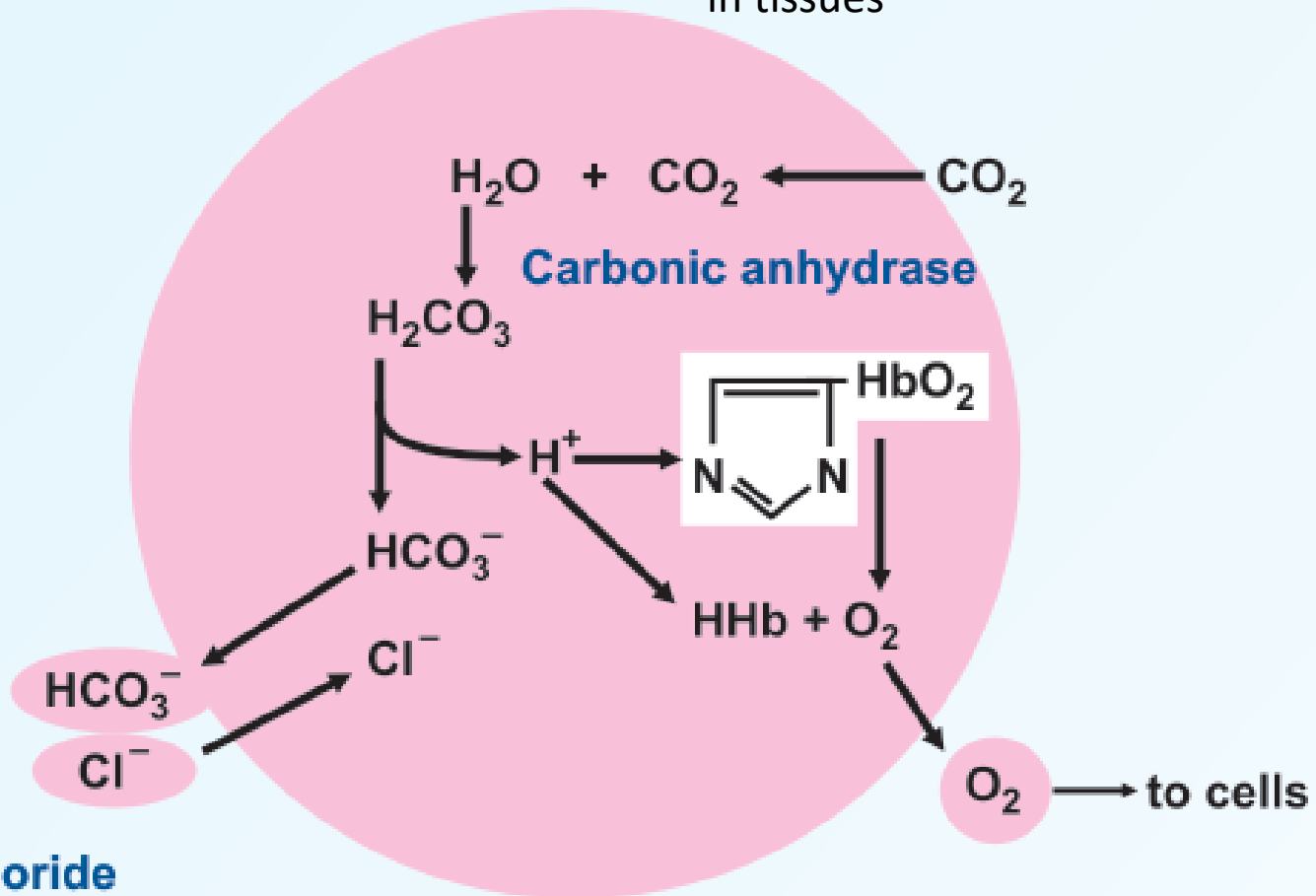
Hemoglobin serves to transport the CO_2 formed in the tissues, with minimum change in pH. (**isohydric transport**).

Side by side, it serves to generate bicarbonate or alkali reserve by the activity of the **carbonic anhydrase system**.



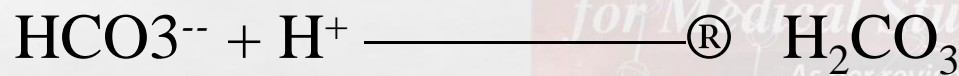
Erythrocyte in tissue capillary

Chloride shift; reactions in tissues



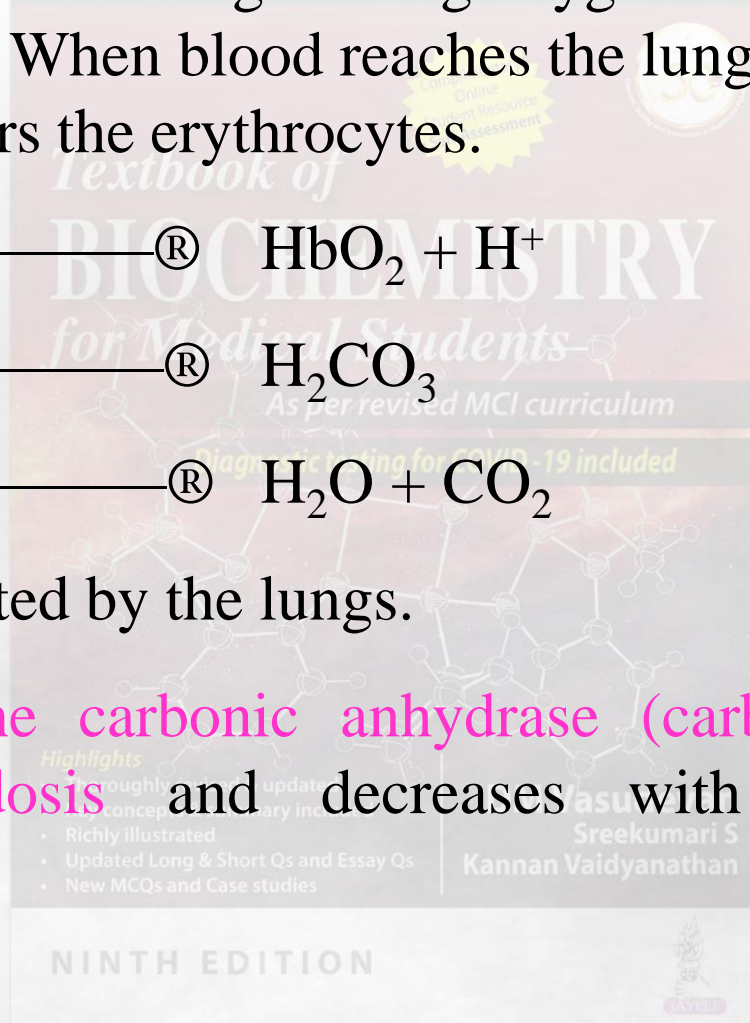
Chloride enters into RBC

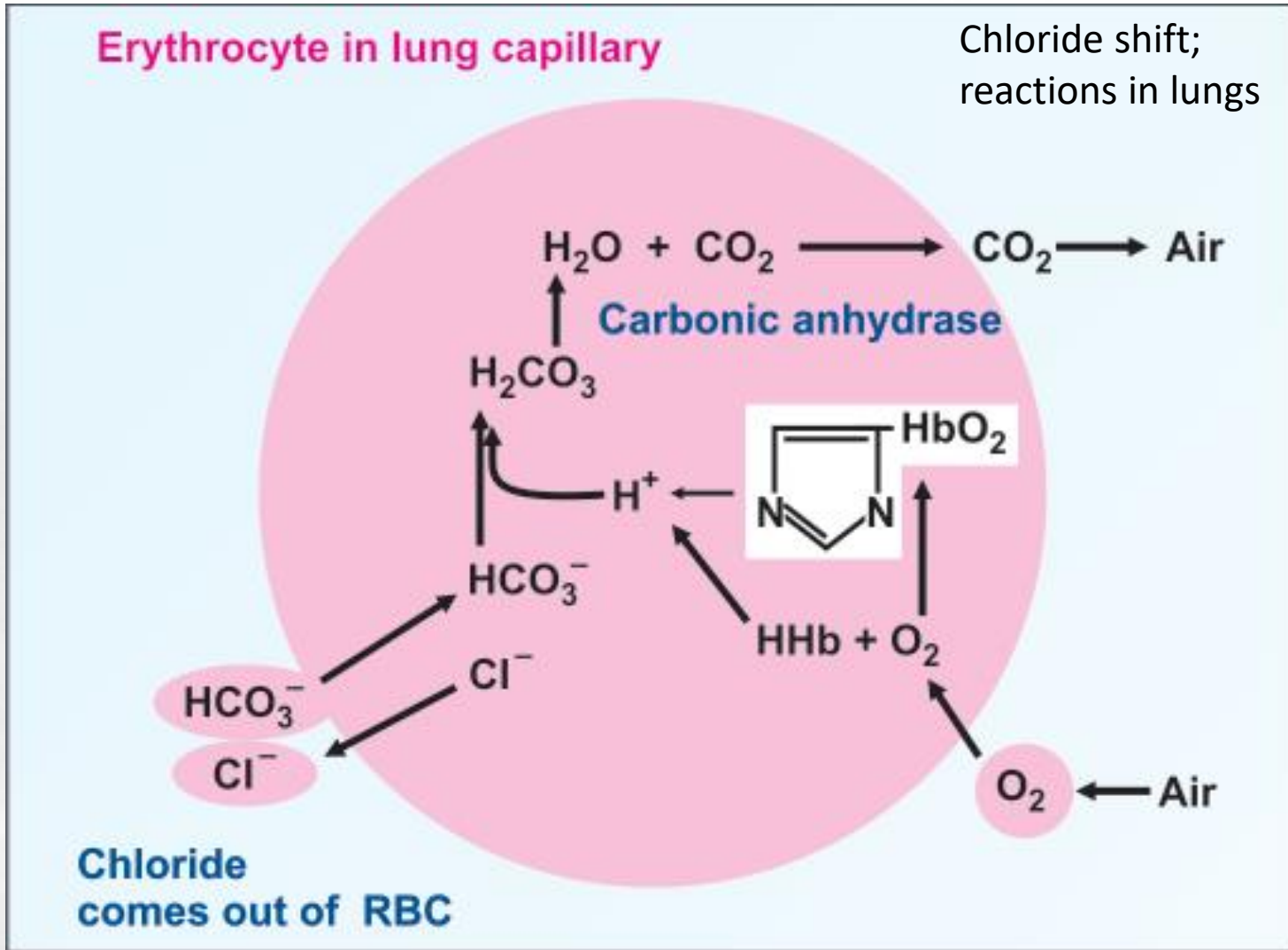
The reverse occurs in the lungs during oxygenation and elimination of CO₂. When blood reaches the lungs, the Bicarbonate re-enters the erythrocytes.



