

PHSIOLOGY

Acid Base Balance 2



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Acidosis and Alkalosis

Brief revision *Ph of the blood is 7.4 Less than 7.4 = acidosis and more than 7.4 = alkalosis

*we have 3 defense mechanisms for acid-base regulation and balance:

Buffer system and its composed of the main 3 buffers of our body:
 A) Bicarbonate buffer
 B) phosphate buffer
 C) protein buffer
 All of those buffers are <u>RAPIDLY ACTING (RAPID MECHANISM)</u>

2) Respiratory mechanism: it's regulated by the concentration of those molecules (H+ = acidosis, CO2=hypercapnia, O2 = hypoxia) Which stimulates respiratory centers in the brain by chemoreceptors.

- 1- Central chemoreceptor which is more sensitive to hypercapnia
- 2- Peripheral chemoreceptors which are more sensitive to hypoxia

* Metabolic H+ stimulates the peripheral chemoreceptors only because it can't cross the BBB.

*CO2 can cross the BBB to react with H2O and converts into H2CO3 which rapidly dissociates into HCO3- AND H+. The reaction happens in the CSF near to the central chemoreceptors and H+ stimulates them.

*the respiratory mechanism is <u>AN INTERMEDIATE MECHANISM AND</u> <u>DOESN'T FULL CORRECT ACIDOSIS OR ALKALOSIS.</u> 3) The renal mechanism: which is delayed and fully corrects acidosis or alkalosis.

* The R.S mechanism has a certain limit to its acid-base regulation because as we washout CO2 its amount is decreased in our body whereas the amount of O2 is increased, until certain limit. As we reach that limit apnea will occur in order to increase the amount of CO2 back to its normal value and balance the amount of CO2 and O2 in our body

* the renal mechanism does full correction to acidosis by secreting H+ and reabsorbing HCO3- and this occurs in 2 parts of the nephron:

1- Proximal convoluted tubule (PCT): the H+ and HCO3- ions in the PCT come from this reaction which occurs in the PCT tubular cells. CO2+H2O=H2CO3=HCO3- and H+

* In acidosis we secrete H+ and reabsorb HCO3-

* in PCT H+ secretion occurs by NA+-H+ counter transport

2- Distal convoluted tubule (DCT): we have 2 cells that help in regulating our body acid-base balance:

A)Principal cells: which is the site of action for ALDOSTERONE. *those cells reabsorb H2O and Na+ and they secrete K+ and H+ under the influence of ALDOSTERONE.

- **B) Intercalated cells:**
- 1. Type A: correction of acidosis by secreting H+ to the tubular lumen and reabsorb HCO3-. They secrete H+ by the act of H+pump which is facing the luminal border of the cell.
- 2. Type B: it does the opposite of type A by secreting HCO3- to the tubular lumen and reabsorbing H+.
 * The H+ pump in this cell is facing the blood vessel so it's found in the basolateral border of the cell.

NOTE: H+ and K+ exchange so their levels are parallel in our body. So if we secrete H+ we will absorb K+ and vice versa. That process occurs by H+ and K+ counter transport.

<u>Acidosis</u>

Two types:

Respiratory acidosis. Due to pathology.
 2-Metabolic acidosis.

<u>1-Respiratory acidosis:</u>

Characterized by an <u>increase in PCO2</u> of arterial blood more than 45 mmHg, which is normally 40 mmHG, this leads to an <u>increase in H2CO3</u> in the blood, while <u>*HCO3 remains*</u> <u>*constant*</u>. (Accumulation of CO2)

Causes.

- 1. **Inadequate ventilation** to remove the produced CO2 leading to its retention as in <u>bronchial asthma</u> and <u>emphysema</u> that leads to inadequate alveolar ventilation.
- 2. **Disorders in diffusion of CO2** from the blood to alveolar air across the pulmonary membrane e.g. <u>pulmonary fibrosis</u>, <u>pulmonary edema</u> and <u>pneumonia</u>.
- 3. Disorders that lead to decreasing the movement of thoracic cage e.g. <u>chest trauma</u>, <u>deformities</u>, <u>weakness</u> or <u>paralysis of respiratory muscles</u> e.g. due to poliomyelitis. (other causes are myelitis, kyphosis, disk prolapse)
- 4. Conditions of decreased rate or depth of respiration caused usually by <u>depression of</u> <u>respiratory center</u> due to use of some narcotic drugs (e.g. morphine) or sedatives e.g. barbiturates . (morphine prevents the firing of neurons so in the Respiratory centers in the brain if large doses are taken by the patient respiratory centers will not give impulses to the muscles in the thoracic cavity thus the patient will die due to morphine toxicity)

Compensation.

