

Chapter 9: Acid Base Balance

Ashleigh N. Rushing, MD and C. Lydia Wraight, MD

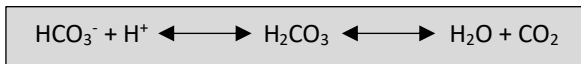
A. Blood Gases

- pH, $p\text{CO}_2$, $p\text{O}_2$ are measured directly
- HCO_3^- , O_2 saturation are calculated

Normal Blood Gas Values	
pH	7.35-7.45
$p\text{CO}_2$	35-45*
HCO_3^-	22-26
Base Excess	-1 to -3

* For infants ventilated > 7 days, permissive hypercapnea ($p\text{CO}_2$ 45-60) is accepted to decrease barotrauma.

- Acid-base balance is maintained with buffers



B. Interpretation of blood gases

- **Step 1:** Is the pH acidic or basic?

pH < 7.35	Acidosis
pH > 7.45	Alkalosis

- **Step 2:** Is the disturbance respiratory or metabolic? Look at $p\text{CO}_2$.
 - Respiratory = pH and $p\text{CO}_2$ change in **opposite** directions
 - Metabolic = pH and $p\text{CO}_2$ change in the **same** direction

	Acidosis		Alkalosis	
	pH	$p\text{CO}_2$	pH	$p\text{CO}_2$
Respiratory	↓	↑	↑	↓
Metabolic	↓	↓	↑	↑

• **Step 3:** Is there compensation?

- Compensation occurs to bring the pH closer to normal when the derangement is chronic, it will not correct the pH to normal.

	Acute		Compensation	
	pCO_2	HCO_3	pCO_2	HCO_3
Respiratory Alkalosis	< 40	normal	< 40	low
Respiratory Acidosis	> 40	normal	> 40	high
Metabolic Alkalosis	normal	> 26	> 40	> 26
Metabolic Acidosis	normal	< 22	< 40	< 22

• **Step4:** Is there an anion gap?

$$\text{Anion Gap} = [\text{Na}^+] - ([\text{Cl}^-] + [\text{HCO}_3^-])$$

– Normal anion gap < 15 mEq/L

C. Respiratory Acidosis

- Most common cause of acidosis
- Poor ventilation results in CO_2 retention (CO_2 is high)

D. Respiratory Alkalosis

- Often iatrogenic, resulting from hyperventilation (ie we are giving the baby more support than they need)

E. Metabolic Acidosis

- Results from excess acid production or increased loss of base.
- **Normal anion gap:**
 - Renal: Immaturity, Renal Tubular Acidosis, Obstruction, Dysplasia
 - GI: Diarrhea, Short Gut

- Endo: Congenital Adrenal Hyperplasia
- High Protein Formula
- Administration of Cl⁻ containing compounds:
 - TPN, NH₄Cl, CaCl₂ especially in ELBW
- Compensation of respiratory alkalosis
- **Increased anion gap:**
 - Lactic acidosis: Shock with poor tissue perfusion and oxygenation
 - Acute renal failure
 - Inborn errors of metabolism: Organic acidemias, mitochondrial disorders, glycogen storage disease (type 1), galactosemia
 - Toxins
- **Systemic effects of Metabolic Acidosis:**
 - Pulmonary vasoconstriction (Pulmonary hypertension)
 - Decreased myocardial contractility
 - Respiratory compensation: Increased work of breathing
 - CNS damage with severe acidosis
- **Management**
 - Treat the underlying cause when possible
 - TPN associated mild acidosis may be treated with decreasing chloride and increasing acetate for a few days.
 - Volume expansion if signs of hypovolemia
 - Excessive volume expansion is poorly tolerated in presence of decreased myocardial contractility
 - May use normal saline, packed RBCs or FFP as appropriate
 - Pharmacologic options
 - NaHCO₃
 - Only use when infant has excellent ventilation
 - Risks of NaHCO₂ administration

- Acute expansion of intravascular volume with risk of IVH
- Shift of Hgb dissociation curve to left (\uparrow binding of O_2 to Hgb)
- Increased Na load and increased CO_2
- THAM – an organic buffer
 - Can consider with severe acidosis with Na overload and high CO_2 /poor ventilation
 - Associated with risk of apnea and hypoglycemia and is not as effective

F. Metabolic Alkalosis

- Commonly iatrogenic, resulting from diuretic use (ex: furosemide)
- Other etiologies:
 - Loss of gastric fluid: large gastric aspirates, emesis or diarrhea with Cl^- loss
 - Compensation for chronic respiratory acidosis
 - Excessive administration of alkali (acetate in TPN)
- **Management**
 - Replace Cl^- deficit, remove any acetate in TPN
 - Replace ongoing fluid and electrolyte losses
 - Adjust ventilator if respiratory acidosis

References:

1. Brodsky, D. and Martin, C. **Acid Base Balance**. *Neonatology Review 3rd Ed.* 2020. Vol 4: 34-3.