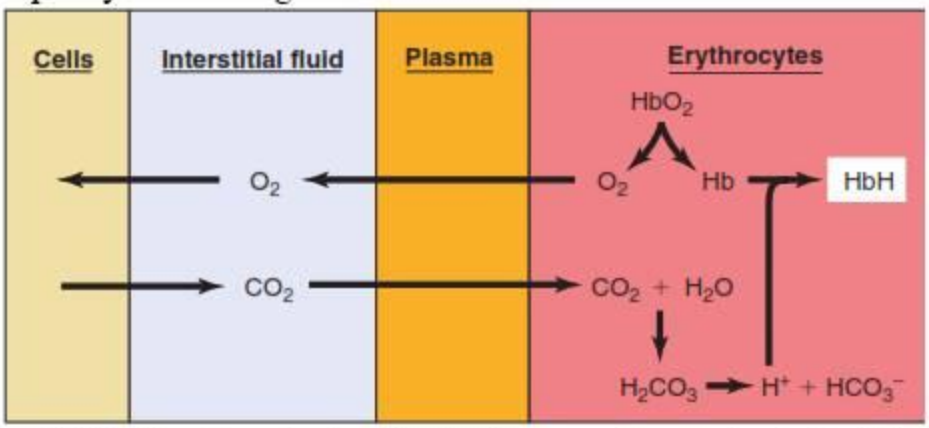
Acidosis and alkalosis

Blood hydrogen ion concentration [H^+] is maintained with in tight limits in health. Normal levels lie between 35 and 45 nmol/L. Values greater than 120 nmol/L or less than 20 nmol/L require urgent treatment.

A buffer: is a solution of a weak acid and its salt or a weak base and its salt that is able to bind H^+ and therefore resist changes in PH. Buffering does not remove H^+ from the body. Rather, buffers temporarily mop up any excess H^+ that is produced, in the same way that a sponge soaks up water. Buffering is only a short-term solution to the problem of excess H^+. Ultimately, the body must get rid of the H^+ by renal excretion. The body contains a number of buffers to even out sudden changes in H^+ production:

(1Proteins can act as buffers, and the haemoglobin in the erythrocytes has a high capacity for binding $H.^+$



(2In the ECF, bicarbonate buffer is the most important. In this buffer system, bicarbonate ($HCO3^-$) combines with H^+ to form carbonic acid (H_2CO_3). This buffer system is unique that the (H_2CO_3) can dissociate to water and carbon dioxide.

Whereas simple buffers rapidly become ineffective as the association of the H+ and the anion of the weak acid reaches equilibrium, the bicarbonate system keeps working because the carbonic acid is removed as carbon dioxide.

Only when all the bicarbonate is used up does the system have no further buffering capacity. The acid–base status of patients is assessed by consideration of the bicarbonate system of plasma.

Secretion of H+ by the tubular cells serves initially to reclaim bicarbonate from the glomerular filtrate so that it is not lost from the body. When all the bicarbonate has been recovered, any deficit due to the buffering process is regenerated. The excreted H^+ in urine [H^+] would rise to very high.

Acid–base disorders

are clinical terms that define the primary acid–base disturbance.

Tow type of acid –base disorders

A. „**Metabolic' acid–base disorders** are those that directly cause a change in the bicarbonate concentration. Examples include diabetes mellitus, where altered intermediary metabolism in the absence of insulin causes a build up of H^+, from it the ionization of acetoacetic and β-hydroxybutyric acids, or loss of bicarbonate from the extracellular fluid, e.g. from a duodenal fistula.

i. *Metabolic acidosis.* The primary disorder is a decrease in bicarbonate concentration.

ii. *Metabolic alkalosis.* The primary disorder is increased bicarbonate.

B. „**Respiratory' acid–base disorders** affect directly the PCO_2. Impaired respiratory function causes a build-up of CO_2 in blood, whereas, less commonly ‘ hyperventilation can cause a decreased PCO_2.

i. *Respiratory acidosis.* The primary disorder is an increased PCO_2

ii. *Respiratory alkalosis.* The primary disorder is a decreased PCO_2

Compensation

The simple relationships of the bicarbonate buffer system with complicated of physiological mechanisms that try to return a disordered $[H^+]$ to normal. Tow route for compensation:

A. Where lung function is compromised, the body attempts to increase the excretion of H^+ via the renal route. This is known as *renal compensation* for the primary respiratory disorder. Renal compensation is slow to take effect.

B. Metabolic disorders, some compensation is possible by the lungs. This is known as *respiratory compensation* for the primary metabolic disorder. Respiratory compensation is quick to take effect.

