Sodium bicarbonate | 2016

Alert	If sodium bicarbonate is used during prolonged resuscitation, it should be given only after adequate ventilation and circulation is established with CPR. Rapid infusion of sodium bicarbonate is associated with increased incidence of intraventricular haemorrhage in preterm infants. Conversion factor for sodium bicarbonate: 1 mmol = 1 mEq Avoid simultaneous administration of sodium bicarbonate and catecholamines through the same IV catheter or tubing as the sodium bicarbonate solution will inactive the catecholamine.
Indication	Metabolic acidosis Chronic renal failure Renal tubular acidosis Prolonged resuscitation
Action	Neutralises excess hydrogen ion and raises pH of the blood. Increases the excretion of free bicarbonate ions in urine, raising urinary pH.
Drug Type	Electrolyte, alkalinising agent
Trade Name	Sodium Bicarbonate Injection 8.4% w/v BP [Phebra]; Sodium Bicarbonate Infusion [Baxter]
Presentation	8.4% (1 mmol/mL) 10 mL injection
Dosage/Interval	Usual dose: 1–2 mmol/kg To calculate dosage required based on base deficit: Sodium bicarbonate dose (mEq) = 0.3 x weight (kg) x base deficit (mEq/L) (Administer half of the calculated dose, then assess need for remainder) Dilute to a maximum concentration of no greater than 0.5 mmol/mL (osmolarity = 1000 mOsm/L).
Route	IV PO
Preparation/Dilution	IV: Draw up 10 mL (10 mmol) and add 10 mL of water for injection to make a final volume of 20 mL with a concentration of 0.5 mmol/mL. It can also be diluted with sodium chloride 0.9%, dextrose 5% or other standard electrolyte solutions. PO: IV ampoules may be used orally. Draw up 10 mL (10 mmol) and add 10 mL of water for injection to make a final volume of 20 mL with a concentration of 0.5 mmol/mL.
Administration	IV: Infuse over at least 30 minutes (via central IV line if possible). Maximum rate in a medical emergency is 10 mmol/minute. PO: Administer 1–3 hours after feeds.
Monitoring	Monitor acid-base balance. Monitor local infusion site for signs of extravasation.
Contraindications	Respiratory or metabolic alkalosis.
Precautions	Hypercarbia or hypernatraemia
Drug Interactions	Concurrent use of ketoconazole may decrease ketoconazole exposure. Avoid simultaneous administration of sodium bicarbonate and catecholamines (dopamine, dobutamine, adrenaline (epinephrine), noradrenaline (norepinephrine)) through the same IV catheter or tubing as the sodium bicarbonate solution will inactive the catecholamine.
Adverse Reactions	Hypernatraemia, hyperosmolality, hypocalcaemia, hypokalaemia. May increase intracellular acidosis. If administered during inadequate ventilation, $PaCO_2$ may rise — thereby exacerbating acidosis. Rapid correction may be associated with IVH. Local tissue necrosis — thrombosis at site of administration Metabolic alkalosis and tetany. Abdominal cramping, nausea, vomiting.
Compatibility	Fluids: Glucose 5%, glucose 10%, glucose in sodium chloride solutions, sodium chloride 0.9%, sodium chloride 0.45%. Y site: Aciclovir, amifostine, amikacin, atropine, aztreonam, bivalirudin, ceftaroline fosamil, ceftazidime, dexamethasone, dexmedetomidine, digoxin, doripenem, fentanyl, filgrastim, fluconazole, furosemide, gentamicin, granisetron, heparin sodium, hydrocortisone sodium succinate, ibuprofen lysine, indometacin, insulin, lignocaine, linezolid, metronidazole, morphine,

