Sodium Carbonate Anhydrous

Carbonato de sodio anhidro; Cenizas de Soda; E500; Exsiccated Sodium Carbonate; Natrii Carbonas; Natrii carbonas anhydricus; Natrio karbonatas, bevandenis; Natrium Carbonicum Calcinatum; Natrium Carbonicum Siccatum; Natriumkarbonaatti, vedetön; Natriumkarbonat, vattenfritt; Sodium (carbonate de) anhydre; Uhličitan sodný; Uhličitan sodný bezvodý; Vízmentes nátrium-karbonát.

Na_2CO_3 = 106.0.

CAS — 497-19-8.

NOTE. Soda ash is a synonym for the technical grade of sodium carbonate anhydrous.

Pharmacopoeias. In *Eur.* (see p.vii) and *Jpn. USNF* allows the anhydrous substance or the monohydrate.

Ph. Eur. 6.2 (Sodium Carbonate, Anhydrous). A white or almost white, slightly granular, hygroscopic powder. Freely soluble in water; practically insoluble in alcohol. A 10% solution in water is strongly alkaline. Store in airtight containers.

USNF 26 (Sodium Carbonate). Colourless crystals, or white, crystalline powder or granules. Soluble 1 in 3 of water and 1 in 1.8 of boiling water.

Sodium Carbonate Decahydrate

Carbonato de sodio decahidratado; Cristales de Sosa; E500; Natrii Carbonas; Natrii carbonas decahydricus; Natrio karbonatas dekahidratas; Natrium Carbonicum Crystallisatum; Natriumkarbonaattidekahydraatti; Nátrium-karbonát-dekahidrát; Natriumkarbonatdekahydrat; Sodium (carbonate de) décahydraté; Uhličitan sodný dekahydrát.

$Na_2CO_3,10H_2O$ = 286.1.

CAS — 6132-02-1.

NOTE. Washing soda is a synonym for the technical grade of sodium carbonate decahydrate.

Pharmacopoeias. In *Eur.* (see p.vii) and *Jpn.*

Ph. Eur. 6.2 (Sodium Carbonate Decahydrate). Colourless, efflorescent, transparent crystals or a white or almost white crystalline powder. Freely soluble in water; practically insoluble in alcohol. A 10% solution in water is strongly alkaline. Store in airtight containers.

Sodium Carbonate Monohydrate

Carbonato de sodio monohidratado; E500; Natrii carbonas monohydricus; Natrio karbonatas monohidratas; Natriumkarbonaattimonohydraatti; Nátrium-karbonát-monohidrát; Natriumkarbonatmonohydrat; Sodium (carbonate de) monohydraté; Sodu węglan jednowodny; Uhličitan sodný monohydrát.

Na_2CO_3,H_2O = 124.0.

CAS — 5968-11-6.

Pharmacopoeias. In *Eur.* (see p.vii). *USNF* allows the anhydrous substance or the monohydrate.

Ph. Eur. 6.2 (Sodium Carbonate Monohydrate). A white or almost white, crystalline powder or colourless crystals. Freely soluble in water; practically insoluble in alcohol. A 10% solution in water is strongly alkaline. Store in airtight containers.

USNF 26 (Sodium Carbonate). Colourless crystals, or white, crystalline powder or granules. When exposed to dry air above 50°, it effloresces and at 100° it becomes anhydrous. Soluble 1 in 3 of water and 1 in 1.8 of boiling water.

Profile

Sodium carbonate is used in antacid preparations. Anhydrous sodium carbonate and the monohydrate are also used as reagents. The decahydrate has been used in alkaline baths. Sodium carbonate in its anhydrous or hydrated form is also used as a water softener.

Sodium carbonate may be irritating or mildly corrosive to skin, mucous membranes, and eyes.

Preparations

BPC 1973: Surgical Chlorinated Soda Solution;
USP 31: Citric Acid, Magnesium Oxide, and Sodium Carbonate Irrigation.

Proprietary Preparations (details are given in Part 3)

Multi-ingredient: ***Arg.:*** Alikal; Otocalmia Biotic; Otocerol; Otoclean Gotas Oticas; Sal de Fruta Eno; Sincerum; Uvasal; Yasta; ***Austral.:*** Eno; ***Braz.:*** Digestbem; Sal de Fruta Eno; Sonrisal; ***Fr.:*** Bactident; Hydralin; ***Hong Kong:*** Eno†; Hydralin; ***Irl.:*** Cymalon; ***Israel:*** Eno; Urikal; ***Ital.:*** Gastrotuss; ***Port.:*** Eno; Gastropensan; ***Spain:*** Sal de Fruta Eno; Tanasid†; ***Switz.:*** Saltrates Rodell†; ***UK:*** Cymalon; Eno; Resolve; ***Venez.:*** Eno.