CO₂ is thus eliminated by the lungs.

The activity of the carbonic anhydrase (carbonate dehydratase) increases in acidosis and decreases with decrease in H⁺ concentration.





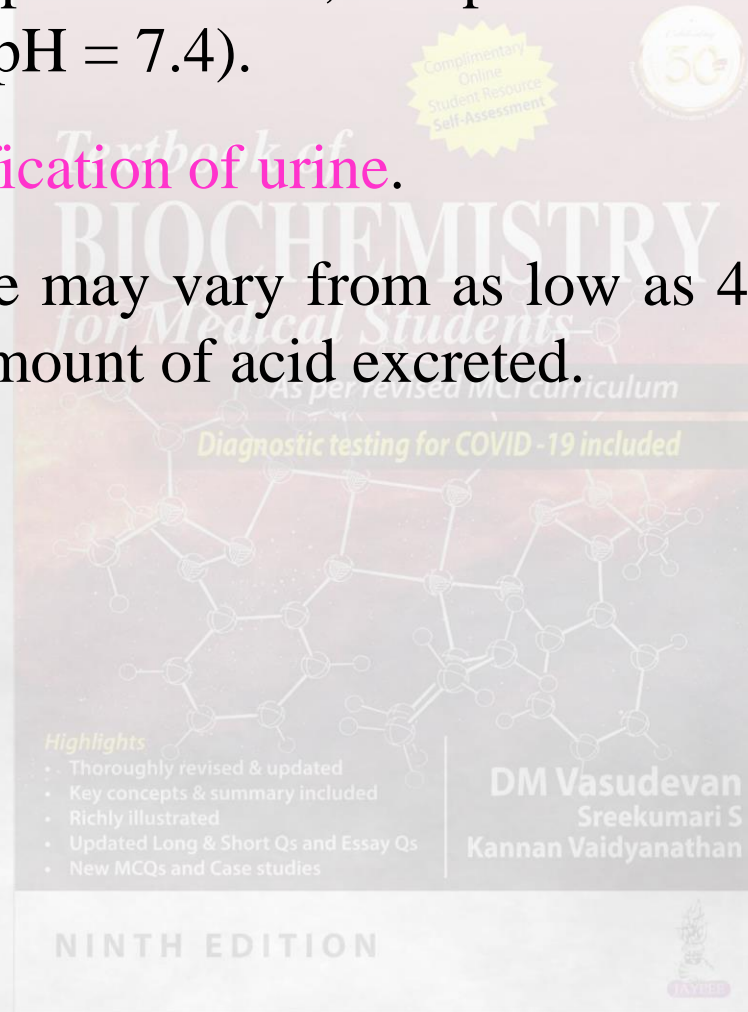
3. Renal Regulation of pH



Normal urine has a pH around 6; this pH is lower than that of extracellular fluid (pH = 7.4).

This is called **acidification of urine**.

The pH of the urine may vary from as low as 4.5 to as high as 9.8, depending on the amount of acid excreted.



Major Renal Mechanisms:

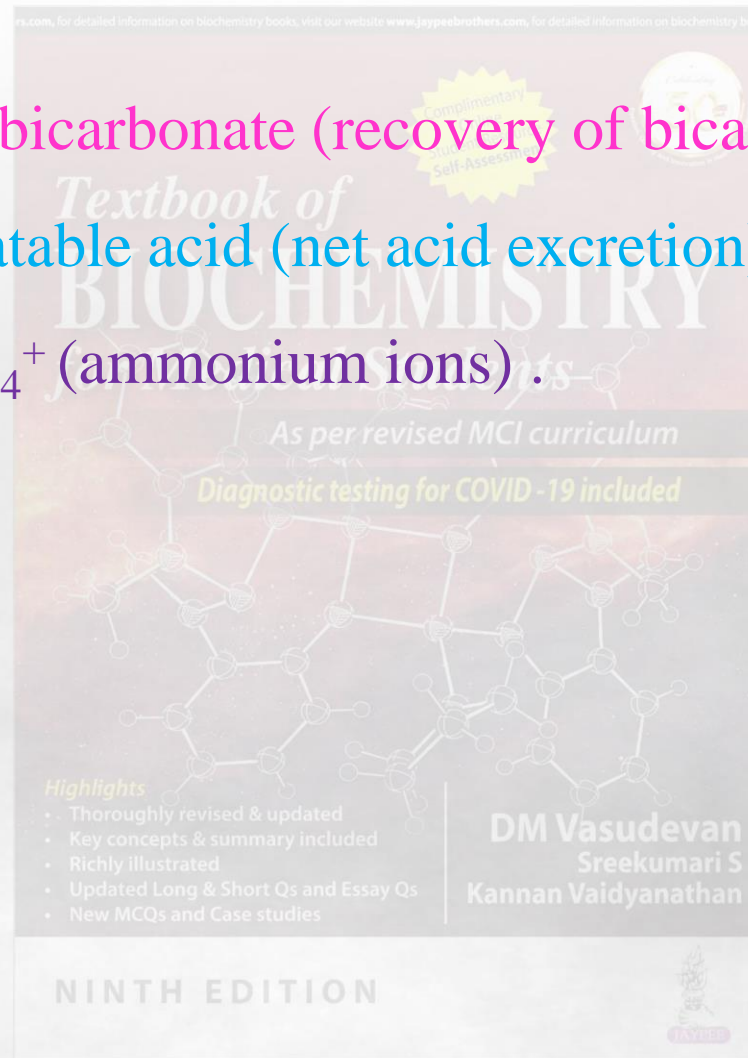


A. Excretion of H^+

B. Reabsorption of bicarbonate (recovery of bicarbonate)

C. Excretion of titratable acid (net acid excretion)

D. Excretion of NH_4^+ (ammonium ions)



3.A. Excretion of H⁺; Generation of Bicarb



Occurs in proximal convoluted tubules.

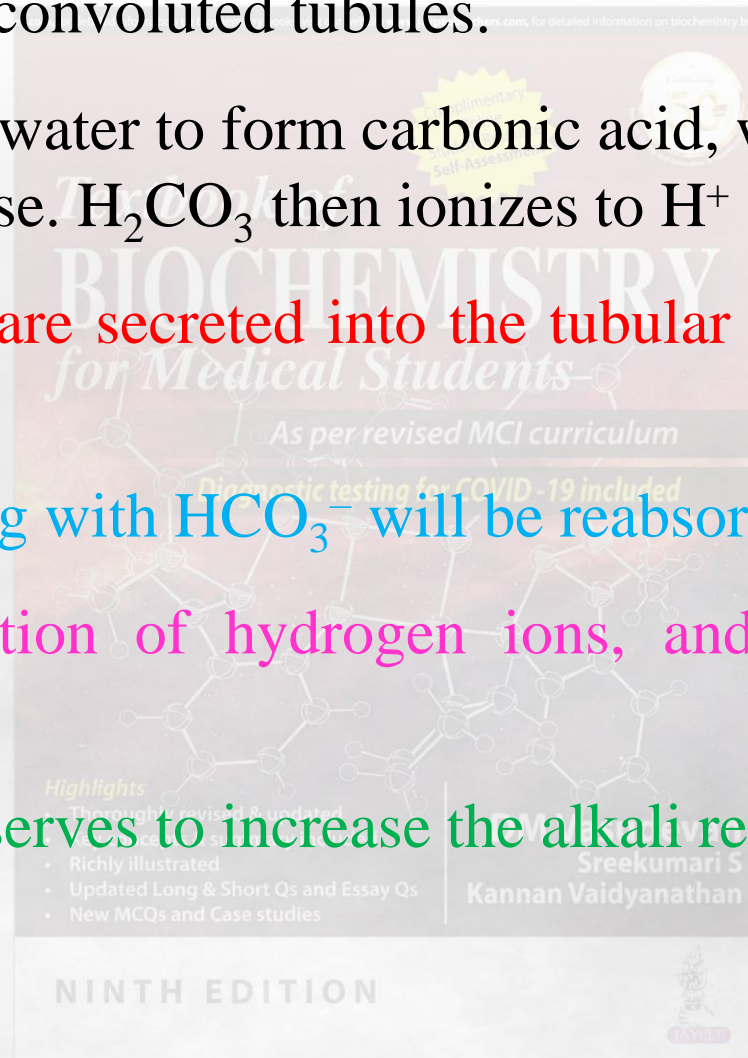
CO₂ combines with water to form carbonic acid, with the help of carbonic anhydrase. H₂CO₃ then ionizes to H⁺ and bicarbonate.