If compensation is complete, the $[H^+]$ returns to within reference limits, although the PCO_2 and $[HCO_3^-]$ remain grossly abnormal. The acid–base disorder is said to be „fully compensated."

Metabolic acidosis

In a metabolic acidosis the primary problem is a reduction in the bicarbonate concentration of the extracellular fluid.

The main causes of a metabolic acidosis are: Metabolic acidosis with an elevated anion gap occurs in:

i. Increased production of hydrogen ions:

- **Diabetic ketoacidosis**. Altered metabolism of fatty acids, as a consequence of the lack of insulin‹ causes endogenous production of acetoacetic and β-hydroxybutyric acids.
- **Lactic acidosis**. The presence of a lactic acidosis can be confirmed, if necessary, by the measurement of plasma lactate concentration.This results from a number of causes:

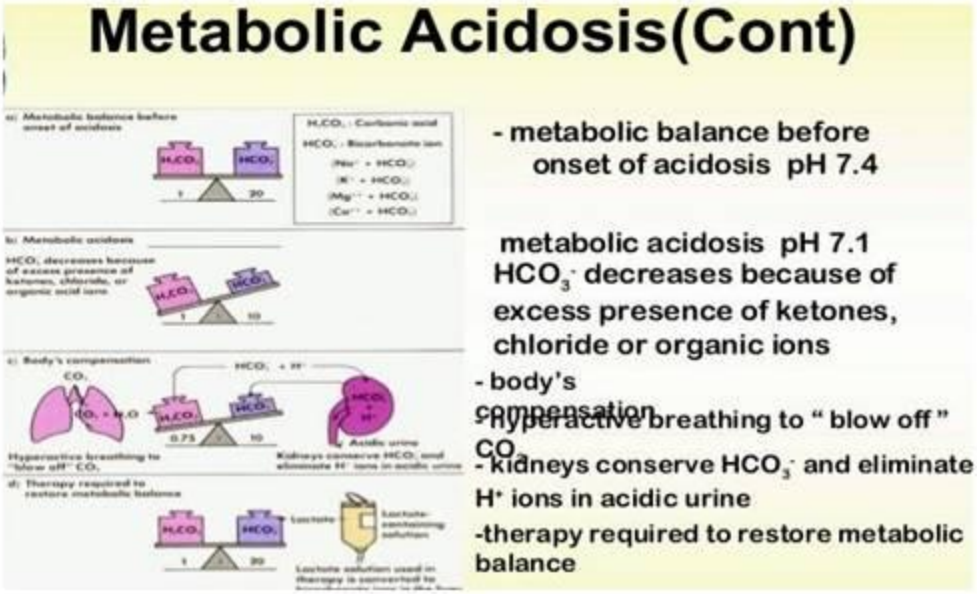
.1Particularly tissue anoxia. In acute hypoxic states such as respiratory failure or cardiac arrest lactic acidosis develops within minutes and is life-threatening.

.2Liver disease.

ii. Ingestion of hydrogen ions, or of drugs that are metabolized to acids: Certain cases of over dosage or poisoning. The mechanism common to all of these is the production of acid metabolites. Examples include salicylate overdose where build-up of lactate occurs, methanol poisoning when format accumulates, or ethylene glycol poisoning where oxalate is formed.

iii. Impaired excretion of hydrogen ions by the kidneys: *Renal disease*. Hydrogen ions are retained along with anions such as sulphate and phosphate.

iv. Loss of bicarbonate from the gastrointestinal tract or in the urine: *Renal tubular acidosis*. Renal tubular cells are unable to excrete hydrogen ions efficiently, and bicarbonate is lost in the urine.



❖ **Metabolic acidosis with a normal anion gap** is sometimes referred to as a „hyperchloraemic acidosis" because a reduced HCO_3 concentration is balanced by increased Cl^- concentration.

Clinical effects of acidosis

The compensatory response to metabolic acidosis is hyperventilation, since the increased $[H^+]$ acts as a powerful stimulant of the respiratory center. The deep, rapid and gasping respiratory pattern is known as Kussmaul breathing. Hyperventilation is the appropriate physiological response to acidosis and it occurs rapidly.

Metabolic alkalosis

The causes of a metabolic alkalosis are:

i. **Loss of hydrogen ion in gastric fluid during vomiting.** This is especially seen when there is pyloric stenosis preventing parallel loss of bicarbonate-rich secretions from the duodenum.

