

# Arterial Blood Gas (ABG)

## Interpretation

Suspected blood acid-base disorder?

Read ABGs: pH/pCO<sub>2</sub>/pO<sub>2</sub>/HCO<sub>3</sub><sup>-</sup>

Normal values: 7.40/40/80/24

(stick w/ above #s for calculations)

pH: 7.35-7.45

pCO<sub>2</sub>: 37-42

pO<sub>2</sub>: 80-100

HCO<sub>3</sub><sup>-</sup>: 22-26

1. Look at **pH**. What is the acid/base disturbance?

2. Look at **pCO<sub>2</sub>** & **HCO<sub>3</sub><sup>-</sup>**. Which element is driving the pH?

3. Is there a **compensation**? If so, what is it?

(ratios = appropriate compensation; inappropriate comp'n = 2<sup>o</sup> process!)

4. What **caused** this acid-base disorder?

(see if the cause correlates with the clinical picture! i.e. COPD pts usually have resp acidosis)

**Acidemia**  
pH < 7.35

**Normal pH**  
(7.35-7.45)

→ Normal ABGs

→ Mixed Acid-base disorder

**Alkalemia**  
pH > 7.45

HCO<sub>3</sub><sup>-</sup> < 24 mmol/L  
→ **Metabolic Acidosis**

pCO<sub>2</sub> > 40mmHg  
→ **Respiratory Acidosis**

HCO<sub>3</sub><sup>-</sup> > 24 mmol/L  
→ **Metabolic Alkalosis**

pCO<sub>2</sub> < 40mmHg  
→ **Respiratory Alkalosis**

*Lungs expel CO<sub>2</sub>*  
ΔHCO<sub>3</sub><sup>-</sup> : Δ pCO<sub>2</sub>

↓ 10 : ↓ 12

**Calculate Anion Gap using electrolytes!**

*Law of mass action*

*Kidneys retain HCO<sub>3</sub><sup>-</sup>*

**Acute**

ΔHCO<sub>3</sub><sup>-</sup> : Δ pCO<sub>2</sub>  
↑ 1 : ↑ 10

**Chronic**

ΔHCO<sub>3</sub><sup>-</sup> : Δ pCO<sub>2</sub>  
↑ 3 : ↑ 10

*Lungs retain CO<sub>2</sub>*  
Δ HCO<sub>3</sub><sup>-</sup> : Δ pCO<sub>2</sub>

↑ 10 : ↑ 7

**H<sup>+</sup> loss**

(kidneys unable to secrete HCO<sub>3</sub><sup>-</sup>)  
→ Renal excretion (loop diuretics, excess aldosterone)  
→ GI excretion (vomiting, antacids, diarrhea)  
→ Hypokalemia  
→ Conn's Syndrome

*Law of mass action*

*Kidneys expell HCO<sub>3</sub><sup>-</sup>*

**Acute**

ΔHCO<sub>3</sub><sup>-</sup> : Δ pCO<sub>2</sub>  
↓ 2 : ↓ 10

**Chronic**

ΔHCO<sub>3</sub><sup>-</sup> : Δ pCO<sub>2</sub>  
↓ 4 : ↓ 10

**Hyper-ventilation, etc**

(rarer than Resp Acidosis, b/c hypoventilation due to damaged lungs = more common)

**Notes:**

→ If chronic compensation is incomplete, could mean mixed acid-base disturbances!  
→ Easier to lower than to raise pCO<sub>2</sub> or HCO<sub>3</sub><sup>-</sup>. In resp compensation, pCO<sub>2</sub> is lowered more than it's raised. In metabolic compensation, HCO<sub>3</sub><sup>-</sup> is lowered more than its raised.

**High (>12) (acid gain)**

→ Acetylsalicylic acid  
→ Methanol (alcohols)  
→ Uremia  
→ Diabetic ketoacidosis  
→ Propylene glycol  
→ Isoniazid, Iron  
→ Lactic acidosis  
→ Ethylene glycol

**Anion Gap = Na - (HCO<sub>3</sub> + Cl)**

High Anion gap = buffering of abnormal acid w/ HCO<sub>3</sub><sup>-</sup>

**Normal (9-12) (HCO<sub>3</sub><sup>-</sup> loss, replaced by Cl<sup>-</sup>)**

→ Renal loss (RTA, interstitial nephritis)  
→ GI loss (i.e. diarrhea)

**Δ Anion Gap (AG - 12) should = consumption of HCO<sub>3</sub> (24 - HCO<sub>3</sub>)**

Δ Anion gap < Δ HCO<sub>3</sub><sup>-</sup> means anion gap + non-anion gap acidosis (i.e. bicarb loss)

Δ Anion gap > Δ HCO<sub>3</sub><sup>-</sup> means anion gap acidosis + metabolic alkalosis

**Hypo-ventilation**

→ Central causes (drugs, coma, hypo-thyroid)  
→ Damaged lung tissue (COPD, asthma, interstitial lung dx)  
→ Chest wall defects (kypho-scoliosis)  
→ Neuromuscular defects