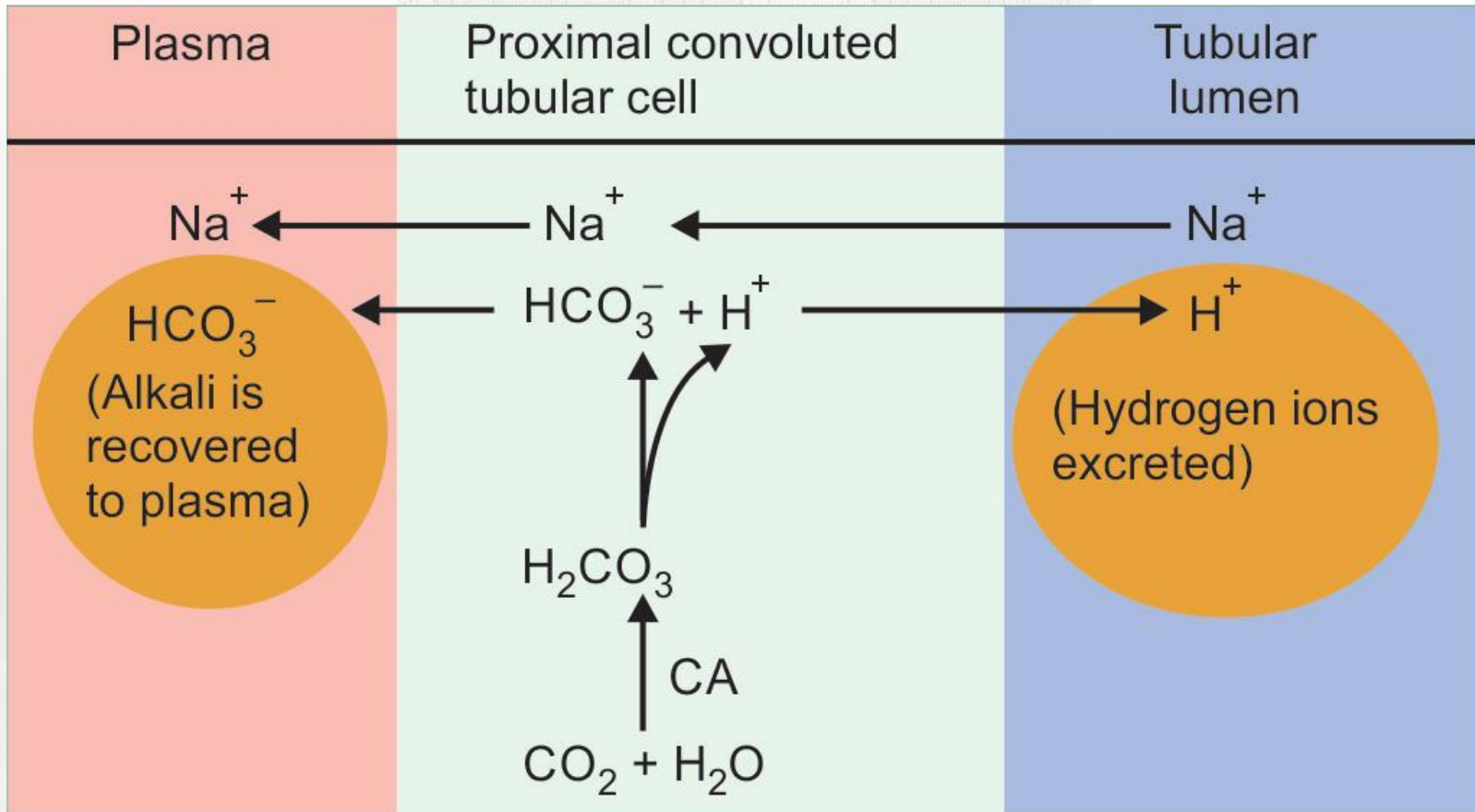
The hydrogen ions are secreted into the tubular lumen; in exchange for Na⁺ reabsorbed.

These Na⁺ ions along with HCO₃⁻ will be reabsorbed into the blood.

There is net excretion of hydrogen ions, and net generation of bicarbonate.

So this mechanism serves to increase the alkali reserve.





Excretion of Hydrogen Ions in the Proximal Tubules

3.B. Reabsorption of Bicarbonate

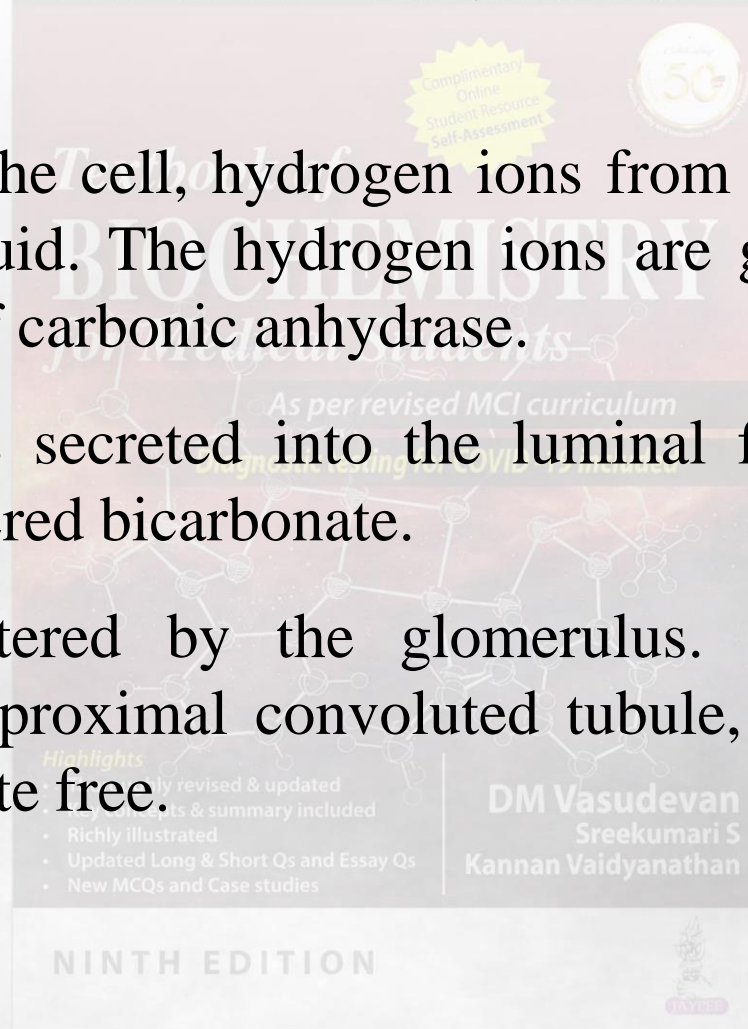


This is mainly a mechanism to conserve base. There is no net excretion of H^+

When Na^+ enters the cell, hydrogen ions from the cell are secreted into the luminal fluid. The hydrogen ions are generated within the cell by the action of carbonic anhydrase.

The hydrogen ions secreted into the luminal fluid is required for reabsorption of filtered bicarbonate.

Bicarbonate is filtered by the glomerulus. This is completely reabsorbed by the proximal convoluted tubule, so that the urine is normally bicarbonate free.

