Bicarbonate loss generally occurs through the kidneys from increased acid production, as in lactic acidosis or ketoacidosis, can exceed maximal renal excretion and cause a rapidly developing, severe acidosis. Metabolic acidosis in the face of normal renal function increases NH4+ production and excretion. NH4+ production and excretion are impaired in chronic renal failure, hyperkalaemia, and renal tubular acidosis.

The management of serious acid-base disorders always demands precise diagnosis and treatment of the underlying disease, and in certain circumstances, it requires steps to combat the death in systemic acidity itself. Administration of sodium bicarbonate will increase the plasma HCO3− concentration and help restore the plasma pH within the normal range (pH 7.35-7.45). Changes in acid-base balance also stimulate compensatory ion-exchange mechanisms. When the extracellular hydrogen ion concentration increases, as in acidosis, there is a redistribution of potassium ions from intracellular to extracellular fluid. Administration of sodium bicarbonate can cause a redistribution of potassium ions into cells in patients with acidosis, by increasing the plasma pH.

The urinary pH will be increased by sodium bicarbonate in patients with normal renal function. Alkalising the urine can increase the solubility of certain weak acids, and can increase the ionisation and urinary excretion of lipid-soluble organic acids (e.g. phenobarbitone, salicylates).

Metabolic Acidosis

Metabolic acidosis is characterized by a primary decrease in serum bicarbonate (HCO3−) concentration to below 24 mmol/L and a compensatory decrease in the CO2 concentration. It occurs from either loss of bicarbonate (HCO3−) or addition of hydrogen ion (H+). Bicarbonate loss generally occurs through the kidneys or the bowel. Acidosis from decreased acid renal excretion generally is slow to develop. In contrast, acidosis leading to hypokalaemia.

PRECAUTIONS

Treatment strategies for metabolic acidosis are primarily directed towards the underlying cause. Bicarbonate therapy is a temporary measure used for severe acidosis. Specialised texts and protocols should be consulted to guide use. Note that sodium bicarbonate 8.4% is a hyperosmotic solution. Whenever respiratory acidosis is present with metabolic acidosis, both pulmonary ventilation and perfusion must be adequately supported to get rid of excess carbon dioxide.

Solutions containing sodium may cause fluid overload when given in excess, resulting in dilution of serum electrolytes, overhydration, congestive conditions or pulmonary oedema. Excessively elevated plasma sodium concentrations may cause dehydration of the brain, resulting in somnolence and confusion, which may progress to convulsions, coma, respiratory failure and ultimately death. Bicarbonate should be given with caution to patients with Type A lactic acidosis (tissue hypoxia). Administration of bicarbonate will tend to limit the available oxygen, increase lactate production and thus worsen the acidosis. Data from the literature are not in favour of the use of bicarbonate in the treatment of diabetic ketoacidosis with pH values between 6.90 and 7.10.

Sodium bicarbonate should be used with caution in patients with cirrhosis. Accidental extravascular injection of hypertonic solutions may cause vascular irritation, chemical cellulitis (because of their alkalinity), subsequently resulting in tissue necrosis, ulceration and/or sloughing at the site of injection. The use of scalp veins should be avoided. Do not use the injection if it contains precipitate. Do not use unless the solution is clear and the container and seal are intact. Discard any unused portion.

Use in Patients with Congestive Heart Failure or Renal Insufficiency

Sodium retention and oedema may occur during sodium bicarbonate therapy, especially when the drug is given in large doses or to patients with renal insufficiency, congestive heart failure or those predisposed to sodium retention and oedema. Sodium and water overload may result in hypervolaemia and hyperviscosity. Severe hyperosmolar states may develop during cardiopulmonary resuscitation when excessive doses of sodium bicarbonate are administered. Serum potassium may decrease during sodium bicarbonate therapy leading to hypokalaemia.

Sodium bicarbonate should be used with extreme caution in patients with congestive heart failure or other cardiogenic oedema or sodium retaining conditions in patients with renal insufficiency, especially those with severe insufficiency such as oliguria or anuria, and in patients receiving corticosteroids or corticotropin, since each gram of sodium bicarbonate contains 12 mEq of sodium.