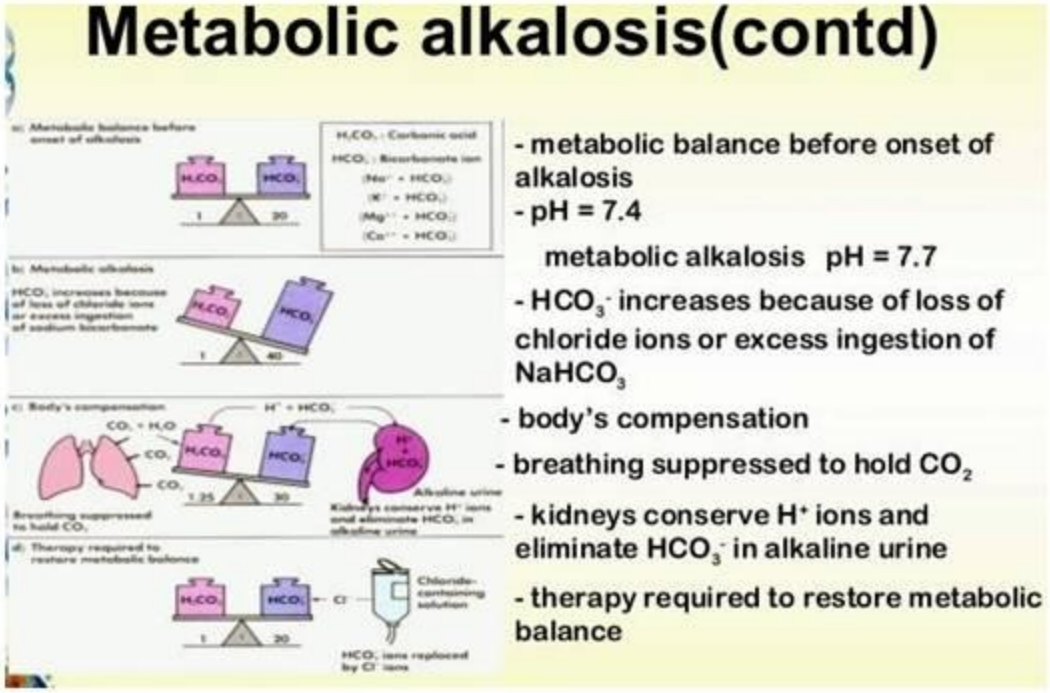
ii. ***Ingestion of an absorbable alkali such as sodium bicarbonate***. Very large doses are required to cause a metabolic alkalosis unless there is renal

iii. impairment.

Potassium deficiency. In severe potassium depletion, often a consequence of diuretic therapy, hydrogen ions are retained inside cells to replace the missing potassium ions. In the renal tubule more hydrogen ions, rather than potassium, are exchanged for reabsorbed sodium.

Clinical effects of alkalosis

The clinical effects of alkalosis include hypoventilation, confusion and eventually coma. Muscle cramps, tetany and paraesthesia may be a consequence of a decrease in the unbound plasma calcium concentration, which is a consequence of the alkalosis.



Respiratory acidosis

Respiratory acidosis may be acute or chronic. Acute conditions occur within minutes or hours. Renal compensation has long time to develop as the mechanisms that adjust bicarbonate reabsorption take 48–72 hours to become fully effective. The primary problem in acute respiratory acidosis is alveolar hypoventilation. If airflow is completely or partially reduced, the PCO_2 in the blood will rise immediately and the $[H^+]$ will raise quickly .A resulting low PO_2 and high PCO_2 causes coma. If this is not relieved rapidly, death results.

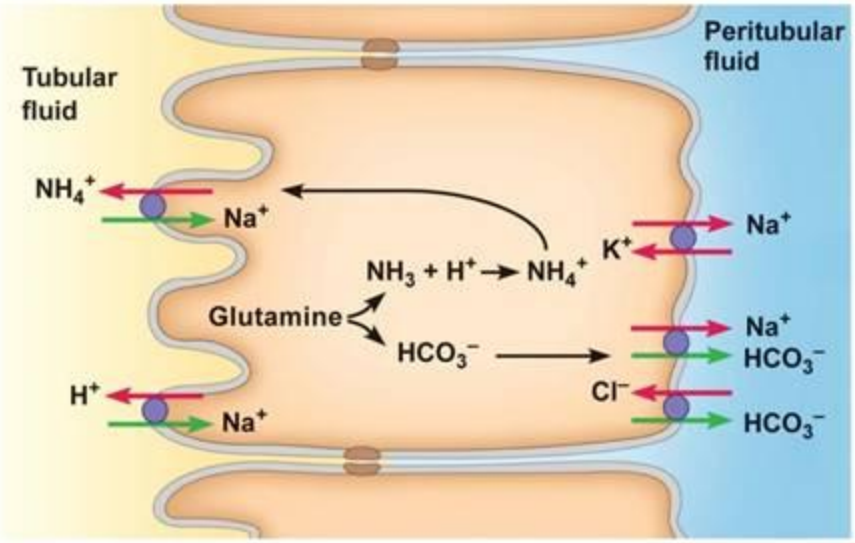
Examples of acute, and hence uncompensated, respiratory acidosis are:

i. choking
ii. bronchopneumonia
iii. acute exacerbation of asthma.

