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ST VINCENT'S PATHOLOGY

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BICARBONATE INFORMATION SHEET

PHYSIOLOGY

Bicarbonate (HCO3-) is the major buffer in the circulation and the second most prevalent serum anion after chloride. Over time the bicarbonate concentration in the blood is regulated by the kidney to attempt to maintain a blood pH close to 7.4.

PATHOLOGY

Serum bicarbonate is closely related to the metabolic acid-base status of the patient. In nearly all circumstances a low serum bicarbonate indicates a metabolic acidosis and high serum bicarbonate indicates a metabolic alkalosis. Note that these changes may primary or may be secondary (compensatory) to primary respiratory disorders. For example, a primary respiratory acidosis (under-ventilation) produces high levels of hydrogen ions in the blood (academia) which leads to renal retention of bicarbonate and elevated serum bicarbonate reflecting the metabolic alkalosis induced to attempt to correct the primary disorder.

LOW SERUM BICARBONATE (METABOLIC ACIDOSIS)

Bicarbonate concentrations in the serum may fall due to consumption by combining with protons (H+) from acids such as lactic acid, keto-acids etc; or by loss from the body from gastro-intestinal or renal sources. Renal loss may be due to compensatory mechanisms for a respiratory alkalosis or renal disease, either non-specific in renal failure from any cause or due to the rarer renal tubular acidoses. Bowel causes of bicarbonate loss include diarrhoea and any cause of loss of biliary fluid.

- Compensatory for respiratory alkalosis (renal bicarbonate loss)
- Increased acid production: diabetic ketoacidosis, lactic acidosis, ingestion of methanol, ethanol etc.
- Decreased renal acid excretion: renal failure, renal tubular acidosis, mineralocorticoid deficiency
- Bicarbonate loss: diarrhoea, pancreatic fistula, diversion of urine to gut.
- Artefact: very low sample volume in venous vacuum tube collection.

HIGH SERUM BICARBONATE (METABOLIC ALKALOSIS)

Elevated bicarbonate concentrations in the serum may be due to retention in the kidney in response to a respiratory alkalosis; ingestion of bicarbonate-containing fluids; or loss of hydrogen ions from the body, such as in vomiting (especially with pyloric stenosis), gastric suction, or renal losses such as with some diuretics.

- Compensation for respiratory acidosis (renal bicarbonate retention)
- Bicarbonate administration, eg antacids
- Gastro-intestinal acid loss: vomiting (especially with pyloric stenosis); gastric suction, chloride diarrhoea

- Renal acid loss: diuretic therapy; mineralocorticoid excess, severe potassium deficiency

MEASUREMENT

Serum bicarbonate may be measured in routine venous serum or heparin plasma collections, or in samples collected for a blood gas analyser. The results from a main laboratory analyser or a blood gas machine give the same information with very little difference between arterial and venous bicarbonate concentrations in an individual. Of note falsely low results may be obtained in venous samples collected with very small amounts on blood in a vacuum tube.

REFERNCE INTERVALS

ADULT:

22-32 mmol/L (from 18 years of age and above)

PAEDIATRIC:

0 to <1 week	15-28mmol/L
1 week - 2 years	16-29 mmol/L
2 years - 10 years	17-30 mmol/L
10 years - 18 years	20-32 mmol/L

These intervals are those recommended for use in Australia by the RCPA and AACB.