Sodium Chlorate

Clorato de potasio; Natrium Chloricum; Sodii Chloras.

$NaClO_3$ = 106.4.

CAS — 7775-09-9.

Profile

Sodium chlorate closely resembles potassium chlorate (p.2371) in its properties and has been used as an astringent. Its main use is as a weedkiller and it is therefore a common household chemical. Poor storage conditions can lead to explosions.

Preparations

Proprietary Preparations (details are given in Part 3)

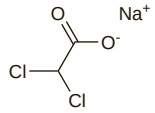
Multi-ingredient: ***Spain:*** Co Bucal.

Sodium Dichloroacetate *(USAN)*

CPC-211; DCA; Dicloroacetato de sodio.

$C_2HCl_2NaO_2$ = 150.9.

CAS — 2156-56-1 (sodium dichloroacetate); 79-43-6 (dichloroacetic acid).



Profile

Dichloroacetic acid activates pyruvate dehydrogenase, a mitochondrial enzyme that catalyses metabolism of pyruvate and lactate, and it inhibits glycolysis. It also stimulates myocardial contractility. Sodium dichloroacetate has been used for the treatment of congenital lactic acidosis, lactic acidosis in patients with severe malaria, homozygous familial hypercholesterolaemia, and for severe brain injury. It is also under investigation for stroke.

Adverse effects. Adverse effects reported with sodium dichloroacetate have mainly involved the central and peripheral nervous systems.[1] Anxiolytic or sedative effects are common. Reversible polyneuropathy has been reported after chronic use, as has asymptomatic elevation of serum transaminases. Reduced urate clearance and elevated serum urate levels have been reported in patients with type 2 diabetes mellitus. See also under Use in Metabolic Acidosis, below for reference to early termination of a study due to development of peripheral neuropathy.

1. Stacpoole PW, *et al.* Pharmacokinetics, metabolism, and toxicology of dichloroacetate. *Drug Metab Rev* 1998; **30:** 499–539.

Pharmacokinetics. References.

1. Henderson GN, *et al.* Pharmacokinetics of dichloroacetate in adult patients with lactic acidosis. *J Clin Pharmacol* 1997; **37:** 416–25.
2. Shangraw RE, Fisher DM. Pharmacokinetics and pharmacodynamics of dichloroacetate in patients with cirrhosis. *Clin Pharmacol Ther* 1999 **66:** 380–90.

Use in metabolic acidosis. In a study[1] in 29 patients with lactic acidosis (p.1667), sodium dichloroacetate 50 mg/kg given by intravenous infusion over 30 minutes, followed by a second dose 2 hours after beginning the first infusion, produced a metabolic response in 23 patients with a short-term increase in survival. However, a subsequent study[2] found that, while dichloroacetate infusion did reduce blood-lactate concentrations, it did not alter haemodynamics or survival in patients with severe lactic acidosis. A review[3] of these and other controlled studies in the treatment of acquired and congenital lactic acidosis concluded that the maximum lactate-lowering effect is dose-dependent but independent of time after dosing. Whether lowering lactate levels contributes to reducing morbidity and mortality in hyperlactataemia remains controversial, although data from recent studies suggest that treatment in mild cases may reduce the risk of death. A review[4] of the treatment of children with dichloroacetate for congenital lactic acidosis hypothesised that it might improve quality of life by reducing the frequency of acid–base decompensations, improving neurological function, and stimulating linear growth. A randomised controlled study[5] of dichloroacetate for the treatment of congenital lactic acidosis in 43 patients ranging in age from 3 months to 18 years found that dichloroacetate for 6 months was well tolerated and reduced blood-lactate response to a carbohydrate challenge but had no effect on basal-lactate concentrations nor did it improve neurologic or other measures of clinical outcome. In another randomised controlled study[6] of the effects of dichloroacetate in the treatment of the multisystem syndrome of mitochondrial myopathy, encephalopathy, lactic acidosis, and stroke-like episodes (MELAS), 13 of 15 patients given the study medication developed peripheral neuropathy, displaying either clinical signs and symptoms or electrophysiological evidence. The study was therefore stopped early, and on this basis, the authors concluded that dichloroacetate could not be recommended for the treatment of MELAS.