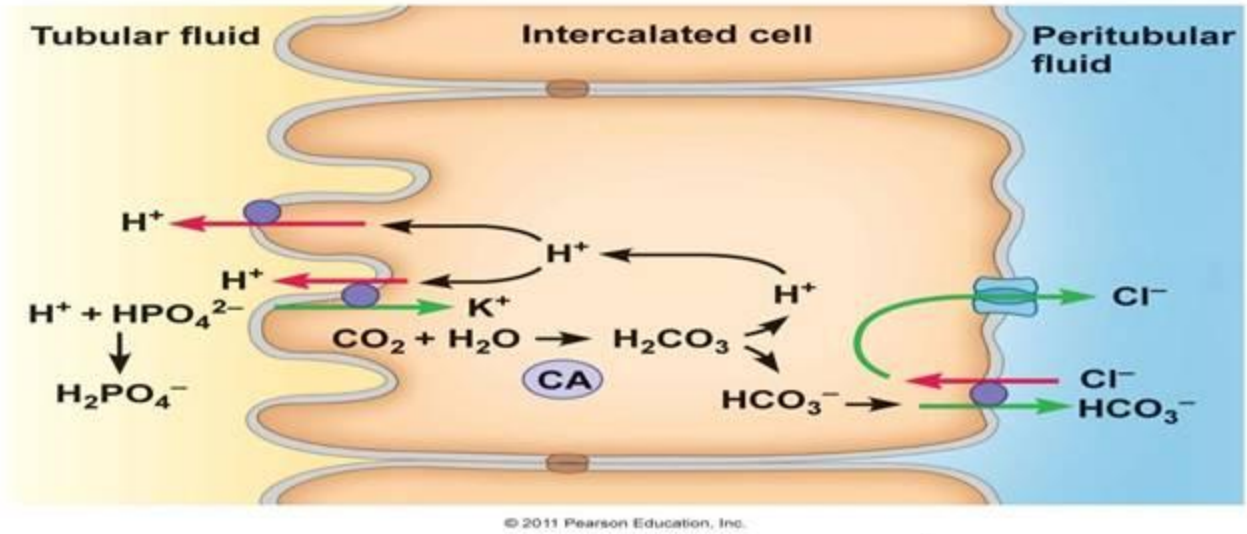
Chronic respiratory acidosis usually results from chronic obstructive airways disease)COAD) and is usually a longstanding condition, accompanied by maximal renal compensation. In a chronic respiratory acidosis the primary problem again is usually impaired alveolar ventilation, but renal compensation contributes markedly to the acid–base picture. Compensation may be partial or complete.

Compensation may mechanism:

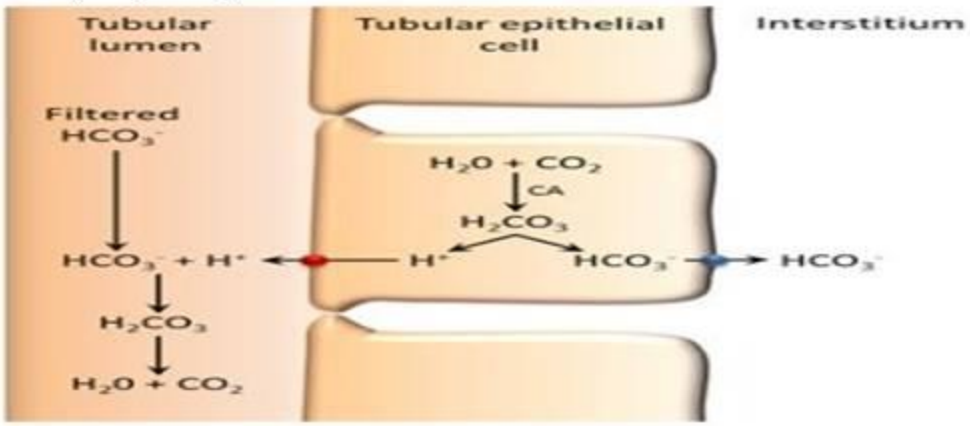
.1renal compensation for respiratory acidosis by glutamine:



