**Name of drug**
Sodium Bicarbonate

**Description**
A sterile, hypertonic, preservative-free solution containing Sodium Bicarbonate 84 mg/mL and Disodium Edetate in Water for Injections. Each mL of solution contains 1 mmol each of sodium and bicarbonate ions.

Molecular Formula: NaHCO₃
CAS Number: 144-55-8

**Pharmacology**

**Mode of action:** Sodium bicarbonate is a systemic alkalising agent which, when given intravenously will increase plasma bicarbonate, buffers excess hydrogen ion concentration, raises blood pH and reverses the clinical manifestations of acidosis.

**Pharmacokinetics:** Sodium bicarbonate dissociates in water to provide sodium (Na⁺) and bicarbonate (HCO₃⁻) ions. Sodium is the principal cation of the extracellular fluid. Bicarbonate is a normal constituent of body fluids and the normal plasma level ranges from 24 to 31 mmol/L. Plasma concentration is regulated by the kidney. The bicarbonate anion, at the correct concentration of hydrogen ion (H⁺) may be converted to carbonic acid (H₂CO₃), then to its volatile form, carbon dioxide (CO₂) which is excreted by the lung. Normally, a ratio of 1:20 (carbonic acid: bicarbonate) is present in the extracellular fluid. In a healthy adult with normal kidney function, practically all the glomerular filtered bicarbonate ion is reabsorbed and less than 1 % is excreted in the urine.

**Indications**
- **Metabolic acidosis** in severe renal disease, uncontrolled diabetes, circulatory insufficiency due to shock or severe dehydration, extracorporeal circulation of blood, cardiac arrest and severe primary lactic acidosis where a rapid increase in plasma total CO₂ content is crucial. Treatment of metabolic acidosis should be concurrent with measures designed to control the cause of the acidosis.
- **Urinary alkalinisation** in the treatment of certain drug intoxications (ie barbiturates, salicylates, lithium, methyl alcohol) and in the haemolytic reactions requiring alkalinisation of the urine to diminish nephrotoxicity of blood pigments. Urinary alkalinisation is also used in methotrexate therapy to prevent nephrotoxicity.
- **Severe diarrhoea** which is often accompanied by a significant loss of bicarbonate.

**Contraindications**
- renal failure
- metabolic alkalosis
- respiratory alkalosis
- hypertension
- oedema
- congestive heart failure
- a history of urinary calculi and coexistent potassium depletion or hypocalcaemia
- hypernatraemia, hypoventilation or chloride depletion
- in patients at risk of developing diuretic induced hypochloraemic alkalosis
Precautions

- Rapid injections (10 mL/min) of hypertonic sodium bicarbonate solutions to neonates and children under 2 years of age may produce hypernatraemia, a decrease in cerebrospinal fluid pressure and possible intracranial haemorrhage. Do not administer more than 8 mmol/kg/day.
- 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. Also, administration of this drug to children undergoing cardiopulmonary resuscitation may worsen respiratory acidosis.
- To minimise the risks of pre-existing hypokalaemia and/or hypocalcaemia, these electrolyte disturbances should be corrected prior to initiation of, or concomitantly with, sodium bicarbonate therapy.
- Arterial blood gas analysis should be performed during the course of sodium bicarbonate treatment to minimise the possibility of overdosage.
- Accidental extravasation of hypertonic solutions may cause cellulitis, tissue necrosis, vascular irritation and sloughing. The use of scalp veins should be avoided. Prompt elevation of the affected area, warmth and local injection of lignocaine or hyaluronidase are recommended to prevent sloughing.
- Whenever respiratory acidosis is present with metabolic acidosis both pulmonary ventilation and perfusion must be adequately supported in order to eliminate excess CO₂.
- Solutions containing sodium may cause fluid overload when given in excess. They should be used with caution in patients with congestive heart failure, renal impairment, cirrhosis, eclampsia, hypertension, aldosteronism or any oedematous state.
- 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.

Use in pregnancy: Animal reproduction studies have not been performed with sodium bicarbonate. It is also not known whether sodium bicarbonate can cause fetal harm when administered to pregnant women. Sodium bicarbonate should be used during pregnancy only when clearly needed and the benefits of therapy outweigh the potential risks.

Use in lactation: It is not known whether sodium bicarbonate is excreted in breast milk. However, problems in humans have not been reported.

Effect on laboratory tests: The high urinary alkalinity sometimes produced by sodium bicarbonate may cause a false positive Labistix test for urinary protein.

