

Acid Base Online Tutorial



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GI Hydrogen Loss
 and Reduction in
 ECV

Contraction Alkalosis
 and Hypokalemia

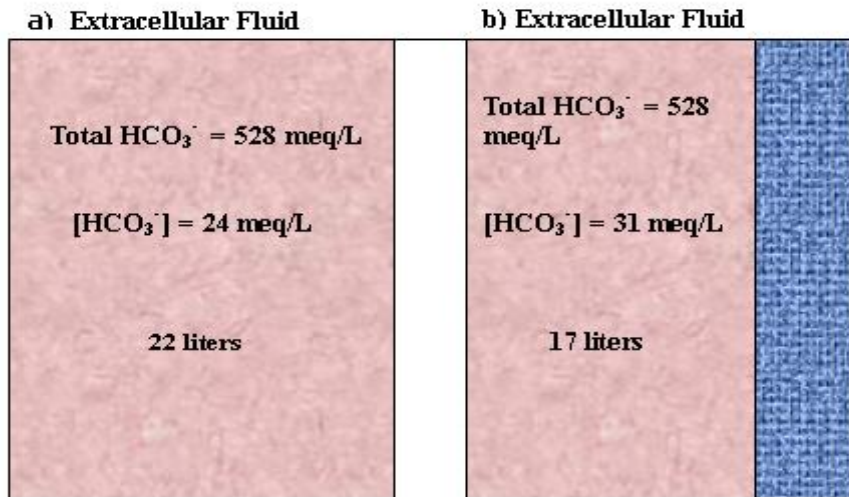
Posthypercapnia and
 Mineralocorticoid
 Excess

Diagnosis and
 Treatment of
 Metabolic Al

Contraction Alkalosis and Hypokalemia

Contraction alkalosis occurs whenever there is a loss in bodily fluid that does not contain HCO_3^- . In this setting, which is most commonly due to diuretics, the extracellular volume contracts around a fixed quantity of HCO_3^- resulting in a rise in $[\text{HCO}_3^-]$. Note that in this setting, the total body bicarbonate is the same as shown in the figure below.

The direct effect of contraction is largely minimized by the release of H^+ from cell buffers, thereby lowering the plasma $[\text{HCO}_3^-]$ toward normal. However, if ECF reduction by diuretics result in hypovolemia, then as in vomiting, the release of angiotensin and aldosterone will be stimulated. This then leads to an increase in HCO_3^- absorption and increased H and K secretion. The increase in potassium secretion result in the development of hypokalemia which also plays a very important role in maintaining the alkalosis.



a) 70 kg man whose extracellular volume has increased from 17 to 22 liters because of CHF.

b) If the excess NaCl is lost isototically after the administration of a diuretic, there will be a reduction in the ECF. Since the quantity of extracellular HCO_3^- is initially unchanged, the $[\text{HCO}_3^-]$ will increase from 24 to 31 meq/L.

Hypokalemia

Hypokalemia is very commonly associated with metabolic alkalosis. This is due to 2 factors: 1) the common causes of metabolic alkalosis (vomiting, diuretics, mineralocorticoid excess) directly induce both H^+ and K loss (via aldosterone) and thus also cause hypokalemia and 2) hypokalemia is a very important cause of metabolic alkalosis. Hypokalemia causes metabolic alkalosis by three mechanisms. The initial effect is by causing a transcellular

shift in which K leaves and H⁺ enters the cells, thereby raising the extracellular pH. The second effect is by causing a transcellular shift in the cells of the proximal tubules resulting in an intracellular acidosis, which promotes **ammonium production** and excretion. Thirdly, in the presence of hypokalemia, hydrogen secretion in the proximal and distal tubules increases. This leads to further reabsorption of HCO₃⁻. The net effect is an increase in the net acid excretion.

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