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⁻] + [HCO₃⁻])

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 HCO₃⁻ 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 HCO₃⁻); 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 O₂-Hgb dissociation curve to right (↓ saturation at a given PO₂)
• 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:
  \[ \text{Dose of alkali (mEq)} = \text{base deficit} \times 0.3 \times \text{body weight (kg)} \]
- Administer alkali IV at a rate not exceeding 1 mEq/kg/min.
- The usual alkali used in newborns is NaHCO₃ and the concentration is 0.5 mEq/mL, so it is hyperosmolar (900 mOsm/L)
- Do not give NaHCO₃ unless the infant is receiving assisted ventilation that is adequate. With inadequate ventilation, NaHCO₃ will worsen acidosis because of the liberation of CO₂.
- With severe acidosis and CO₂ 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 Ca²⁺
- Shift of O₂-Hgb dissociation curve to left (↑ binding of O₂ to Hgb)
- Paradoxical CNS acidosis
- With bicarbonate: sodium load and increased CO₂
- 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.

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