Acid base balance

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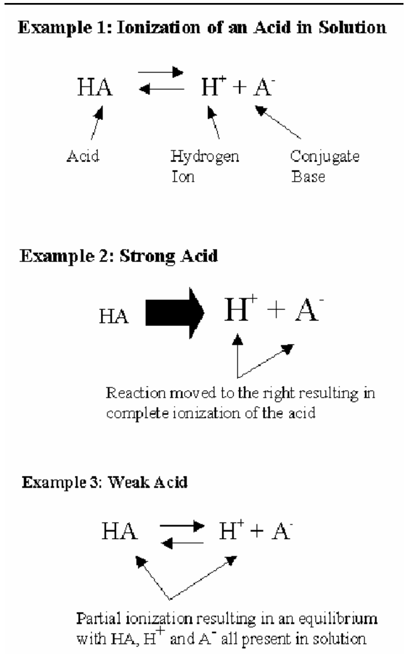
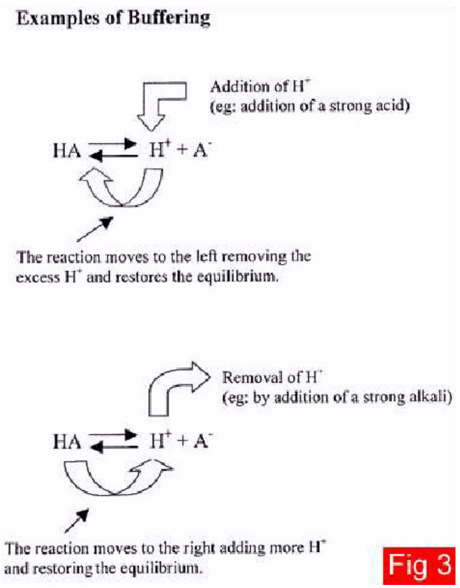
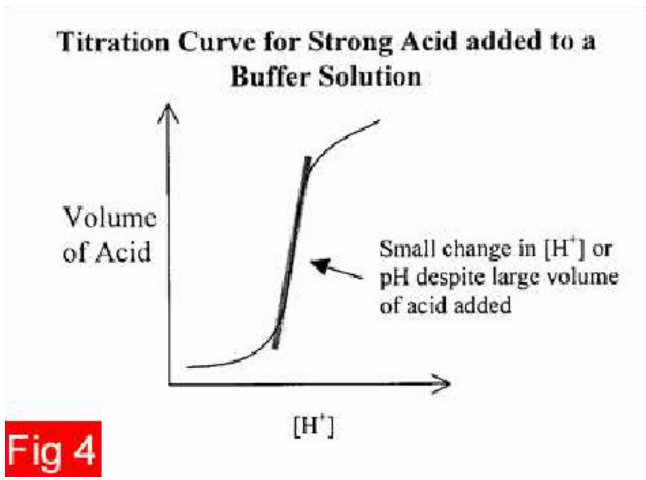
May 2004







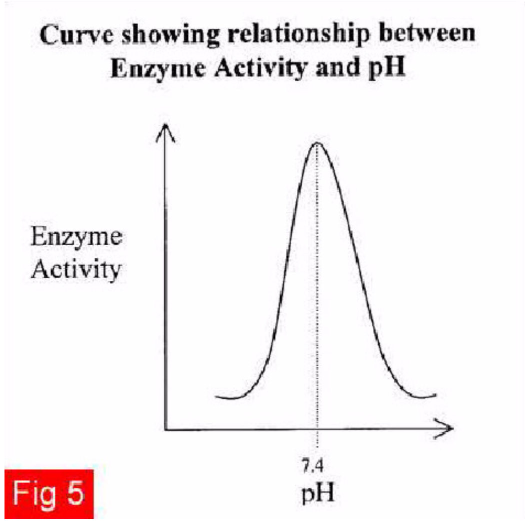


Fig 5

The equilibrium between basis and acid can be calculated and termed as the equilibrium constant = K_a. (sometimes referred as the dissociation constant)

In the reaction of a weak acid:
HA <-----> A^- + H^+

the equlibrium constant can be calculated from the following equation:
$K_a = [H^+][A^-]/[HA]$

