

use of bicarbonate
[created by Paul Young 15/12/07]

important points about bicarbonate

1. Ventilation must be adequate to eliminate the CO₂ produced from bicarbonate
 - Bicarbonate decreases H⁺ by reacting with it to produce CO₂ and water.
 - For this reaction to continue the product (CO₂) must be removed.
 - So bicarbonate therapy can increase extracellular pH only if ventilation is adequate to remove the CO₂. Indeed if hypercapnia occurs then as CO₂ crosses cell membranes easily, intracellular pH may decrease even further with further deterioration of cellular function.
2. Bicarbonate may cause clinical deterioration if tissue hypoxia is present
 - If tissue hypoxia is present, then the use of bicarbonate may be particularly disadvantageous due to increased lactate production (removal of acidotic inhibition of glycolysis) and the impairment of tissue oxygen unloading (left shift of ODC due to increased pH).
 - This means that with lactic acidosis or cardiac arrest then bicarbonate therapy may be dangerous.
3. Bicarbonate is probably not useful in most cases of high anion gap acidosis
 - Lactic acidosis can get worse if bicarbonate is given.
 - Clinical studies have shown no benefit from bicarbonate in diabetic ketoacidosis.
 - In these cases, the only indication for bicarbonate use is probably for the emergency management of severe hyperkalaemia.
4. The preferred management of metabolic acidosis is to correct the primary cause and to use specific treatment for any potentially dangerous complications
 - The organic acid anions serve as bicarbonate precursors to regenerate new bicarbonate once the primary cause is treated. In some forms of acidosis specific treatment to prevent problems is possible (eg ethanol blocking therapy in ethylene glycol poisoning.)
 - If hyperkalaemia is present then [K⁺] can be decreased by bicarbonate therapy. Also, bicarbonate therapy can cause an alkaline diuresis which hastens renal salicylate excretion and may aid in excretion of barbiturates.
5. Bicarbonate therapy may be useful for correction of acidaemia due to non-organic (or mineral) acidosis (ie normal anion gap acidosis)
 - In non-organic acidosis, there is no organic anion which can be metabolised to regenerate bicarbonate. Once the primary cause is corrected, resolution of the acidaemia occurs more rapidly if bicarbonate therapy is used.
 - Amounts sufficient for only partial correction of the disorder should be given. The aim is to increase arterial pH to above 7.2 to minimise adverse effects of the acidaemia and to avoid the adverse effects of bicarbonate therapy.
 - If the patient is improving without serious clinical problems then waiting (for renal bicarbonate regeneration) and watching (for clinical improvement) is a better strategy than giving bicarbonate.

general

- Metabolic acidosis causes adverse metabolic effects.
- In particular the adverse effects on the cardiovascular system may cause serious clinical problems.
- Bicarbonate is an anion and cannot be given alone. Its therapeutic use is as a solution of sodium bicarbonate.
- An 8.4% solution is a molar solution (ie it contains 1mmol of HCO₃⁻ per ml) and is the concentration clinically available in Australia. This solution is very hypertonic (osmolality is 2,000 mOsm/kg).

indications

- The main goal of alkali therapy is to counteract the extracellular acidaemia with the aim of reversing or avoiding the adverse clinical effects of the acidosis (esp the adverse cardiovascular effects).
- Other reasons for use of bicarbonate in some cases of acidosis are:
 - (i) emergency management of hyperkalaemia
 - (ii) to promote alkaline diuresis (eg to hasten salicylate excretion)

adverse effects

- In general, the severity of these effects are related to the amount of bicarbonate used.
- These undesirable effects include:
 - (i) hypernatraemia
 - (ii) hyperosmolality
 - (iii) volume overload
 - (iv) rebound or 'overshoot' alkalosis
 - (v) hypokalaemia
 - (vi) impaired oxygen unloading due to left shift of the oxyhaemoglobin dissociation curve
 - (vii) acceleration of lactate production by removal of acidotic inhibition of glycolysis
 - (viii) CSF acidosis
 - (ix) hypercapnia