In a randomised, double-blind, placebo-controlled study[7] in 124 West African children with severe *Plasmodium falciparum* malaria, a single intravenous infusion of sodium dichloroacetate in a dose of 50 mg/kg given at the same time as quinine increased the rate and magnitude of fall in blood-lactate levels without compromising the plasma kinetics of quinine.

In the UK, the *BNFC* includes the following doses for neonates and children with pyruvate dehydrogenase defects: 12.5 mg/kg 4 times daily by mouth, adjusted according to response up to 200 mg/kg daily.

Sodium dichloroacetate has also been studied[8] in patients with traumatic brain injury for its lactate-lowering effect in cerebrospinal fluid.

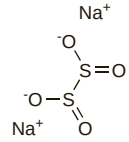
1. Stacpoole PW, *et al.* Dichloroacetate in the treatment of lactic acidosis. *Ann Intern Med* 1988; **108**: 58–63.
2. Stacpoole PW, *et al.* A controlled clinical trial of dichloroacetate for treatment of lactic acidosis in adults. *N Engl J Med* 1992; **327:** 1564–9.
3. Stacpoole PW, *et al.* Efficacy of dichloroacetate as a lactate-lowering drug. *J Clin Pharmacol* 2003; **43:** 683–91.
4. Stacpoole PW, *et al.* Treatment of congenital lactic acidosis with dichloroacetate. *Arch Dis Child* 1997; **77:** 535–41.
5. Stacpoole PW, *et al.* Controlled clinical trial of dichloroacetate for treatment of congenital lactic acidosis in children. *Pediatrics* 2006; **117:** 1519–31.
6. Kaufmann P, *et al.* Dichloroacetate causes toxic neuropathy in MELAS: a randomized, controlled clinical trial. *Neurology* 2006; **66:** 324–30.
7. Agbenyega T, *et al.* Population kinetics, efficacy, and safety of dichloroacetate for lactic acidosis due to severe malaria in children. *J Clin Pharmacol* 2003; **43:** 386–96.
8. Williams PJ, Dichloroacetate: population pharmacokinetics with a pharmacodynamic sequential link model. *J Clin Pharmacol* 2001; **41:** 259–67.

Sodium Dithionite

Ditionito de sodio; Natrii Dithionis; Sodium Hydrosulfite; Sodium Hydrosulphite; Sodium Sulphoxylate; Sodu ditionian; Sodu podsiarczyn.

$Na_2S_2O_4$ = 174.1.

CAS — 7775-14-6.



NOTE. The name sodium hydrosulfite is also applied to $NaHSO_2$ = 88.06.

Pharmacopoeias. In *Pol.*

Profile

Sodium dithionite is used as a reducing agent. It may be used in the form of a simple urine test in the detection of paraquat poisoning. A 0.25% solution has been used to remove phenazopyridine stains from fabric. It is irritant to the skin.

Sodium Gluconate

E576; Gluconato de sodio. Monosodium D-gluconate.

$C_6H_{11}NaO_7$ = 218.1.

CAS — 527-07-1.

Pharmacopoeias. In *US.*

Profile

Sodium gluconate is a food additive.

Gluconates act as acceptors of hydrogen ions produced by metabolic processes and are an indirect source of bicarbonate ions.

Sodium Humate

CAS — 1415-93-6 (humic acids); 68131-04-4 (sodium humates).

Profile

Humic acids are mixtures of complex macromolecules derived from the decomposition of organic material and are found in soils and peats. They have been used topically, usually as sodium humate, for musculoskeletal and joint disorders. They also have industrial applications.

Preparations

Proprietary Preparations (details are given in Part 3)

Ger.: Leukona-Sulfomoor-Bad F†; Rheumasan Moor-Bad S†.

Multi-ingredient: ***Austria:*** Humal; Leukona-Sulfomoor-Bad†; Salhumin; ***Ger.:*** Salhumin Rheuma-Bad; Salhumin Sitzbad N†; Salhumin Teilbad N†.