Interactions with other drugs:

- Urinary alkaliisation will increase the renal clearance of tetracyclines, especially doxycycline, but it will increase the half life and duration of action of basic drugs such as quinidine, amphetamines, ephedrine and pseudoephedrine.
- The addition of sodium bicarbonate to solutions containing calcium should be avoided except where compatibility has been shown. Solutions turning hazy as a result of sodium bicarbonate-calcium admixtures should be discarded.
- Use caution when giving parenteral fluids, especially those containing sodium ions, to patients receiving corticosteroids or corticotrophin.

Incompatibilities: Sodium bicarbonate is incompatible with certain substances in solution and specialised literature should be consulted.

- The following drugs have been reported as susceptible to inactivation when mixed with sodium bicarbonate solution: adrenalin hydrochloride, benzylpenicillin, carmustine, glycopyrronium bromide, isoprenaline hydrochloride, potassium and suxamethonium chloride.
- The following incompatibilities have been reported: acids, acid salts and many alkaloïdal salts, calcium and magnesium salts, cisplatin, corticotrophin, hydromorphone hydrochloride, dobutamine hydrochloride, insulin, labelatal hydrochloride, magnesium sulfate, mexitilin sodium, narcotic salts, noradrenaline acid tartrate, pentobarbitone sodium, procaine hydrochloride, promazine hydrochloride (in
dextrose injection), streptomycin sulfate, tetracycline hydrochloride, thiopentone sodium, vancomycin hydrochloride, lactated Ringer's injection, sodium lactate injection or Ringer's injection.

Adverse reactions
- Alkalosis and/or hypokalaemia may result from prolonged use
- Extravasation of intravenous hypertonic solutions of sodium bicarbonate may cause cellulitis, with tissue necrosis or sloughing at the site of infiltration
- Hyperirritability or tetany may occur

Dosage and administration
Sodium Bicarbonate Intravenous Infusion is administered intravenously preferably into a large vein.

In cardiac arrest: Administration is determined based on the results of arterial blood pH, PaCO₂ and calculation of base deficit. For adults, an initial dose of 1mmol/kg followed by 0.5 mmol/kg every 10 minutes of arrest, depending on arterial blood gases. In cardiac arrest, the risks from acidosis exceed those of hypernatraemia.

Children: The usual dose is 1 mmol/kg (1 mL/kg) given by slow intravenous injection.

For infants up to 2 years of age: A 4.2% solution is recommended at a rate not exceeding 8mmol/kg/day. This will minimise the risk of the possibility of hypernatremia, decreasing cerebrospinal fluid pressure and intracranial haemorrhage. Diluents may be sterile, physiological solution, glucose 5%, or other standard electrolyte solutions but each should be tested for compatibility.

In mild conditions of metabolic acidosis: Sodium Bicarbonate Intravenous Infusion may be admixed with other intravenous fluids if compatibility is proven.

The amount of bicarbonate to be given to older children and adults over a 4 to 8 hour period is approximately 2 to 5 mmol/kg, depending upon the severity of the acidosis as judged by the lowering of total CO₂ content, blood pH and clinical condition of the patient. Initially, an infusion of 2 to 5mmol/kg over 4 to 8 hours will produce improvement in the acid-base status of the blood.

Therapy should be planned in a step by step method as the degree of response from a given dose is not precisely predictable.

In general it is unwise to try and fully correct a low total CO₂ content during the first 24 hours of therapy. This may be accompanied by unrecognised alkalosis due to a delay in the readjustment of normal ventilation.

Overdosage
Alkalosis is a direct result of overdosage. Administration of sodium bicarbonate should be immediately discontinued. In order to control the symptoms of alkalosis, the patient should rebreathe expired air, and the patient treated with intravenous sodium chloride 0.9% and potassium chloride if hypokalaemia is present. Any accompanying hyperirritability or tetany can be controlled with calcium gluconate. Ammonium chloride may be indicated in severe cases (except in patients with pre-existing hepatic disease).

Treatment of hypernatremia usually requires water replacement. In some cases, restricted sodium intake and oral water may be sufficient. If more severe, glucose 5% may be administered by slow intravenous infusion. If total body sodium is too high, loop diuretics combined with an infusion of 5% glucose and potassium supplementation may be necessary.

Presentation
AUST R 10801 Sodium Bicarbonate Intravenous Infusion BP 8.4% 100 mL glass vial

Storage
Store below 30°C. Single use only. Discard any unused portion.
The expiry date (month/year) is stated on the package after EXP.
Poison schedule
Australia – Nil

Manufacturer
Pfizer (Perth) Pty Limited
ABN 32 051 824 956
15 Brodie Hall Drive,
Bentley WA 6102 Australia

Sponsor in Australia:
Pfizer Australia Pty Ltd
ABN 50 008 422 348
38-42 Wharf Road
West Ryde NSW 2114 Australia

This information was approved by the TGA on 10 July 2000.

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