Acid-Base Balance

INTRODUCTION: The newborn infant is subject to numerous conditions that may disturb acid-base homeostasis. Management of ventilation, which controls the respiratory component of acid-base balance, is discussed in the section on Respiratory Support (P. 10). This section is a brief discussion of the metabolic aspects of acid-base balance.

METABOLIC ACIDOSIS, defined as a **base deficit >5 mEq/L on the first day and >4 mEq/L thereafter**, occurs from:

- Loss of buffer (mainly bicarbonate) *or*
- Excess production of acid or decreased excretion of acid

The anion gap is a useful calculation in assessing metabolic acidosis.

Anion gap = $[Na^+] - ([Cl^-] + [HCO_3^-])$

Loss of buffer has no effect on anion gap. Accumulation of organic acid (*e.g.*, lactic acid) causes an increase in anion gap.

Normal anion gap: <15 mEq/L

Increased anion gap:

>15 mEq/L in LBW infants (<2,500 g)
>18 mEq/L in ELBW infants (<1,000 g)

Newborn infants normally have a base deficit of 1 to 3 mEq/L.

Common causes of metabolic acidosis:

- Bicarbonate loss, especially via immature kidney or from GI tract
- Lactic acidosis from inadequate tissue perfusion and oxygenation (*e.g.*, from asphyxia, shock, severe anemia, hypoxemia, PDA, NEC, excessive ventilator pressures with ↓ cardiac output)
- Hypothermia
- Organic acidemia due to an inborn error of metabolism (see P. 155)
- Excessive Cl in IV fluids
- Renal failure
- Excessive acid load from high protein formula in preterm (late metabolic acidosis of prematurity)
- Excretion of HCO_3^- as metabolic compensation for respiratory alkalosis

Dilution acidosis is caused by excessive volume expansion (with saline, Ringer's lactate or dextrose solutions). The extracellular space becomes "diluted" (relative decrease of HCO_3^-); carbonic acid dissociates more and liberates more H^+. Therefore, pH falls.

Effects of metabolic acidosis: Major physiological effects of metabolic acidosis include:

- Pulmonary vasoconstriction (with risk of persistent pulmonary hypertension)
- ↓ myocardial contractility
- Shift of O_2-Hgb dissociation curve to right (↓ saturation at a given PO_2)
- CNS damage with severe acidosis

- ↑ work of breathing as compensation for acidosis

Management of metabolic acidosis:

- Treat underlying cause when possible
- Do not treat metabolic acidosis by hyperventilation (other than briefly while preparing to give alkali). This may correct pH but has deleterious effects on cardiac output and pulmonary blood flow.
- Volume expansion (*i.e.*, bolus 10 mL/kg of 0.9% NaCl) should not be used to treat acidosis unless there are signs indicative of hypovolemia. A volume load is poorly tolerated in severe acidosis because of the ↓ myocardial contractility.
- **Alkali treatment** should be used only if significant metabolic acidosis is present (*e.g.*, pH <7.30 with base deficit >7)
- Dose of alkali for treatment of metabolic acidosis can be calculated by:

  Dose of alkali (mEq) = base deficit x 0.3 x body weight (kg)

- Administer alkali IV at a rate not exceeding 1 mEq/kg/min.
- The usual alkali used in newborns is $NaHCO_3$ and the concentration is 0.5 mEq/mL, so it is hyperosmolar (900 mOsm/L)
- Do not give $NaHCO_3$ unless the infant is receiving assisted ventilation that is adequate. With inadequate ventilation, $NaHCO_3$ will worsen acidosis because of the liberation of CO_2.
- With severe acidosis and CO_2 retention despite vigorous assisted ventilation, consider use of the organic buffer, THAM™. This is provided as a 0.3 molar solution (*i.e.*, 0.3 mEq/mL)

Risks of alkali administration include:

- Acute hyperosmolality with rapid shift of water from intracellular to extracellular space
- The intracellular dehydration increases the risk of intracranial hemorrhage
- Acute expansion of intravascular volume
- ↓ ionized Ca^{++}
- Shift of O_2-Hgb dissociation curve to left (↑ binding of O_2 to Hgb)
- Paradoxical CNS acidosis
- With bicarbonate: sodium load and increased CO_2
- With THAM™, risk of apnea and hypoglycemia

For correction of acidosis in an emergency, see section on Resuscitation (P. 1).

For **chronic mild metabolic acidosis** in small premature infants on hyperalimentation, maximize acetate and minimize chloride in the solution.

METABOLIC ALKALOSIS is usually iatrogenic in premature infants related to diuretic use or GI losses and occurs in combination with contracted intravascular and ECF volumes.

Cause of Metabolic Alkalosis	Treatment
Compensation for respiratory acidosis	Correct ventilation
Diuretic Rx (especially furosemide) (contraction alkalosis)	Decrease diuretic dose, add spironolactone, replace K^+ and Cl^- deficit
Loss of gastric fluid from vomiting or diarrhea with Cl^- loss	Replace deficit and give fluids and electrolytes to keep pace with continuing losses.
Increased alkali load from feedings (alkalosis of prematurity)	↑ Cl^- administration as KCl or Arginine Cl
Excessive administration of alkali (Excess acetate in parenteral nutrition)	↑ Cl^- as KCl or Arginine Cl
Bartter syndrome (rare)	Replace electrolyte losses