As in the case of the ion product:
$pK_a = -\log K_a$

Therefore, in obtaining the -log of both sides of the equation describing the dissociation of a weak acid we arrive at the following equation:
$-\log K_a = -\log[H^+][A^-]/[HA]$

Since as indicated above $-\log K_a = pK_a$ and taking into account the laws of logrithms:
$pK_a = -\log[H^+] - \log[A^-]/[HA]$
$pK_a = pH - \log[A^-]/[HA]$

Henderson - Hasselbach equation:
$pH = pK_a + \log[A^-]/[HA]$
$pH = 6.1 + \log [HCO_3^-/(0.03 \cdot PCO_2)]$

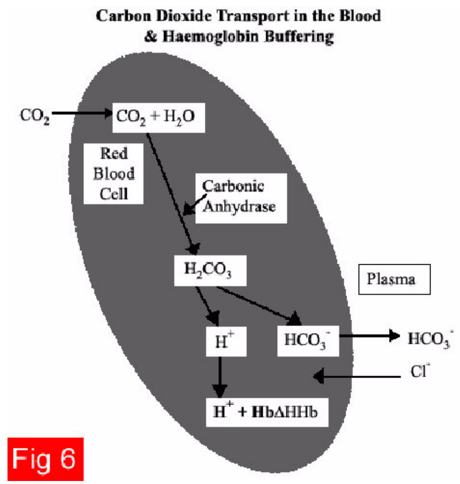


Fig 6

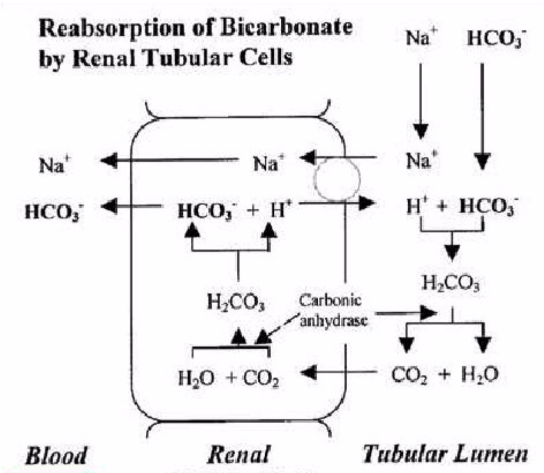


Fig 7

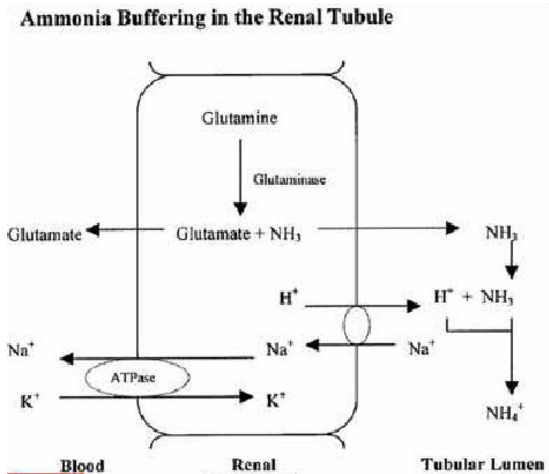


Fig 9

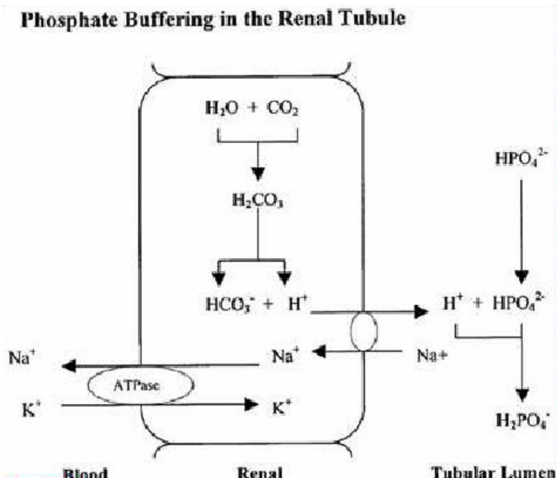


Fig 8

Terms and definitions

pH: - Log (Base 10) of [H+] in Mol/L
The pH is the negative logarithm of the hydrogen ion concentration
PCO2 is the partial pressure of carbon dioxide. The normal value in arterial blood is 40 mmHg (or 5.33 kPa)
Acidemia/Alkaliemia merely means that the blood pH is acid/alkaline compared to the normal pH of 7.4.