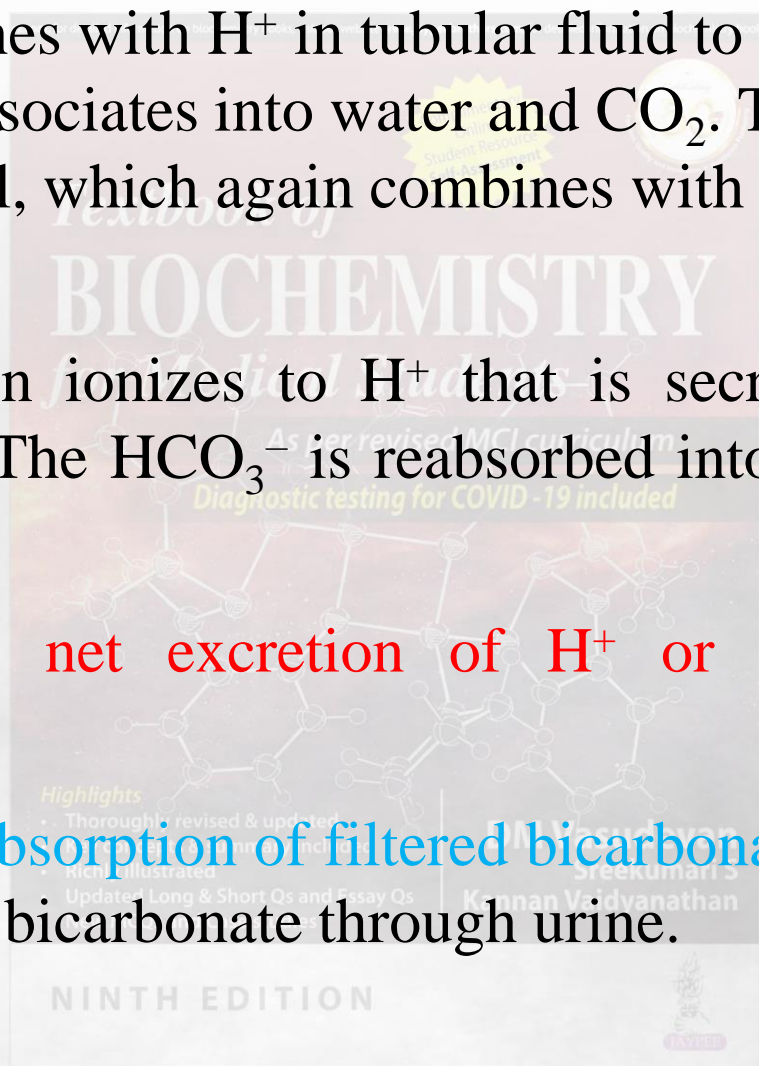


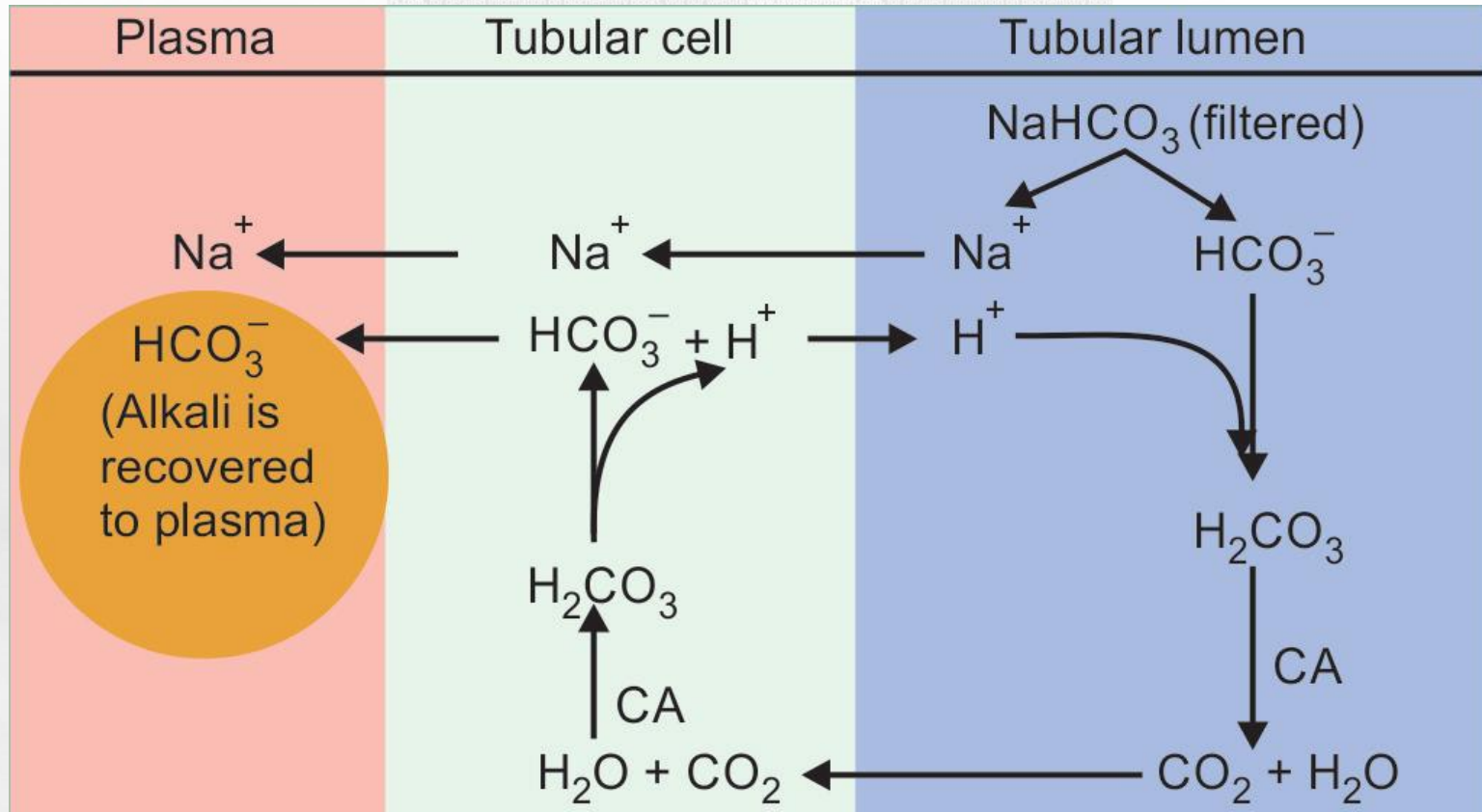
Bicarbonate combines with H^+ in tubular fluid to form carbonic acid. It dissociates into water and CO_2 . The CO_2 diffuses into the cell, which again combines with water to form carbonic acid.

In the cell, it again ionizes to H^+ that is secreted into lumen in exchange for Na^+ . The HCO_3^- is reabsorbed into plasma along with Na^+ .

Here, there is no net excretion of H^+ or generation of new bicarbonate.

Net effect is the reabsorption of filtered bicarbonate. This mechanism prevents the loss of bicarbonate through urine.





Reabsorption of Bicarbonate from the Tubular Fluid

NINTH EDITION

3.C. Excretion of H⁺ as Titratable Acid



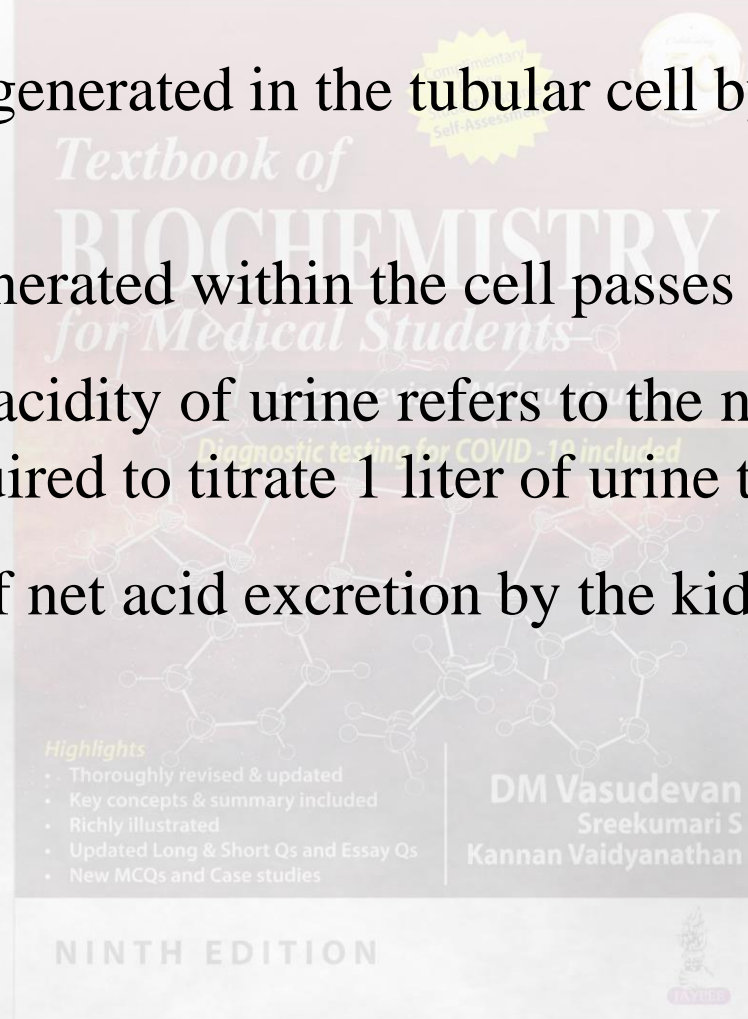
In the distal convoluted tubules net acid excretion occurs.

Hydrogen ions are generated in the tubular cell by carbonic anhydrase.

The bicarbonate generated within the cell passes into plasma.

The term titratable acidity of urine refers to the number of milliliters of N/10 NaOH required to titrate 1 liter of urine to pH 7.4.

This is a measure of net acid excretion by the kidney.



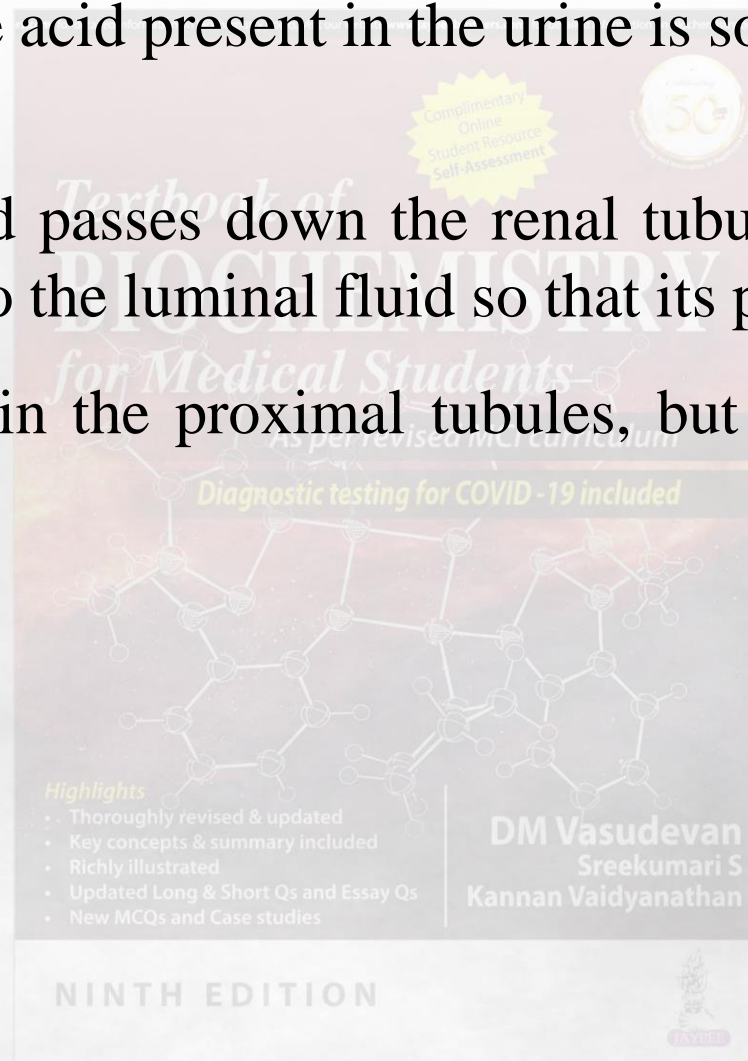
3.C. Excretion of H⁺ as Titratable Acid



The major titratable acid present in the urine is sodium acid phosphate.