Sodium Hydroxide

Ätznatron; Caustic Soda; E524; Hidróxido de sodio; Hydroxid sodný; Natrii hydroxidum; Natrio hidroksidas; Natrium Hydricum; Natrium Hydroxydatum; Nátrium-hidroxid; Natriumhydroksidi; Natriumhydroxid; Soda Lye; Sodium, hydroxyde de; Sodu wodorotlenek; Sosa cáustica.

NaOH = 40.00.

CAS — 1310-73-2.

Pharmacopoeias. In *Chin.*, *Eur.* (see p.vii), *Int.*, and *Jpn.* Also in *USNF.*

Ph. Eur. 6.2 (Sodium Hydroxide). White or almost white, crystalline masses supplied as pellets, sticks, or slabs. It is deliquescent and readily absorbs carbon dioxide. Very soluble in water; freely soluble in alcohol. A 0.01% solution in water has a pH of not less than 11.0. Store in airtight, nonmetallic containers.

USNF 26 (Sodium Hydroxide). White or practically white fused masses, small pellets, flakes, sticks, or other forms. It is hard and

brittle and shows a crystalline fracture. When exposed to air it rapidly absorbs moisture and carbon dioxide. Soluble 1 in 1 of water; freely soluble in alcohol. Store in airtight containers.

Adverse Effects

Sodium hydroxide is strongly alkaline and corrosive, and rapidly destroys organic tissues.

The ingestion of caustic alkalis causes immediate burning pain in the mouth, throat, substernal region, and epigastrium, and the lining membranes become swollen and detached. There is dysphagia, hypersalivation, vomiting with the vomitus becoming blood-stained, diarrhoea, and shock. In severe cases, abdominal pain, asphyxia due to oedema of the glottis, circulatory failure, oesophageal or gastric perforation, peritonitis, or pneumonia may occur. Stricture of the oesophagus can develop weeks or months later.

Caustic alkalis on contact with the skin can produce full thickness burns leading to extensive damage. Alkali burns to the eyes cause conjunctival oedema and corneal destruction; damage may be irreversible.

Treatment of Adverse Effects

Ingestion should not be treated by lavage or emesis. Dilution with water or milk is generally considered controversial for management of corrosive ingestion. However, early dilution therapy of alkalis may reduce oesophageal injury; large volumes of fluid should be avoided. Neutralisation of alkalis is contra-indicated. The airway should be maintained and shock and pain alleviated.

In cases of skin contamination, clothing should be removed immediately and the skin flooded with copious amounts of water for at least 15 minutes. Excision or skin grafting of burnt areas may be necessary in severe cases. Contaminated eyes should be irrigated thoroughly with water or 0.9% sodium chloride until the conjunctival sac pH is normal, which may require irrigation for up to an hour.

Uses and Administration

Sodium hydroxide is a powerful caustic. A 2.5% solution in glycerol has been used as a cuticle solvent. An escharotic preparation of sodium hydroxide and calcium oxide was known as London paste. Sodium hydroxide is also used for adjusting the pH of solutions.

Disinfection. For reference to the possible use of sodium hydroxide for the disinfection of material contaminated by the agent causing Creutzfeldt-Jakob disease, see p.1622.

Preparations

Proprietary Preparations (details are given in Part 3)

Multi-ingredient: ***Austria:*** Leberinfusion; Sulfo-Schwefelbad; ***Ger.:*** Glutarsin E†; ***Switz.:*** Saltrates†.

Sodium Iodoheparinate

Iodoheparinate Sodium; Iodoheparinato de sodio.

ATC — S01XA09.

ATC Vet — QS01XA09.

Profile

Sodium iodoheparinate is a derivative of heparin (p.1301) that has been used topically for the treatment of corneal burns and ulceration.

Sodium Methylarsinate

Metilarsinato de sodio; Natrium Methylarsonicum; Sodium Metharsinite. Disodium monomethylarsonate hexahydrate.

$CH_3AsNa_2O_3,6H_2O$ = 292.0.

CAS — 5967-62-4.

Profile

Sodium methylarsinate is an organic arsenic compound with adverse effects similar to those of arsenic trioxide (p.2260). It was formerly included in some vitamin and mineral preparations. It has also been used as a herbicide.

Sodium Morrhuate *(rINN)*

Morrhuate de Sodium; Morrhuate Sodium; Morruato de sodio; Natrii Morrhuas; Natriummorruatti; Natriummorruat.

Натрия Морруат

CAS — 8031-09-2.

Pharmacopoeias. *Chin.* and *US* include the injection.

