Posthypercapnia

The normal stimulus to respiratory acidosis is a compensatory increase in HCO3- reabsorption by the kidney and thus an increase in plasma [HCO3-]. Treatment with mechanical ventilation in this disorder leads to a rapid reduction in the pCO2. The plasma HCO3- will however remain elevated, resulting in the development of metabolic alkalosis. The maintenance of alkalosis in this setting is unclear. However chronic respiratory acidosis is thought to be associated with Cl- loss in the urine leading to hypovolemia and hypochloremia. Restoration of Cl- and volume balance tends to correct the disorder.

Mineralocorticoid excess

The common causes of metabolic alkalosis cause and maintain metabolic alkalosis due to **hypovolemia induced secondary hyperaldosteronism** which leads to increased acid excretion and hypokalemia. Conditions of mineralocorticoid excess such as Conn's syndrome, excess steroid administration and Cushing's syndrome produce a state of hyperaldosteronism which also leads to **metabolic alkalosis** and **hypokalemia.** In these conditions, the extracellular volume is expanded and the patient may have hypertension. In these patients, metabolic alkalosis is perpetuated by the effects of **hypokalemia** (not hypovolemia) which leads to increased ammonium production, hydrogen secretion and bicarbonate reabsorbtion

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