As the tubular fluid passes down the renal tubules more and more H⁺ are secreted into the luminal fluid so that its pH steadily falls.

The process starts in the proximal tubules, but continues upto the distal tubules.



3.C. Excretion of H⁺ as Titratable Acid



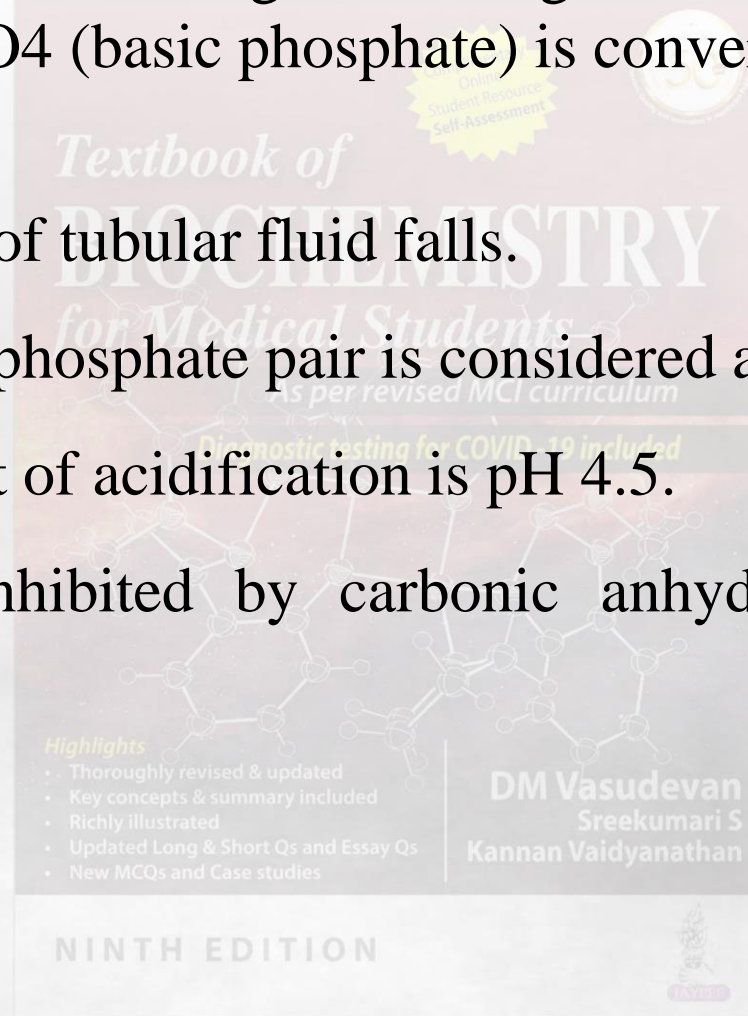
Due to the Na⁺ to H⁺ exchange occurring at the renal tubular cell border, the Na₂HPO₄ (basic phosphate) is converted to NaH₂PO₄ (acid phosphate) .

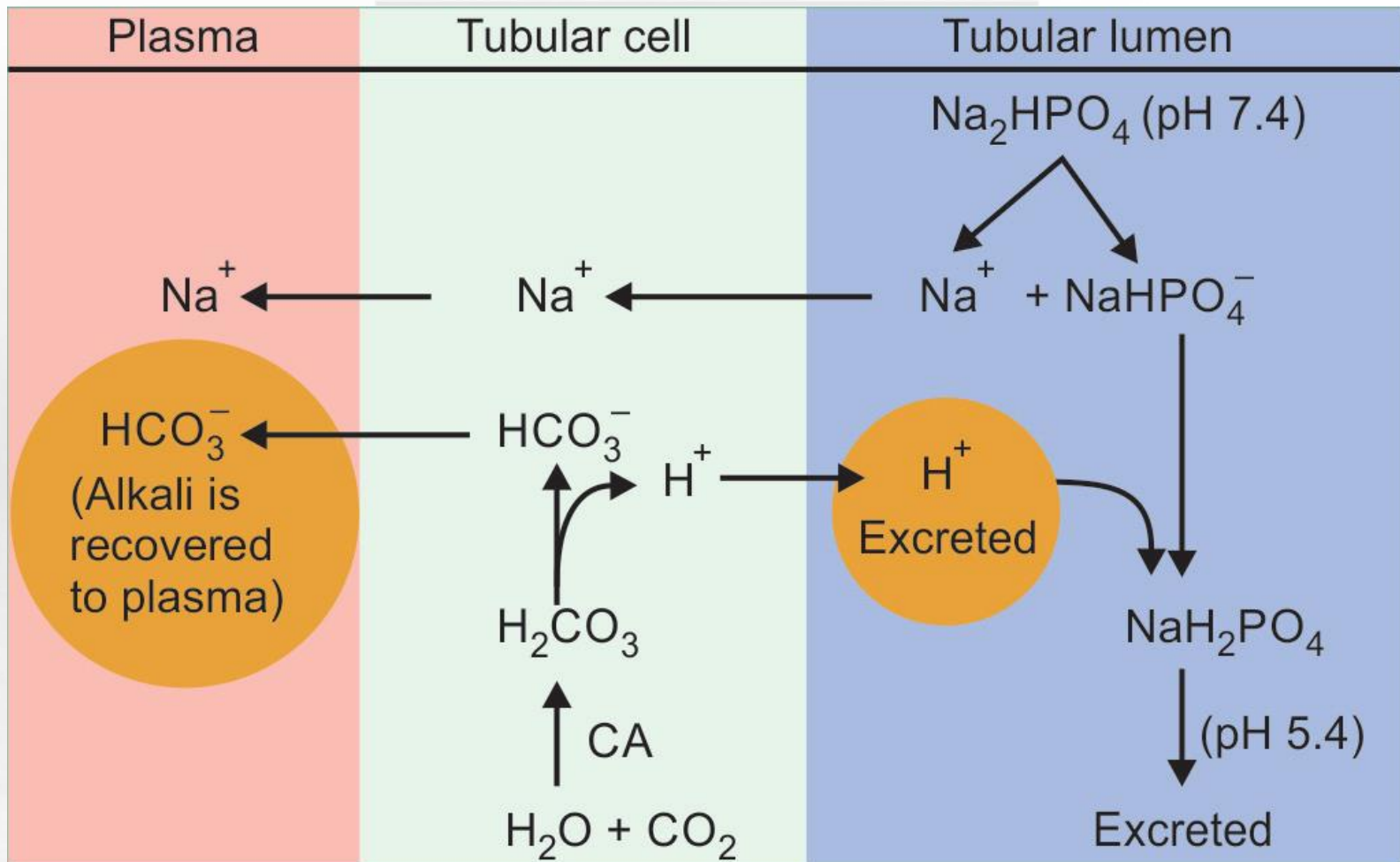
As a result, the pH of tubular fluid falls.

The acid and basic phosphate pair is considered as the urinary buffer.

The maximum limit of acidification is pH 4.5.

This process is inhibited by carbonic anhydrase inhibitors like acetazolamide.





Phosphate Mechanism in Tubules

3.D. Excretion of Ammonium Ions

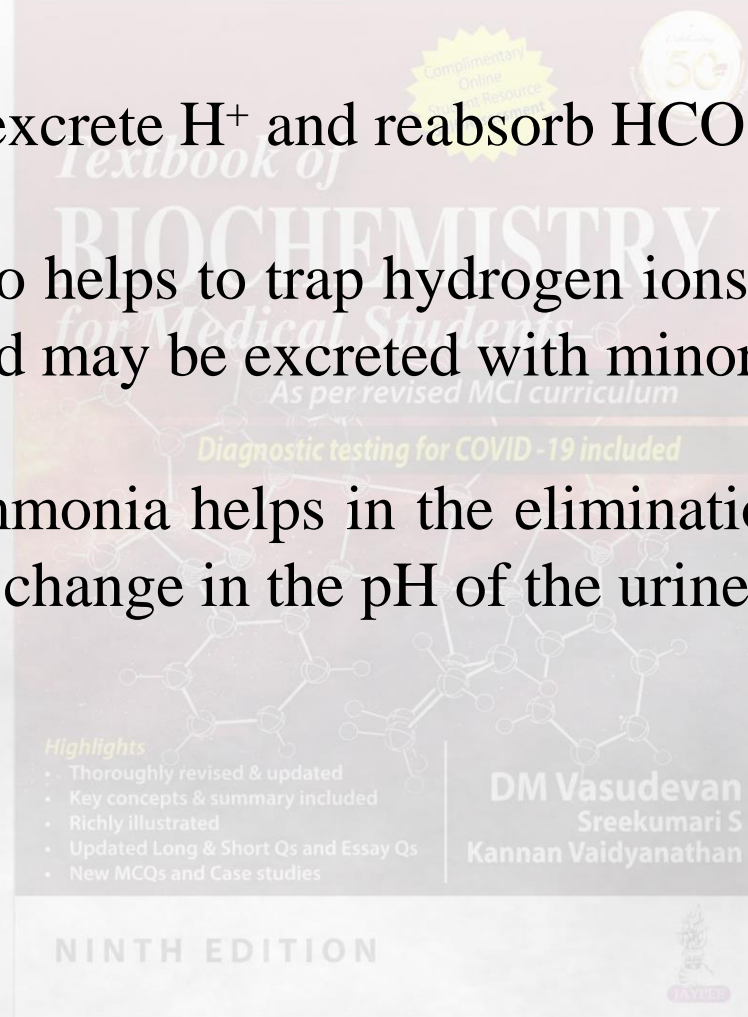


Predominantly occurs at the distal convoluted tubules.