Profile

Sodium morrhuate consists of the sodium salts of the fatty acids of cod-liver oil. It is a sclerosant that has been used in the treatment of varicose veins (p.2347). Usual doses are 50 to 100 mg for small or medium veins or 150 to 250 mg for large veins given as a 5% solution by intravenous injection.

Preparations

USP 31: Morrhuate Sodium Injection.

Proprietary Preparations (details are given in Part 3)

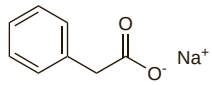
USA: Scleromate.

Sodium Phenylacetate *(USAN)*

Fenilacetato de sodio.

$C_8H_7NaO_2$ = 158.1.

CAS — 114-70-5.



Profile

Sodium phenylacetate is used as adjunctive treatment for acute hyperammonaemia and associated encephalopathy in patients with enzymatic deficiencies in the urea cycle (p.1929). It is given with sodium benzoate (p.1630) as a combined preparation for intravenous infusion in which 1 mL contains 100 mg of each component. The preparation is diluted in sterile glucose injection 10% at ≥25 mL/kg before infusion. Other similar therapies (e.g. oral sodium phenylbutyrate, see below) should be stopped before starting the infusion. A loading dose is infused over 90 to 120 minutes followed by an equivalent maintenance dose infused over 24 hours. Doses of sodium phenylacetate (together with the same amount of sodium benzoate) are 250 mg/kg for patients weighing 20 kg or less, and 5.5 g/m^2 for those over 20 kg. Maintenance infusions are continued until plasma ammonia concentrations are normal or oral nutrition and therapy can be tolerated. Sodium phenylacetate has also been given by mouth.

◊ References.

1. The Urea Cycle Disorders Conference Group. Consensus statement from a conference for the management of patients with urea cycle disorders. *J Pediatr* 2001; **138** (suppl 1): S1–S5.
2. Summar M. Current strategies for the management of neonatal urea cycle disorders. *J Pediatr* 2001; **138** (suppl 1): S30–S39.
3. Batshaw ML, *et al.* Alternative pathway therapy for urea cycle disorders: twenty years later. *J Pediatr* 2001; **138** (suppl 1): S46–S55. Correction. *ibid.* 2002; **140:** 490.
4. MacArthur RB, *et al.* Pharmacokinetics of sodium phenylacetate and sodium benzoate following intravenous administration as both a bolus and continuous infusion to healthy adult volunteers. *Mol Genet Metab* 2004; **81** (suppl 1): S67–S73.
5. Enns GM, *et al.* Survival after treatment with phenylacetate and benzoate for urea-cycle disorders. *N Engl J Med* 2007; **356:** 2282–92.

Preparations

Proprietary Preparations (details are given in Part 3)

Multi-ingredient: ***USA:*** Ammonul; Ucephan.

Sodium Phenylbutyrate *(BAN, USAN)*

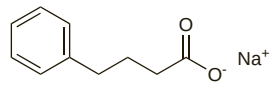
Fenilbutirato de sodio; Natrii phenylbutyras; Natriumfenylbutyrat; Natriumfenyylibutyraatti; Sodium, phénylbutyrate de; Sodyum Fenilbutirat. Sodium 4-Phenylbutyrate.

$C_{10}H_{11}NaO_2$ = 186.2.

CAS — 1716-12-7.

ATC — A16AX03.

ATC Vet — QA16AX03.



Pharmacopoeias. In *Eur.* (see p.vii).

Ph. Eur. 6.2 (Sodium Phenylbutyrate). A white or yellowish-white powder. Freely soluble in water and in methyl alcohol; practically insoluble in dichloromethane. A 2% solution in water has a pH of 6.5 to 7.5.

Profile

Sodium phenylbutyrate is a prodrug for sodium phenylacetate (see above) and is used as adjunctive treatment of hyperammonaemia in patients with urea cycle disorders (p.1929). It is given orally in equally divided doses with meals. The total daily dose for patients weighing under 20 kg is 450 to 600 mg/kg, and for those weighing over 20 kg, 9.9 to 13.0 g/m^2.

Sodium phenylbutyrate is also under investigation for the treatment of some sickle-cell disorders (p.1044) and for use as a potential differentiation-inducing agent in malignant glioma and acute myeloid leukaemia. Sodium phenylbutyrate is also under investigation for spinal muscular atrophy, which is caused by homozygous absence of the SMN1 gene, after reports that it significantly increased SMN transcript expression in both fibroblast cultures and leucocytes from these patients.