.2 New HC03- is also produced if the supply of filtered HC03- is exhausted in the tubules by complete reabsorption. then the transported H+ is combined with filtered HPO42- and excreted



.3 Carbonic anhydrase (CA) catalyses the formation of H^+ and $HC0_3^-$ from water and CO_2. $HC0_3^-$ is transported into the blood and H^+ is transported into the urine where it is partially buffered by filtered HC.-03



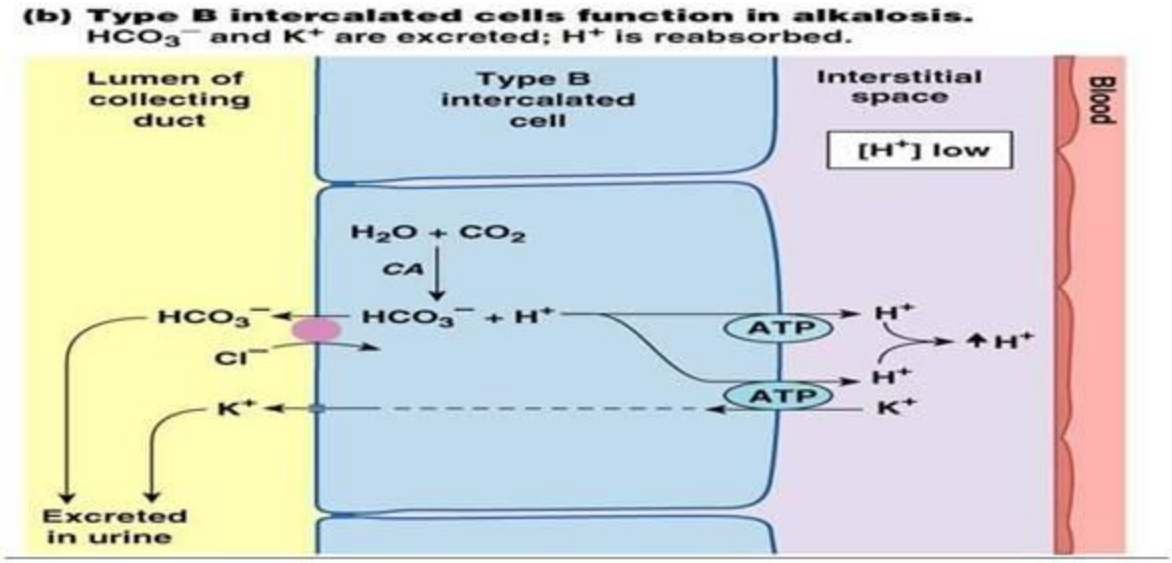
The kidney increases hydrogen ion excretion and ECF bicarbonate levels rise. Blood $[H^+]$ tends back towards normal. It takes some time for the kidneys to respond to a high PCO_2 and a high $[H^+]$, and therefore compensation will only be maximal some days after the onset of the clinical problem. In stable chronic bronchitis the $[H^+]$ may be within the reference interval despite a very high PCO_2. This is achieved only by maintaining a plasma bicarbonate concentration twice that of normal. Examples of chronic respiratory disorders are:

i. chronic bronchitis
ii. emphysema.

Respiratory alkalosis

Respiratory alkalosis is much less common than acidosis but can occur when respiration is stimulated or is no longer subject to feedback control. Usually these are acute conditions, and there is no renal compensation. Examples are:

i. hysterical over breathing
ii. mechanical over-ventilation in an intensive care patient
iii. raised intracranial pressure or hypoxia both of which may stimulate the respiratory center.



Mixed acid–base disorders

It is not uncommon for patients to have more than one acid–base disorder. A patient may have both a metabolic and respiratory acidosis, such as the chronic bronchitis patient who develops renal impairment. In such a patient with a raised $[H^+]$, the PCO_2 will be increased and the bicarbonate concentration will be low, both expected findings in primary respiratory and primary metabolic acidosis.

Where the two acid–base conditions are antagonistic in the way they affect the $[H^+]$, one of the disorders may mimic the compensatory response. A patient may present with a metabolic acidosis

and a coexistent respiratory alkalosis. The respiratory disorder may appear, at first sight, to be simply the compensatory response.

Other examples of mixed acid–base disorders commonly encountered are:

- A patient with chronic obstructive airways disease, causing a respiratory acidosis, with thiazide-induced potassium depletion and consequent metabolic alkalosis
- Hyperventilation causing a respiratory alkalosis, with prolonged nasogastric suction that causes a metabolic alkalosis
- Salicylate poisoning in which respiratory alkalosis occurs due to stimulation of the respiratory Centre, together with metabolic acidosis due to the effects of the drug on metabolism.