An approach to ABG interpretation

Western Health

1. Is there **hypoxia present**? Assess for an alveolar-arterial gradient
   a. https://www.mdcalc.com/a-a-o2-gradient
   b. Normal A-a gradient 5-15 mmHg (increases with age/smoker/higher Fi02 (i.e. 3+(0.21xage))
   c. If elevated, suspect disease of V/Q mismatch (improves with O2 therapy e.g. asthma, COPD, ILD, pulmonary vascular disease), or shunt (doesn't improve with O2 therapy e.g. atelectasis, APO, vascular shunt in lungs)

2. Is there an **acid base disorder** present?
   a. pH < 7.35 acidemia
   b. pH > 7.45 alkalemia

	Arterial
pH	7.35-7.45
PaO2 (mmHg)	80-100
PaCO2 (mmHg)	35-45
HCO3 (mmol/L)	22-26
Base excess (mmol/L)	-2 - +2

Table 1: Blood gas reference ranges

3. Is the **disturbance respiratory or metabolic** (table 2)?
   a. In primary **respiratory** disorders, the pH and PaCO2 change in ***opposite*** directions
   b. In **metabolic** disorders the pH and $PaCO_2$ change in the ***same*** direction.

Acidosis	Respiratory	pH ↓	$PaCO_2$ ↑
Acidosis	Metabolic&	pH ↓	$PaCO_2$ ↓
Alkalosis	Respiratory	pH ↑	$PaCO_2$ ↓
Alkalosis	Metabolic	pH ↑	$PaCO_2$ ↑

Table 2: Type of disturbance

4. Is there **appropriate compensation** for the primary disturbance (Table 3)?
   a. If the observed compensation is not the expected compensation, it is likely that more than one acid-base disorder is present

Disorder	Expected compensation	Correction factor
Metabolic acidosis	$PaCO_2$ = (1.5 x $[HCO_3^-]$) +8	± 2
Acute respiratory acidosis	Increase in $[HCO_3^-]$= Δ $PaCO_2$/10	± 3
Chronic respiratory acidosis (3-5 days)	Increase in $[HCO_3^-]$= 3.5(Δ $PaCO_2$/10)	
Metabolic alkalosis	Increase in $PaCO_2$ = 40 + 0.6(ΔHCO_3^-)	
Acute respiratory alkalosis	Decrease in $[HCO_3^-]$= 2(Δ $PaCO_2$/10)	
Chronic respiratory alkalosis	Decrease in $[HCO_3^-]$ = 5(Δ $PaCO_2$/10) to 7(Δ $PaCO_2$/10)	

Table 3: Assessing for compensation

5. Calculate the **anion gap if a metabolic acidosis** exists: AG= [Na+]- [Cl-] - $[HCO_3^-]$
   a. Normal anion gap: 8-12meq/L, normal range lower in hypoalbuminaemic pts (2.5 meq/L lower for each 1 gm/dL decrease in the plasma albumin concentration)
   b. If high anion gap, consider calculating osmolal gap
      i. OSM gap = measured OSM – (2[Na+] - glucose/18 – urea/2.8); normal OSM gap < 10

6. Is there a **mixed/complex acid base disturbance** (table 4)?

Disorder	Characteristics	Selected situations
Respiratory acidosis with metabolic acidosis	↓in pH ↓ in HCO_3 ↑ in $PaCO_2$	• Cardiac arrest • Intoxications • Multi-organ failure
Respiratory alkalosis with metabolic alkalosis	↑in pH ↑ in HCO_3^- ↓ in $PaCO_2$	• Cirrhosis with diuretics • Pregnancy with vomiting • Over ventilation of COPD
Respiratory acidosis with metabolic alkalosis	pH in normal range ↑ in $PaCO_2$, ↑ in HCO_3^-	• COPD with diuretics, vomiting, NG suction • Severe hypokalemia
Respiratory alkalosis with metabolic acidosis	pH in normal range ↓ in $PaCO_2$ ↓ in HCO_3	• Sepsis • Salicylate toxicity • Renal failure with CHF or pneumonia • Advanced liver disease
Metabolic acidosis with metabolic alkalosis	pH in normal range HCO_3^- normal	• Uremia or ketoacidosis with vomiting, NG suction, diuretics, etc.

Table 4: Selected mixed and complex acid-base disturbances

Produced by: Dr Fiona Smith, Dr Juliana Dong

Causes of acid-base disturbances

Respiratory acidosis	Metabolic acidosis	Respiratory alkalosis	Metabolic alkalosis

• COPD, asthma, other obstructive lung disease
• CNS depression – opioids, sedatives
• OSA/OHS
• Neuromuscular impairment
• Increased CO2 production: shivering, rigors, seizures, malignant hyperthermia, hypermetabolism, increased intake of carbohydrates
	<u>Elevated</u> anion gap: MUD PILES
	• Methanol intoxication
	• Uraemia
	• Diabetic ketoacidosis, alcoholic ketoacidosis, starvation ketoacidosis
	• Paraldehyde toxicity
	• Isoniazid
	• Lactic acidosis
	  ○ Type A: tissue ischemia
	  ○ Type B: Altered cellular metabolism
	• Ethanol or ethylene glycol intoxication (osmolal gap)
	• Salicylate intoxication
	
	<u>Normal</u> anion gap: will have increase in [Cl-]
	• GI loss of HCO_3-
	  ○ Diarrhea, ileostomy, proximal colostomy, ureteral diversion
	• Renal loss of HCO_3-
	  ○ proximal RTA
	  ○ carbonic anhydrase inhibitor (acetazolamide)
	• Renal tubular disease
	  ○ ATN
	  ○ Chronic renal disease
	  ○ Distal RTA
	  ○ Aldosterone inhibitors or absence
	  ○ NaCl infusion, TPN, NH_4+ administration
		• Hyperventilation (fever, pain, fear, anxiety, CVA, CNS trauma/tumor/ infection
		• Hypoxemia or hypoxia (profound anemia, low FiO2)
		• Lung diseas – APO, pleural effusion, pneumonia, pneumothorax, pulmonary embolus
		• Pregnancy, liver disease, sepsis, hyperthyroidism
			Hypovolemia with Cl- depletion
			• GI loss of H+
			○ Vomiting, gastric suction, villous adenoma, diarrhea with chloride-rich fluid
			• Renal loss H+
			○ Loop and thiazide diuretics, post-hypercapnia (especially after institution of mechanical ventilation)
			
			Hypervolemia, Cl- expansion
			• Renal loss of H+:
			  ○ Oedematous states (heart failure, cirrhosis, nephrotic syndrome),
			  ○ Endocrines disorder: hyperaldosteronism, hypercortisolism, excess ACTH, exogenous steroids, hyperreninemia
			  ○ Severe hypokalemia
			  ○ Renal artery stenosis
			  ○ Bicarbonate administration

Reference link for more details:
https://www.thoracic.org/professionals/clinical-resources/critical-care/clinical-education/abgs.php
https://www.nps.org.au/australian-prescriber/articles/the-interpretation-of-arterial-blood-gases_

Produced by: Dr Fiona Smith, Dr Juliana Dong