This would help to excrete H^+ and reabsorb HCO_3^-

This mechanism also helps to trap hydrogen ions in the urine, so that large quantity of acid may be excreted with minor changes in pH.

The excretion of ammonia helps in the elimination of hydrogen ions without appreciable change in the pH of the urine.



3.D. Excretion of Ammonium Ions

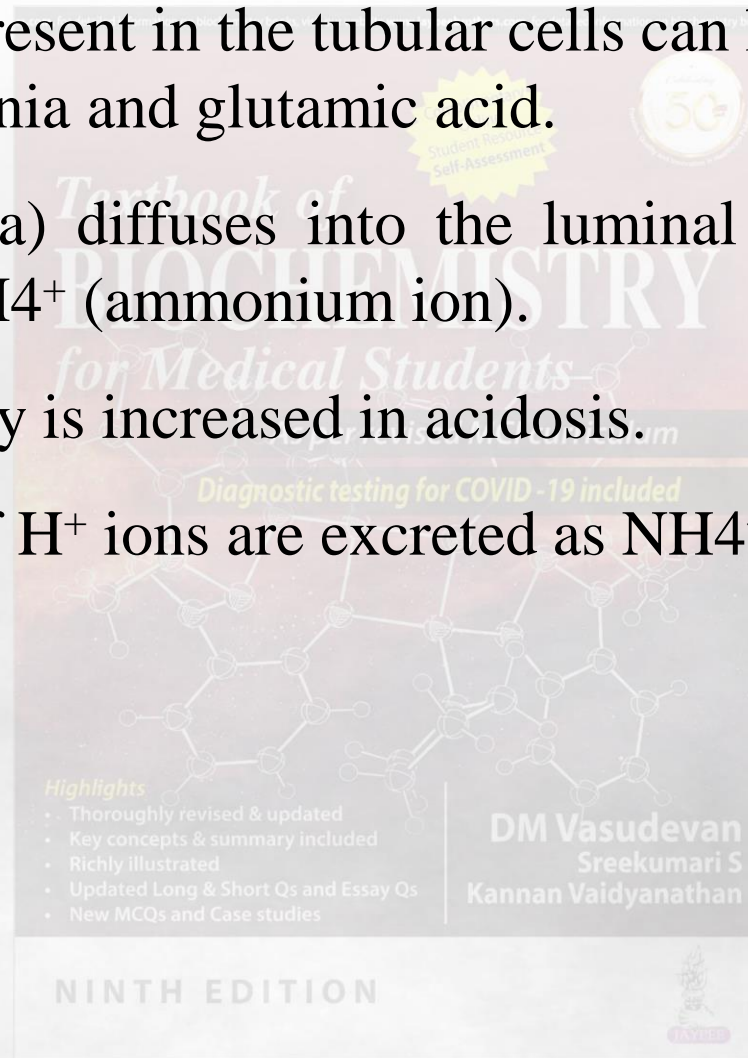


The Glutaminase present in the tubular cells can hydrolyze glutamine to ammonia and glutamic acid.

The NH_3 (ammonia) diffuses into the luminal fluid and combines with H^+ to form NH_4^+ (ammonium ion).

Glutaminase activity is increased in acidosis.

So large quantity of H^+ ions are excreted as NH_4^+ in acidosis.



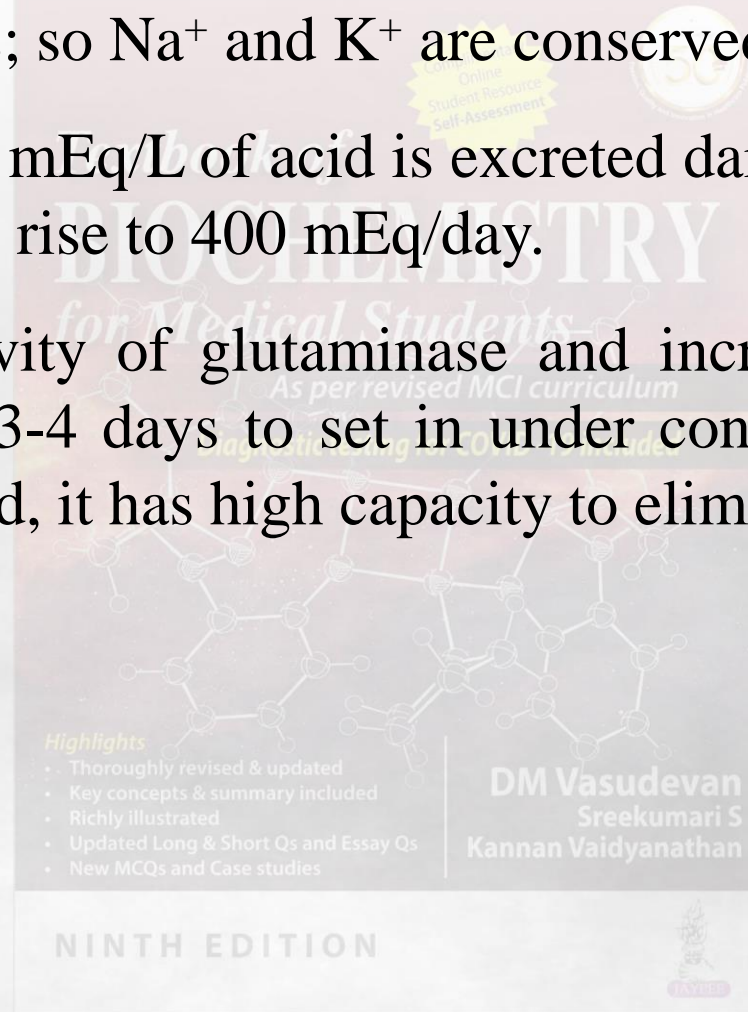
3.D. Excretion of Ammonium Ions



Since it is a positively charged ion, it can accompany negatively charged acid anions; so Na^+ and K^+ are conserved.

Normally, about 70 mEq/L of acid is excreted daily; but in condition of acidosis, this can rise to 400 mEq/day.

The enhanced activity of glutaminase and increased excretion of NH_4^+ takes about 3-4 days to set in under conditions of acidosis. But once established, it has high capacity to eliminate acid.