◊ References.

1. Batshaw ML, *et al.* Alternative pathway therapy for urea cycle disorders: twenty years later. *J Pediatr* 2001; **138** (suppl 1): S46–S55. Correction. *ibid.* 2002; **140:** 490.
2. Mercuri E, *et al.* Randomized, double-blind, placebo-controlled trial of phenylbutyrate in spinal muscular atrophy. *Neurology* 2007; **68:** 51–5.
3. Caruthers RL, Johnson CE. Stability of extemporaneously prepared sodium phenylbutyrate oral suspensions. *Am J Health-Syst Pharm* 2007; **64:** 1513–15.

Preparations

Proprietary Preparations (details are given in Part 3)

Cz.: Ammonaps; ***Fr.:*** Ammonaps†; ***Ger.:*** Ammonaps†; ***Ital.:*** Ammonaps; ***Neth.:*** Ammonaps; ***Pol.:*** Ammonaps; ***Port.:*** Ammonaps; ***Spain:*** Ammonaps; ***UK:*** Ammonaps; ***USA:*** Buphenyl.

Sodium Polymetaphosphate

E452 (sodium polyphosphates); Polimetafosfato de sodio.

CAS — 50813-16-6.

NOTE. Although Sodium hexametaphosphate has been used as a synonym for the polymetaphosphate, the latter also exists in much higher degrees of polymerisation.

Profile

Sodium polymetaphosphate has been used as a 5% dusting powder in hyperhidrosis and bromhidrosis, and as a prophylactic against athlete's foot.

Sodium polymetaphosphate combines with calcium and magnesium ions to form complex soluble compounds and is used as a water softener.

Sodium Pyrophosphate *(USAN)*

Sodu pirofosforan; Tetrasodium Pyrophosphate; TSPP.

$Na_4P_2O_7$ = 265.9.

CAS — 7722-88-5.

Profile

Sodium pyrophosphate acts as a calcium chelator and is used in products for dental care to reduce tartar formation. It is also used as a food additive, and as a water softener in detergents and for industrial applications. Potassium pyrophosphate (tetrapotassium pyrophosphate) and sodium acid pyrophosphate (disodium pyrophosphate) are used similarly.

Sodium pyrophosphate is also used in kits for the preparation of technetium-99m pyrophosphate.

Preparations

Proprietary Preparations (details are given in Part 3)

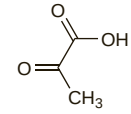
Multi-ingredient: ***Arg.:*** Esmedent Dientes Sens Blanq + Ctrol Sarro; Fluordent PX; Odol Control Sarro†; ***Braz.:*** Malvatricin Antiplaca; ***Chile:*** FKD; ***Ital.:*** AZ Tartar Control; ***USA:*** Plax.

Sodium Pyruvate

Piruvato de sodio. Sodium α-ketopropionate; sodium 2-oxopropanoate.

$C_3H_4NaO_3$ = 111.1.

CAS — 127-17-3 (pyruvic acid); 113-24-6 (sodium pyruvate).



(pyruvic acid)

Profile

Sodium pyruvate has been given intravenously in the diagnosis of disorders of pyruvate metabolism.

◊ Relative serum concentrations of lactate and pyruvate after a 10-minute intravenous infusion of sodium pyruvate 500 mg/kg have been used as an aid to the diagnosis of disorders of pyruvate metabolism.[1] Death shortly after pyruvate loading in a 9-year-old child with restrictive cardiomyopathy suggests that the test should not be performed when cardiac function is decreased.[2]

1. Dijkstra U, *et al.* Friedreich's ataxia: intravenous pyruvate load to demonstrate a defect in pyruvate metabolism. *Neurology* 1984; **34:** 1493–7.
2. Matthys D, *et al.* Fatal outcome of pyruvate loading test in child with restrictive cardiomyopathy. *Lancet* 1991; **338:** 1020–1.

Sodium Silicate

Silicato de sodio; Soluble Glass; Water Glass.

CAS — 1344-09-8.

Profile

Concentrated aqueous solutions of sodium silicate are commercially available and have many industrial uses. The solutions vary in composition, viscosity, and density; the greater the ratio of Na_2O to SiO_2 the more tacky and alkaline the solution.