Bicarbonate was was defined as the bicarbonate concentration under given conditions: actual PCO2, actual temperature and pO_2

Standard Bicarbonate was defined as the bicarbonate concentration under standard conditions: PCO2=40 mmHg, and 37ºC, and saturated with oxygen.

Base Excess is quantity of acid or alkali required to return the plasma in-vitro to a normal pH under standard conditions

Standard Base Excess is the Base Excess value calculated for anemic blood (Hb = 5 g/dl) on the principle that this closely represents the behavior of the whole human being.
The rationale for this is that in the whole body, hemoglobin effectively buffers the plasma and the much larger extracellular fluid, i.e., the behavior is that of anemic blood. The method predicts the quantity of Acid or Alkali required to return the plasma in-vivo to a normal pH under standard conditions.

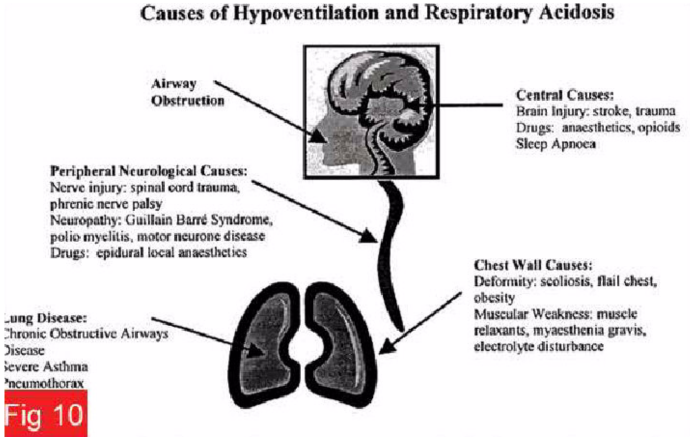
Anion Gap.

Gap = Na+ + K+ - Cl- - HCO3-
15 = 140 + 5 - 105 - 25 mMol/L

Some causes of metabolic acidosis, e.g., lactic acidosis, release anions into the extracellular fluid which are not normally measured. When this occurs there will be an unexpected discrepancy between the sums of the principal cations and anions.

Respiratory Acidosis (high PCO2)

causes molecules of CO2 and water to form carbonic acid which ionizes to increase both [HCO3-] and [H+]. The [H+] changes relatively slightly due to buffering of H+, mostly by hemoglobin. At this raised PCO2, the kidney compensates by reducing [H+]. To maintain chemical equilibrium the [HCO3-] rises further, i.e., respiratory acidosis raises the bicarbonate level and metabolic compensation raises it further. Occurs inhypoventilation



Metabolic Acidosis
is a pH which is too acid for the PCO2. Causes: increased acid production (diabetic ketoacidosis, pyruvate), decreased elimination (renal failure), poisoning (ethylenglykol)

Dilutional Acidosis:
Diluting the normal slightly alkaline mixture of extracellular electrolytes, also dilutes the alkalinity. This moves the pH closer to neutral at body temperature

Respiratory Akalosis (low PCO2)
is often caused by hyperventilation.

Metabolic alkalosis
is pH to alkaline for the PCO2. Is caused often by HCL loos.

Contraction Alkalosis:
When this electrolyte mixture is concentrated by dehydration, the relative alkalinity is more marked and the pH is further away from neutral.

Compensation
maintains the pH normal

Rapid Respiratory Compensation.
The power of the lungs to excrete large quantities of carbon dioxide enables them to compensate rapidly. Unless the respiratory system is diseased or depressed, metabolic disturbances stimulate a prompt response, i.e., metabolic acidosis and metabolic alkalosis normally elicit characteristic, partial respiratory compensation almost immediately.

Slow Metabolic Compensation.

The smaller capacity of the kidneys corresponds to a relatively slower rate of compensation; a patient can be ventilated at an abnormal PCO_2 for a day or two before the characteristic, partial compensation is achieved. In the operating room and in the emergency room, therefore, an abnormal PCO_2 is not usually associated with a metabolic "compensation". When a metabolic acidosis or alkalosis is detected, therefore, it usually reflects either a separate metabolic disturbance, or compensation for a chronic respiratory problem.