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	penicillin G, phenobarbitone, potassium chloride, ranitidine, remifentanil, vancomycin.
Incompatibility	Amino acid solution, adrenaline (epinephrine) hydrochloride, amiodarone, amoxicillin, amphotericin B, ampicillin, anidulafungin, atracurium, azathioprine, buprenorphine, calcium folinate, calcium salts, caspofungin, cefotaxime, cefoxitin, clindamycin, chlorpromazine, clonazepam, diazoxide, dobutamine, dolasetron, dopamine, ganciclovir, glycopyrrolate, haloperidol lactate, hydromorphone, imipenem-cilastatin, ketamine, labetalol, lipid emulsion, magnesium salts, metoclopramide, midazolam, mycophenolate mofetil, noradrenaline (norepinephrine), ondansetron, pentamidine, pethidine, promethazine, streptomycin, suxamethonium, thiopentone, ticarcillin-clavulanate, vancomycin, verapamil.
Stability	
Storage	Store vials below 30°C. Diluted solutions may be stored for up to 24 hours at 2–8°C.
Special Comments	Rapid onset of action after IV administration.
Evidence summary	**During resuscitation** If effective spontaneous cardiac output is not restored despite adequate ventilation and adequate chest compressions, reversing intracardiac acidosis may improve myocardial function and achieve a spontaneous circulation. There are insufficient data to recommend routine use of bicarbonate in resuscitation of the newly born. The hyperosmolarity and carbon dioxide-generating properties of sodium bicarbonate may impair myocardial and cerebral function. Use of sodium bicarbonate is not recommended during brief CPR. If it is used during prolonged arrests unresponsive to other therapy, it should be given only after adequate ventilation and circulation is established with CPR. A dose of 1–2 mmol/kg may be given by slow intravenous injection after adequate ventilation and perfusion have been established (ILCOR 2015 recommendations).[1,2] **Preterm neonates with metabolic acidosis** Lawn et al, in their Cochrane review, found two small randomised controlled trails that fulfilled the eligibility criteria (Corbet 1977; Dixon 1999) and one unpublished pilot trial (Lawn 2005). Corbet 1977 compared treating infants with sodium bicarbonate infusion (N = 30) versus no treatment (N = 32) and did not find evidence of an effect on mortality [relative risk (RR) 1.39 (95% confidence interval 0.72 to 2.67)] or in the incidence of intra/periventricular haemorrhage [RR 1.24 (95% confidence interval 0.47 to 3.28)]. Addition of the unpublished data of Lawn 2005 does not change the overall estimate of effect on mortality [typical RR 1.45 (95%CI 0.82 to 2.56)]. Dixon 1999 compared treatment with sodium bicarbonate (N = 16) versus fluid bolus (N = 20). The primary outcome assessed was arterial blood pH/base excess two hours after the intervention. Other clinical outcomes were not reported. Neither trial assessed longer term neurodevelopmental outcomes. There is insufficient evidence from randomised controlled trials to determine whether infusion of base or fluid bolus reduces morbidity and mortality in preterm infants with metabolic acidosis. **Rapid correction of metabolic acidemia in the first 24 hours of life in preterm neonates** There is no evidence available from randomised controlled trials to support or refute the rapid correction of metabolic acidaemia, in LBW infants in the first 24 hours of life, as compared with slow or no correction.[4] **Correction of chronic metabolic acidosis in chronic kidney conditions** Metabolic acidosis is a feature of chronic kidney disease (CKD) due to the reduced capacity of the kidney to synthesise ammonia and excrete hydrogen ions. It has adverse consequences on protein and muscle metabolism, bone turnover and the development of renal osteodystrophy. Metabolic acidosis may be corrected by oral bicarbonate supplementation or, in dialysis patients, by increasing the bicarbonate concentration in dialysate fluid. Roderick et al performed a Cochrane review to examine the benefits and harms of treating metabolic acidosis in patients with CKD, both prior to reaching end-stage renal disease (ESRD) and whilst on renal replacement therapy (RRT), with sodium bicarbonate or increasing the bicarbonate concentration of dialysate. They identified three trials in adult dialysis patients (n = 117). There were insufficient data for most outcomes for meta-analysis. In all three trials, acidosis improved in the intervention group though there was variation in achieved bicarbonate concentration. There was no evidence of effect on blood pressure or sodium concentrations. Some measures of nutritional status/protein

metabolism (e.g. SGA, NP NA) were significantly improved by correction in the one trial that looked at these in detail. There was heterogeneity of the effect on serum albumin in two trials. Serum PTH fell significantly in the two trials that estimated this, with no significant effect on calcium or phosphate though both fell after correction. Complex bone markers were assessed in one study, with some evidence for a reduction in bone turnover in those with initial high bone turnover and an increase in low turnover patients. The studies were underpowered to assess clinical outcomes; in the one study that did there was some evidence for a reduction in hospitalisation after correction. In conclusion, the evidence for the benefits and risks of correcting metabolic acidosis is very limited with no RCTs in pre-ESRD patients, none in children and only three small trials in dialysis patients. These trials suggest there may be some beneficial effects on both protein and bone metabolism but the trials were underpowered to provide robust evidence.

Slow infusion versus rapid IV bolus

van Alfen-van der Velden et al performed an RCT to study the effects of $NaHCO_3$ administration on cerebral haemodynamics and oxygenation in preterm neonates. Twenty-nine preterm infants with metabolic acidosis were randomised into two groups (values are mean ± SD): In group A (GA 30.5 ± 1.7 weeks, b.w. 1,254 ± 425 g) $NaHCO_3$ 4.2% was injected as a bolus. In group B (GA 30.3 ± 1.8 weeks, b.w. 1,179 ± 318 g) $NaHCO_3$ 4.2% was administered over a 30-min period. Concentration changes of oxyhemoglobin (cO_2Hb) and deoxyhemoglobin (cHHb) were assessed using near-infrared spectrophotometry. Changes in HbD (= cO2Hb − cHHb) represent changes in cerebral blood oxygenation and changes in ctHb (= cO2Hb + cHHb) reflect changes in cerebral blood volume. Cerebral blood flow velocity was intermittently measured using Doppler ultrasound. Longitudinal data analysis was performed using linear mixed models, to account for the fact that the repeated observations in each individual were correlated. Administration of $NaHCO_3$ resulted in an increase of cerebral blood volume which was more evident if $NaHCO_3$ was injected rapidly than when infused slowly. HbD and cerebral blood flow velocity did not show significant changes in either group. Conclusion: To minimise fluctuations in cerebral hemodynamics, slow infusion of sodium bicarbonate is preferable to rapid injection.

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