## Acid Base Online Tutorial

Introduction Pearls Acid Base Physiology

Acid Base Abnormalities

Cases

Back

## GI Hydrogen Loss and Reduction in ECV

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Contraction Alkalosis and Hypokalemia

Posthypercapnia and Mineraldocorticoid Excess

Diagnosis and Treatment of Metabolic Alkalosis Loss of hydrogen ions leading to metabolic acidosis most commonly occurs via the GI tract in the form of **vomiting** or **nasogastric suction**. Gastric juice contains a high concentration of HCL and lesser concentration of KCL. Each meg of H+ ion secreted generates 1 meg of HCO3- which is then absorbed in the plasma. Under normal conditions the increase in the plasma HCO3- concentration is only transient. since the entry of acid into the duodenum stimulates an equal amount of HCO3- secretion from the pancreas. However there is no stimulus to HCO3- secretion if gastric juice is removed during vomiting and NG suction. The net result is an increase in the plasma IHCO3-1 and metabolic alkalosis.

Under normal conditions, the excess HCO3- generated would be excreted in the urine by the kidney and thus alkalosis would not be maintained. However vomiting or nasogastric suction also results in a decrease in the extracellular fluid compartment and the effective circulating volume (ECV). The reduction in the ECV leads to decreased GFR (less bicarb filtered), and also serves as a stimulus to increase angiotensin and aldosterone production leading to an increase in Na and HCO3- reabsorption by the proximal tubules. An increase in Na reabsorption leads to increased HCO3- reabsorption because of the increase in hydrogen secretion as Na is exchanged for H+ across the Na-H+ transporter in the proximal tubule. The secreted hydrogen ions combine with filtered HCO3- leading to reabsorption as previously described. Aldosterone primarily acts distally to increase H+ and K secretion resulting in increased acid and potassium excretion. The net result is a hypokalemic metabolic alkalosis. The almost complete reabsorption of HCO3- in the setting of reduced ECV. leads to the paradoxical finding of an acidic urine despite the presence of extracellular alkalosis.

NEXT >

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