Use in Children

Rapid injection (10 mL/min) of hypertonic Sodium Bicarbonate Injection solutions into neonates and children under 2 years of age may produce hypernatraemia, a decrease in cerebral fluid pressure and possible intracranial haemorrhage. In emergency situations, such as cardiac arrest, the risk of rapid infusion of the drug must be weighed against the potential for death from acidosis. It should also be noted that administration of sodium bicarbonate to children undergoing cardiopulmonary resuscitation may worsen respiratory acidosis. Do not administer more than 8 mmol/kg/day (See DOSAGE AND ADMINISTRATION).

Use in Pregnancy and Lactation

Animal reproduction studies have not been conducted with sodium bicarbonate. Safety in pregnancy and lactation has not been established.

The use of Sodium Bicarbonate Injection, as with any drug, in pregnant or lactating women should only be undertaken if the expected benefit outweighs the possible risk to the mother and fetus or child.

INTERACTIONS WITH OTHER MEDICINES

Alkalisation of the urine leads to increased renal clearance of acidic drugs such as salicylates, tetracyclines, (especially doxycycline), barbiturates and tricyclic antidepressants. Conversely, it prolongs the half life and duration of basic drugs such as quinidine, amphetamines, ephedrine and pseudoephedrine and may result in toxicity. Sodium bicarbonate enhances lithium excretion.

Solutions containing sodium ions should be used with great care, if at all, in patients receiving corticosteroids or corticotropin.

Hypochloeraemic acidosis may occur if sodium bicarbonate is used in conjunction with potassium depleting diuretics such as bumetanide, ethacrynic acid, frusemide and thiazides. Concurrent use in patients taking potassium supplements may reduce serum potassium concentration by promoting an intracellular ion shift.

The following drug may have enhanced or prolonged effects due to concomitant administration with sodium bicarbonate: flecainide.

The following drugs may have decreased effectiveness due to concomitant administration with sodium bicarbonate: aspirin and other salicylates, barbiturates and lithium.

The following drugs have been reported to be susceptible to inactivation on mixing with sodium bicarbonate solution: adrenaline HCl, benzylpenicillin potassium, carbamazepine, glycopyrrolate, isoprenaline HCl and suxamethonium chloride.
Sodium Bicarbonate 8.4% Injection can be diluted with 5% glucose injection or 0.9% sodium chloride injection. To reduce microbiological hazard, use as soon as practicable after dilution. If storage is necessary, hold at 2°C-8°C for not more than 24 hours. (See DOSAGE AND ADMINISTRATION)

Sodium bicarbonate should only be given if the child is being effectively ventilated as any carbon dioxide that exists.

Adequate alveolar ventilation should be ensured during cardiac arrest and administration of sodium bicarbonate, since adequate ventilation contributes to the correction of acidosis and since administration of sodium bicarbonate is followed by release of carbon dioxide.

Children – The usual dose is 1 mmol/kg (1 mL/kg of an 8.4% sodium bicarbonate injection) given by slow intravenous injection.

Infants (up to 2 years of age) - In infants (up to 2 years of age) the solution should be diluted with an equal amount (1:1 ratio) of 5% glucose or water for injections (to make 4.2% sodium bicarbonate solution) for slow intravenous administration and at a dose not to exceed 8 mmol/kg/day, and according to the appropriate treatment protocol and guidelines. This diluted solution is hypertonic. Slow administration rates and a 4.2% solution are recommended in order to minimise the possibility of producing hypernatraemia, decreasing cerebrospinal fluid pressure and inducing intracranial haemorrhage. (See PRECAUTIONS and ADVERSE EFFECTS)

Sodium bicarbonate should only be given if the child is being effectively ventilated as any carbon dioxide that is released by the process of acid neutralisation must be removed from the body via the lungs or paradoxical intracellular acidosis will result.

Intravenous Infusion - In less urgent forms of metabolic acidosis, Sodium Bicarbonate Injection may be added to 5% glucose for intravenous infusion. (See COMPATIBILITY / INCOMPATIBILITY)