ACID BASE BALANCE

BY
NANA YANNICK

INTRODUCTION

- Acid: A chemical that can donate a Hydrogen ion (H^+ : proton) upon dissociation e.g. HCl and H_2CO_3.

$HCl \longrightarrow H^+ + Cl^-$

-Base: A chemical that can accept a hydrogen ion e.g. OH^- and HCO_3^- to form water and H_2CO_3 respectively

$OH^- + H^+ \longrightarrow H_2O$

- Some chemicals are both acids and bases (Ampholytes) because they can donate and accept H+ e.g. $H_2PO_4^-$

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

$H_2PO_4^- + H^+ \longrightarrow H_3PO_4$

NORMAL ACID BASE PHYSIOLOGY

-A normal adult diet contains 70 to 100mmols of acid.

-There exist buffers that minimises any changes in blood P^H that this ingested H^+ might cause.

-Such buffers include intracellular proteins (Hb) and tissue components ($CaCO_3$ and $Ca_3PO_4^-$ in bones)

- The most important is the Hydrogen carbonate/carbonic acid buffer system expressed in Handerson-Hasselbalch equation

$$P^H = P^K + \log \frac{[HCO_3^-]}{[H_2CO_3]}$$

- $H^+ + HCO_3^- \rightleftharpoons H_2CO_3 \rightleftharpoons CO_2 + H_2O$
- Increase in H^+ leads to a fall in P^H is termed acidaemia while decrease in H^+ and a rise in P^H is termed Alkalaemia

CONTROL OF BLOOD P^H

- Blood P^H is tightly regulated and maintained between 7.38 and 7.42.
- Deviation causes a change in H^+ concentration because blood P^H is $-\log[H^+]$
- Increase acidity is due to production of CO_2 during cell respiration
- This dissociates into H^+, HCO_3^-. The H^+ are buffered by Hb

RENAL REGULATION OF ACID BASE BALANCE

- Kidney plays a major role in the control of acid base balance

-Mechanisms involved facilitate understanding of the pathophysiology and treatment of metabolic acidosis.

The control depends on:

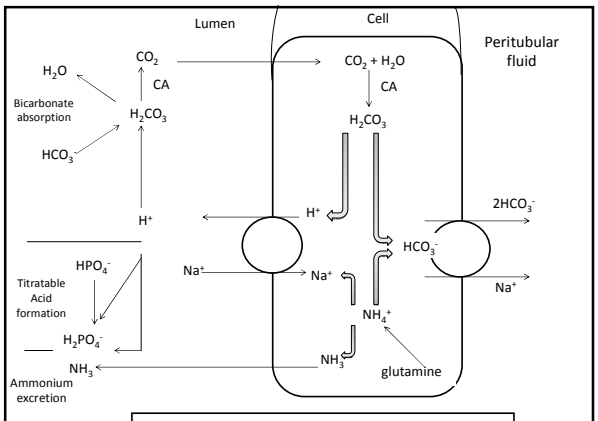
1.Reabsorption of filtered bicarbonates from tubular fluid.

2.Excretion of H^+ and NH_3 into tubular fluid.

BICARBONATE REABSORPTION IN THE PROXIMAL CONVOLUTED TUBULE(PCT)

- About 85% of filtered bicarbonates is reabsorbed in the PCT. The following steps occurs simultaneously
- Reabsorption is indirect in that H^+ excreted into the lumen combines with HCO_3^- to form H_2CO_3
- Breakdown of H_2CO_3 to H_2O and CO_2 catalysed by carbonic anhydrase in the brush border cell.

- CO_2 enters the cell combines again with H_2O catalysed by intracellular carbonic anhydrase
- H_2CO_3 dissociates to H^+ and HCO_3^-.
- H^+ is excreted into the lumen coupled to the reabsorption of Na^+ by a H^+-Na^+ antiport pump while HCO_3^- is returned to the blood by Na^+.HCO_3^- cotransport.



Mechanism of Bicarbonate Absorption and NH_3 excretion

AMMONIUM EXCRETION

-The Kidney produces NH_4^+ from glutamine metabolism.

-This occurs in the PCT

-$NH4^+$ rather than NH_3 is produced

-$NH4^+$ dissociates to NH_3 which diffuses out of the tubular cell into the luminal fluid

-NH_3 produced in the PCT cells is reabsorbed in the loop of henlé and the final excretion is the distal nephron

MECHANISM OF ACIDOSIS AND ALKALOSIS

- Blood PH depends on the HCO_3^-/Pa CO_2 ratio
- A change in HCO_3^- concentration leads to a change in P^H.
- A reduction in P^H caused by a primary decrease in HCO_3^- is called metabolic Acidosis
- Increase in P^H caused by primary increase in HCO_3^- is called metabolic alkalosis

- Increase in P^H resulting from a primary decrease in Pa CO_2 is called respiratory alkalosis
- Decrease in P^H resulting from primary increase in Pa CO_2 is called respiratory alkalosis

CAUSES OF ACID BASE DISTURBANCE

- Abnormal CO_2 removal in the lungs (respiratory acidosis and alkalosis)
- Abnormalities in the regulation of bicarbonates and buffers in blood (metabolic acidosis and alkalosis).
- Metabolic acidosis causes hyper-ventilation, this increase removal of CO_2 from the lungs and partially compensates for acidosis

-Respiratory acidosis causes retention of CO_2, hence Pa CO_2 and $[H^+]$rise.

-Renal retention of bicarbonate may partly compensate, returning the H^+ towards normal

ANION GAP

-This is to identify if acidosis is due to retention of H^+ Cl^- or to another acid.

-Examples of anions present in the plasma are Cl^- and HCO_3^- while cations present in the plasma are Na^+ and K^+

$[Na^+] + [K^+]-[HCO_3^-] +[Cl^-]$= Anion Gap

PATHOLOGIES OF ACID/BASE BALANCE

Metabolic Acidosis With A Normal Anion Gap

-If the anion Gap is normal in the presence of Acidosis, it suggests that H^+, Cl^- is retained or Na^+, HCO_3^- is being lost.

RENAL TUBULAR ACIDOSIS

- This is systemic acidosis caused by impairment of the renal tubule to maintain a normal acid base balance.

TYPE I RENAL TUBULAR ACIDOSIS (RTA)

-This is due to failure of H+ excretion in the distal tubule.

TYPE II RTA

-Caused by failure of $NaHCO_3$ reabsorption in the proximal convoluted tubule

TYPE III RTA

- Caused by mutations resulting in carbonic anhydrase type II deficiency

TYPE IV RTA

-Here plasma Renin and Aldosterone are found to be low even after measures which will normally stimulate their secretion.

METABOLIC ACIDOSIS WITH A HIGH ANIONIC GAP

- If the anionic gap increases, one may conclude that an unmeasured anion is present in increased quantities

CAUSES

1.Lactic Acidosis: Increase lactic acid production occurs when cellular respiration is abnormal due to O_2 lack in the tissues.

2.Diabetic Ketoacidosis: results from accumulation of organic acids owing to increase production and some reduced peripheral utilisation.

3.Uraemic Acidosis: Kidney disease may cause acidosis by reducing the number of functional nephrons. This reduces the capacity to excrete NH_3 and H^+ in the urine.

THANKS FOR LISTENING