Sodium bicarbonate
Newborn use only

2021

Alert	Rapid infusion is associated with increased incidence of intraventricular haemorrhage (IVH) in preterm infants. Avoid simultaneous administration of sodium bicarbonate and catecholamines through the same IV catheter or tubing as the sodium bicarbonate solution will inactive the catecholamine. During prolonged resuscitation sodium bicarbonate should only be given after adequate ventilation and circulation is established with CPR. Conversion factor for sodium bicarbonate: 1 mmol = 1 mEq
Indication	Metabolic acidosis Prolonged resuscitation Renal tubular acidosis Chronic renal failure Gastro-intestinal bicarbonate loss
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	Alkalinising agent
Trade name	Sodium Bicarbonate 8.4% Injection [Phebra]; Pfizer (Australia) Sodium Bicarbonate 8.4% Injection BP, Sodibic-840 mg capsule
Presentation	**IV:** 8.4% (1 mmol/mL) 10 mL or 100mL Vial. **ORAL:** IV preparation as oral. Sodium bicarbonate 10mmol capsule (Sodibic-840 mg).
Dose	**IV** 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 re-assess for the need of remainder. **ORAL** 1-2 mmol/kg/day in 3-4 divided doses. Dose is adjusted according to response.
Dose adjustment	
Maximum dose	
Total cumulative dose	
Route	IV PO
Preparation	Dilute to a maximum concentration of no greater than 0.5 mmol/mL (osmolarity = 1000 mOsm/L). **IV and Oral using IV preparation**: Draw up 10 mL (10 mmol) sodium bicarbonate and add 10 mL of water for injection or glucose 5% or sodium chloride 0.9% to make a final volume of 20 mL with a concentration of 0.5 mmol/mL. **Oral sodium bicarbonate using Sodibic-840 mg capsule**: Disperse the contents of the capsule (10 mmol) in 10 mL of water for injections to make a final concentration of 1 mmol/mL. Make a fresh preparation for each dose.
Administration	IV: Infuse over at least 30 minutes preferably via central IV line. Flush the cannula and IV line with sodium chloride 0.9% following administration to avoid inactivation and precipitation of other medications. Maximum rate in a medical emergency is 10 mmol/minute. Oral: Administer 1–3 hours after feeds.
Monitoring	Acid-base balance. Local infusion site for signs of extravasation.
Contraindications	Respiratory or metabolic alkalosis.
Precautions	Hypercarbia or hypernatraemia. Slow administration rate is recommended to minimise the possibility of producing hypernatraemia, decreasing cerebrospinal fluid pressure and inducing intracranial haemorrhage.
Drug interactions	May decrease effectiveness of aspirin, phenobarbitone and lithium. May inactivate drugs such as benzylpenicillin, potassium, isoprenaline and suxamethonium on mixing. Hyperchloraemic alkalosis may occur if sodium bicarbonate is used in conjunction with potassium depleting diuretics such as furosemide and hydrochlorothiazide. 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, exacerbating acidosis. Rapid correction may be associated with IVH. Local tissue necrosis and 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, amikacin, atropine, aztreonam, benzylpenicillin, cefalotin, cefazolin, ceftazidime, ceftriaxone, clindamycin, dexamethasone, dexmedetomidine, digoxin, esmolol, fentanyl, filgrastim, fluconazole, furosemide, gentamicin, heparin sodium, hydrocortisone sodium succinate, ibuprofen lysine, indometacin, insulin,[14] lignocaine, linezolid, metronidazole, methylprednisolone sodium succinate, morphine, naloxone, octreotide, phenobarbitone, piperacillin/tazobactam, potassium chloride, protamine, pyridoxine, ranitidine, remifentanil, sodium nitroprusside, tobramycin, vancomycin,[14] vasopressin, vecuronium, voriconazole.[14]
Incompatibility	Amino acid solution, adrenaline (epinephrine) hydrochloride, amiodarone, amoxicillin, amphotericin B, ampicillin, atracurium, calcium folinate, calcium salts, cefotaxime, cefoxitin, clonazepam, diazoxide, dobutamine, dopamine, ganciclovir, hydromorphone, imipenem-cilastatin, ketamine, labetalol, lipid emulsion, magnesium salts, metoclopramide, midazolam, noradrenaline (norepinephrine), suxamethonium, thiamine, thiopentone.
Stability	
Storage	Store below 30°C. Diluted solutions may be stored for up to 24 hours at 2–8°C.
Excipients	Disodium edetate, water for injections.
Special comments	Rapid onset of action after IV administration.
Evidence	**<u>During resuscitation</u>** There is insufficient evidence from randomised controlled trials to determine whether the infusion of sodium bicarbonate reduces mortality and morbidity in infants receiving resuscitation in the delivery room at birth.[2] **<u>Preterm neonates with metabolic acidosis</u>** 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. **<u>Rapid correction of metabolic acidemia in the first 24 hours of life in preterm neonates</u>** 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] **<u>Correction of chronic metabolic acidosis in chronic kidney conditions</u>** 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. **<u>Slow infusion versus rapid IV bolus</u>** 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.
Practice points	2020 Neonatal Resuscitation Algorithm has made no recommendation for sodium bicarbonate for neonatal resuscitation.[1]
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