Because the problem is of respiratory origin, the body cannot carry respiratory compensation, any compensation must be through renal mechanisms that secrete H+ and absorb HCO3

Secreted H+ allows urine to be more acidic

Reabsorption of HCO3 provides additional buffer that combines with H+ lowering the amount of free H+ and also raising the PH.

2-Metabolic Acidosis:

It occur when dietary and metabolic input of acids exceeds acid excretion Metabolic causes include. (Increase H+ from metabolic reaction)

- 1. Lactic acidosis which result from anaerobic metabolism.
- 2. **Keto- acidosis** when there is excessive breakdown of fats or certain amino acids. (Sulfur containing amino acids)
- 3. Ingested toxins that cause metabolic acidosis as methanol, aspirin, and ethylene glycol.

4. Diarrhea, due to loss of HCO3 from intestine.

* Respiratory acidosis causes an increased amount of Co2 which in result increases the amount of H+, whereas metabolic acidosis causes direct secretion of H+. Thus, we can say that respiratory acidosis causes increased amount of both CO2 and H+ whereas metabolic acidosis increases H+ concentration only

There is elevated H+ concentration Decreased HCO3 concentration.

Compensation.

Respiratory compensation: Elevated H+ and CO2 stimulate ventilation leading to decreased PCO2 to normal or even below normal due to hyperventilation. (Peripheral Chemoreceptor)

Renal compensation: increased secretion of H+, with Reabsorption of HCO3

• Renal compensation takes several days to reach full effectiveness so usually no seen in acute disturbances since it starts within 24 – 48 hours.

<u>Alkalosis</u>

Two types:

1-Respiratory alkalosis

2-Metabolic alkalosis.

<u>1-Respiratory alkalosis</u>.

Causes.

1-Hyperventilation due to wash of CO2.

Drop of CO2 decreases both H+ and HCO3, so low HCO3 in alkalosis indicate respiratory disorder, the most common physiological cause of respiratory alkalosis is <u>hysterical</u> <u>hyperventilation due to anxiety</u>.

The problem can be corrected by allowing the patient to <u>rebreathe the exhaled CO2</u> by allowing the patient to breath into a paper bag, the rise of arterial PCO2 corrects the problem.

ملاحظة جانبية: اللهث ناتج عن التوتر وبما ان لا جهد عضلي بذل يؤدي ألى انخفاض شديد في تركيز ال CO2

2-Metabolic alkalosis.

Caused by.

1-Excessive vomiting of acidic gastric contents.

2-Excess ingestion of bicarbonate containing antacids.

In both cases the resulting alkalosis reduces H+ concentration.

Metabolic alkalosis is characterized by

Decreased H+ concentration and PCO2 Increased HCO3 concentration.

Compensation.

Respiratory compensation: is very rapid

Increased PH and drop of PCO2 depress ventilation leading to increased CO2 and creating more H+ and HCO3 so ventilatory compensation help to correct PH but elevates HCO3.

Renal compensation: by HCO3 excretion and H+ Reabsorption

(And inhibiting the Na+ - H+ counter transport in the PCT also HCO3- secretion and H+ reabsorption in the PCT.

In the DCT intercalated cells type B will secrete HCO3- and reabsorb H+ by the H+ pump which will transport H+ from the intercalated cell to the basolateral side of the cell (blood vessel side))

Some More Notes:

- Buffer system compensation: it's immediate (the fastest) and occurs in respiratory or metabolic acidosis or alkalosis.
- Diarrhea = Alkaline loss
- Vomiting = Acid loss
- Respiratory compensation is limited due to its sensitivity to O₂ (As the respiratory center undergo apnea it will be stimulated by hypoxia to rebreathe again)
- Whereas the Renal compensation has no limit (intercalated cells) \rightarrow full correction
- Peptic ulcer means hyper acidity of the stomach which in turn means that the stomach is making large amounts of HCL which hurt the wall of the stomach. So, to treat it we should inhibit the making of HCL in the stomach (by preventing the stomach secretion of H+) and give the patient antacids (bases) to neutralize the acidity in the stomach by HCO3-