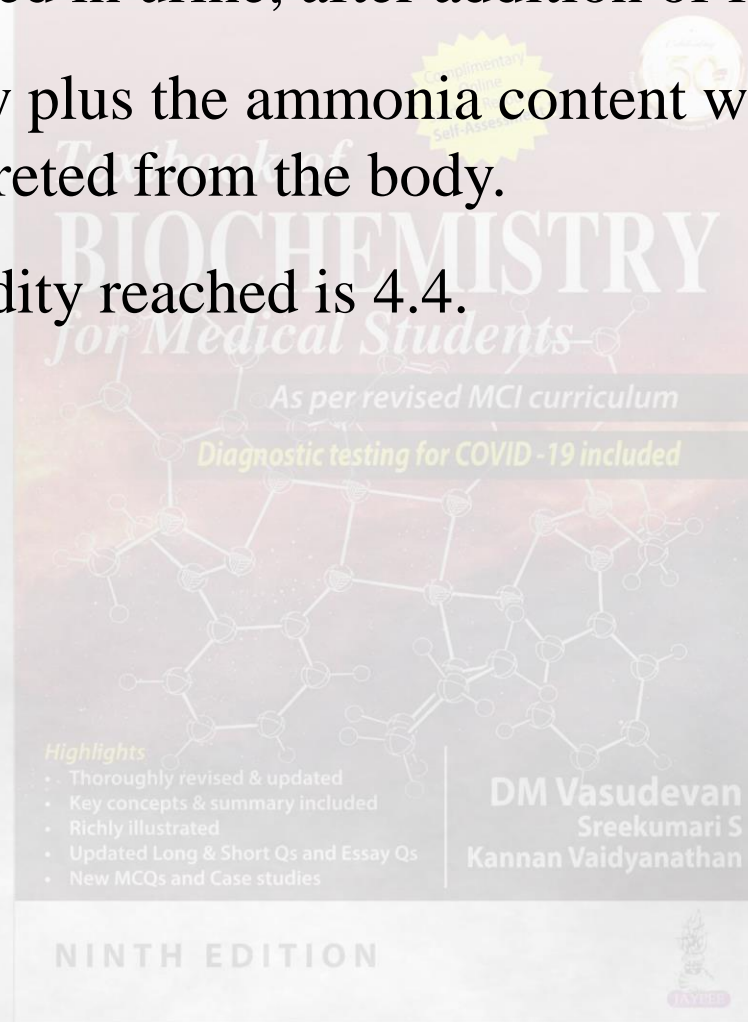
3.D. Excretion of Ammonium Ions

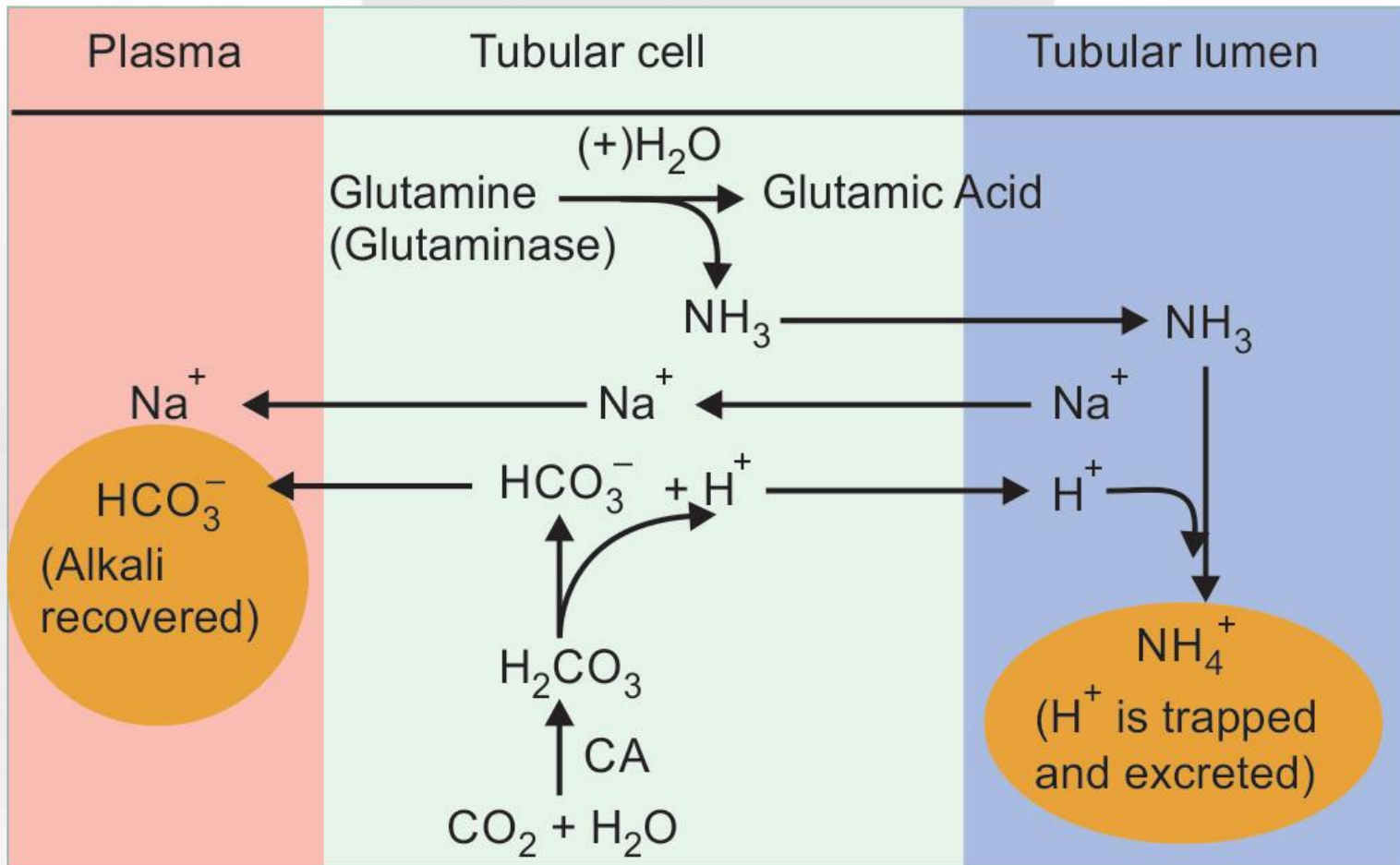


Ammonia is estimated in urine, after addition of formaldehyde.

The titratable acidity plus the ammonia content will be a measure of acid excreted from the body.

Maximum urine acidity reached is 4.4.



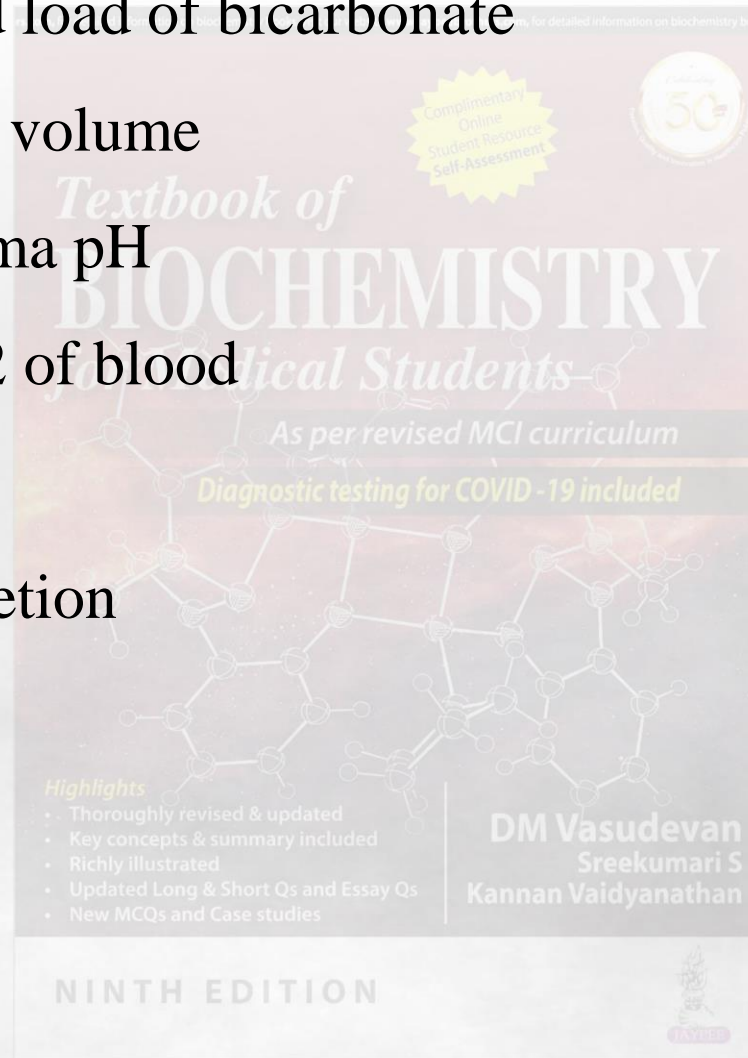


Ammonia Mechanism

Factors affecting Renal Acid Excretion



1. Increased filtered load of bicarbonate
2. Decrease in ECF volume
3. Decrease in plasma pH
4. Increase in $p\text{CO}_2$ of blood
5. Hypokalemia
6. Aldosterone secretion



Relationship of pH with K⁺ Ion Balance

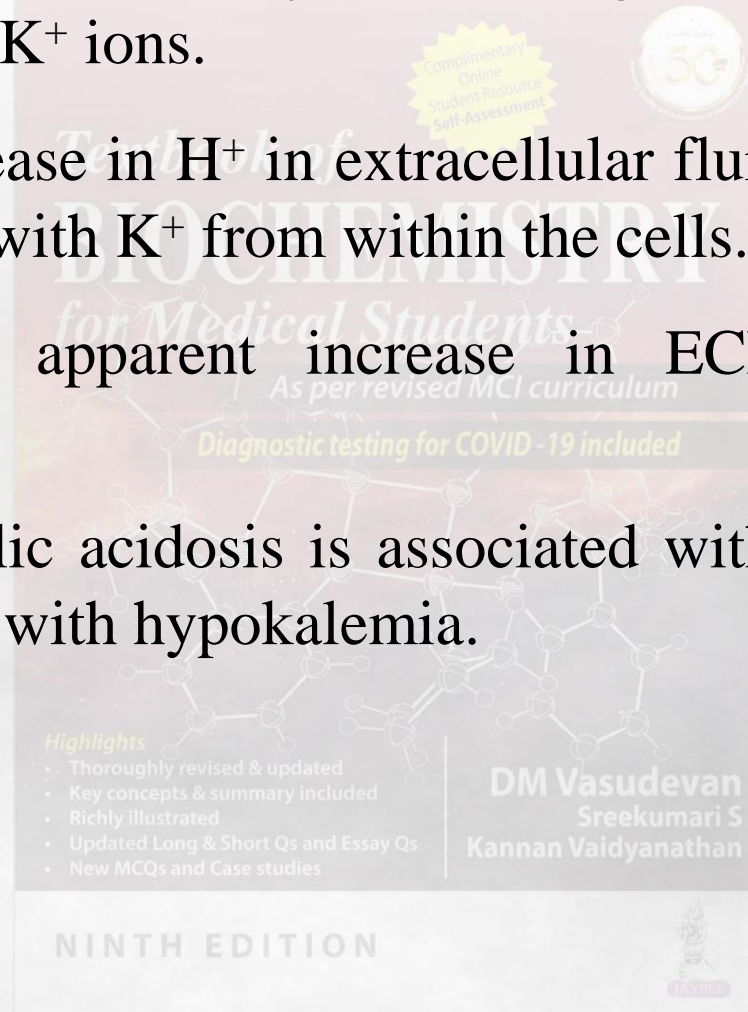


Buffering of acid is achieved by the exchange of H⁺ that enters into the cells for Na⁺ or K⁺ ions.

When there is increase in H⁺ in extracellular fluid (ECF), there may be exchange of H⁺ with K⁺ from within the cells.

Net effect is an apparent increase in ECF potassium level (hyperkalemia).

In general, metabolic acidosis is associated with hyperkalemia and metabolic alkalosis with hypokalemia.



Relationship of pH with K⁺ Ion Balance

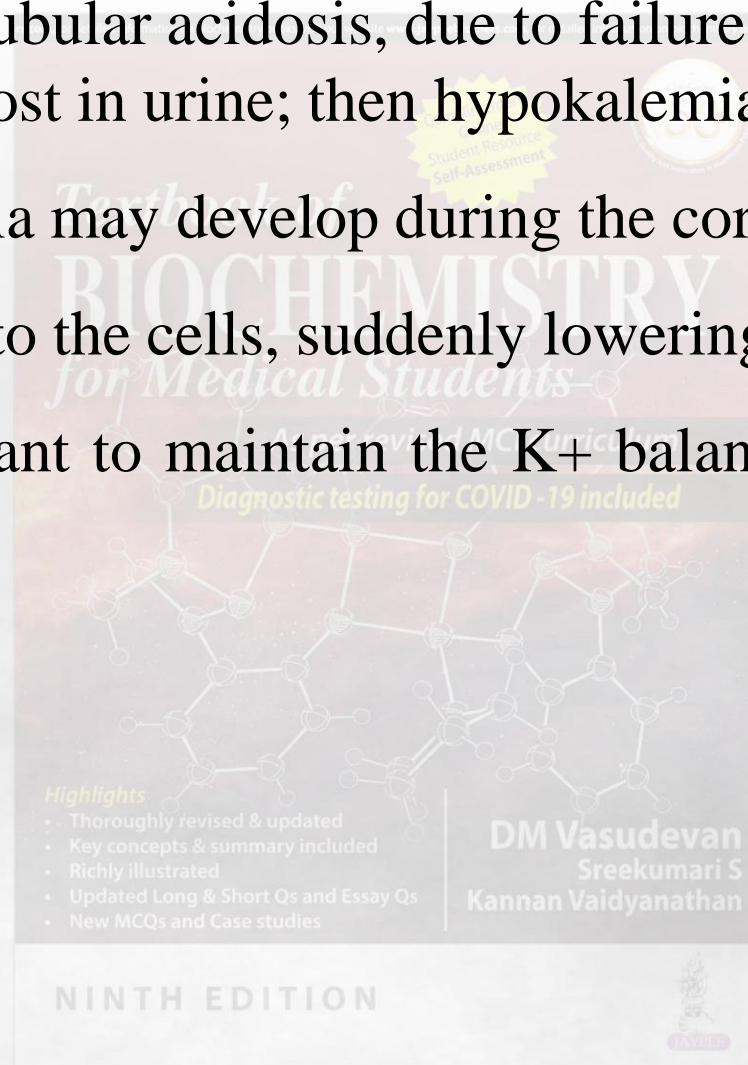


However, in renal tubular acidosis, due to failure to excrete hydrogen ions, potassium is lost in urine; then hypokalemia results.

Sudden hypokalemia may develop during the correction of acidosis.

K⁺ may go back into the cells, suddenly lowering the plasma K⁺.

Hence, it is important to maintain the K⁺ balance during correction of acidosis.



Buffering against Acid Load

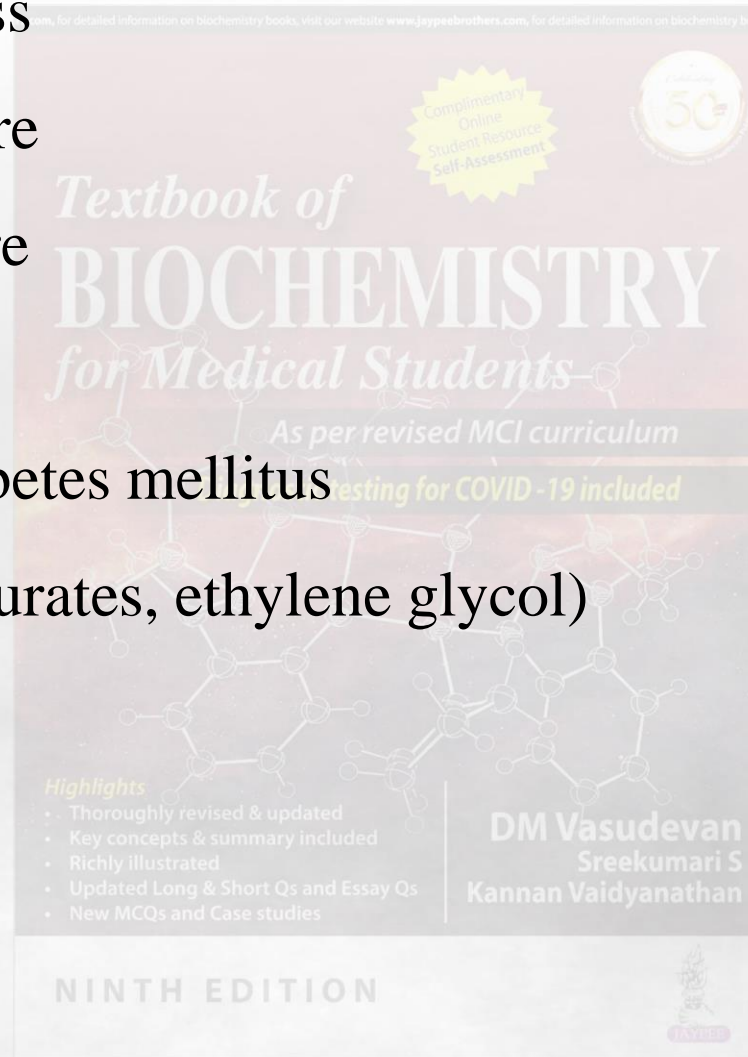


Stages	Features	Buffer components
Normal	Normal ratio = 20:1 Normal pH = 7.4	$\frac{\text{HCO}_3^- \text{ (N)}}{\text{H}_2\text{CO}_3 \text{ (N)}}$
First line of defense Plasma buffer system	Acidosis; H^+ enters blood, bicarbonate is used up	$\text{HCO}_3^- \text{ (}\downarrow\downarrow\text{)}$
Second line defense Respiratory compensation	Hyperventilation $\text{H}_2\text{CO}_3 \rightarrow \text{H}_2\text{O} + \text{CO}_2 \uparrow$	$\text{H}_2\text{CO}_3 \text{ (}\downarrow\text{)}$
Partially compensated acidosis	Bicarbonate \downarrow ; pH \downarrow	$\frac{\text{HCO}_3^- \text{ (}\downarrow\downarrow\text{)}}{\text{H}_2\text{CO}_3 \text{ (}\downarrow\downarrow\text{)}}$
Third line of defense kidney mechanism	Excretion of H^+ ; Reabsorption of bicarbonate; Ratio and pH tend to restore	$\frac{\text{HCO}_3^- \text{ (}\downarrow\downarrow\text{)}}{\text{H}_2\text{CO}_3 \text{ (}\downarrow\downarrow\text{)}}$

Acid-base Parameters are to be Checked in Patients with



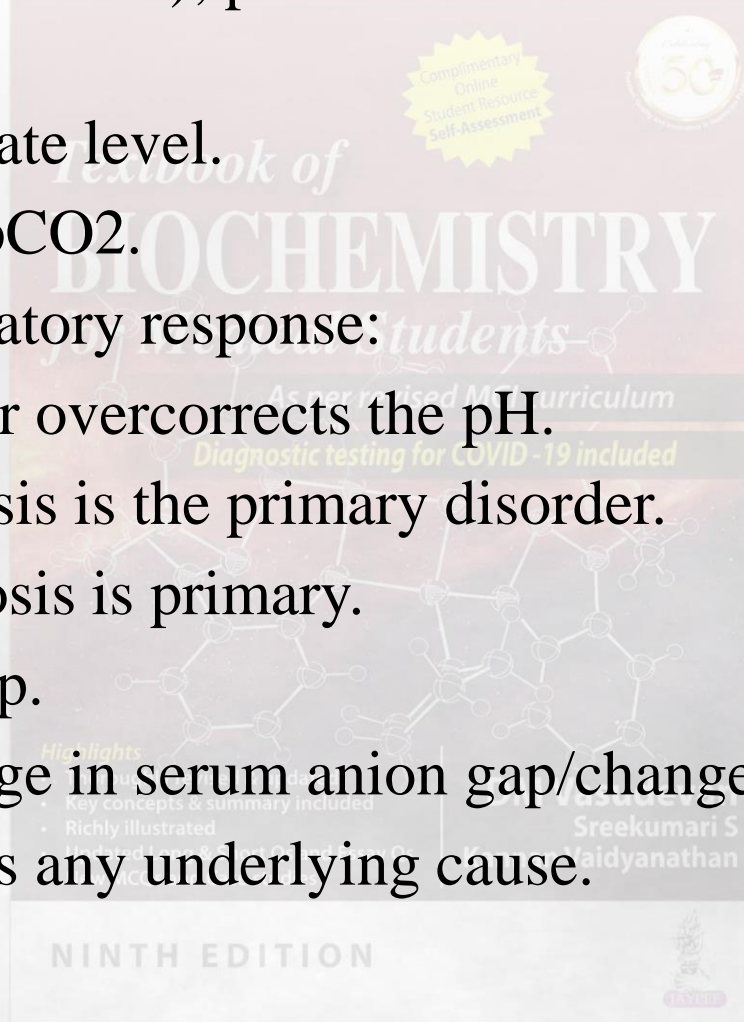
1. Any serious illness
2. Multi-organ failure
3. Respiratory failure
4. Cardiac failure
5. Uncontrolled diabetes mellitus
6. Poisoning (barbiturates, ethylene glycol)



Steps to the Clinical Assessment



1. Assess pH (normal 7.4); pH <7.38 is acidemia and >7.42 is alkalemia
2. Serum bicarbonate level.
3. Assess arterial pCO₂.
4. Check compensatory response:
Compensation never overcorrects the pH.
If pH is <7.4, acidosis is the primary disorder.
If pH is >7.4, alkalosis is primary.
5. Assess anion gap.
6. Assess the change in serum anion gap/change in bicarbonate.
7. Assess if there is any underlying cause.



Classification of Acid-Base Disturbances



1. Acidosis (fall in pH)

- a. **Respiratory acidosis**: Primary excess of carbonic acid.
- b. **Metabolic acidosis**: Primary deficit of bicarbonate.

2. Alkalosis (Rise in pH)

- a. **Respiratory alkalosis**: Primary deficit of carbonic acid.
- b. **Metabolic alkalosis**: Primary excess of bicarbonate

3. Compensatory responses

- a. Uncompensated
- b. Partially compensated
- c. Fully compensated.

