

BLOOD GAS INTERPRETATION

I. SOURCE OF BLOOD GAS

- 1) ABG: Gives you information about acid-base status as well as oxygenation.
- 2) VBG: Gives you information about acid-base status. Also tells you about the metabolic demands of the body. SvO₂ should be 20-25% less than SaO₂. If the (SaO₂-SvO₂) > 25%, this indicates decreased CO and inadequate tissue perfusion. If the (SaO₂-SvO₂) < 25%, this indicates high output CO state (i.e. early sepsis, high output cardiac failure).

Typically, in a VBG, the pH will run 0.5-1 pH units lower than an ABG, and the pCO₂ will run 5-10mmHg higher than an ABG.

II. POTENTIAL SAMPLING ERRORS

- 1) Air in the sample: Will elevate the pO₂ in the sample, rendering it inaccurate.
- 2) Too much time before analyzing: Gas must be placed on ice if not analyzed w/in 20 min. Certain cells may continue to consume O₂ and produce CO₂ while in the syringe. This will affect your pO₂ and pCO₂ unless the sample is not analyzed right away.
- 3) Anticoagulants: Heparin can dilute the concentrations of gases in the blood sample (will decrease pCO₂).
- 4) Temperature: The temperature of the blood affects the pO₂ and pCO₂. Blood gases measured in the lab are measured at 37°C and are called *alpha-stat*. Blood gases can also be corrected to the patient's temperature, otherwise known as *pH-stat*. However, routine correction for temperature is not recommended because we do not understand the physiologic implications of normal blood gas tensions at various temperatures.

III. BLOOD GAS INTERPRETATION

COMPONENTS

pH. This is the negative log of the hydrogen ion concentration in the blood: $-\log [H^+]$

2. pCO₂. This is the partial pressure of CO₂ in the blood.

(i) Determined by the ratio of CO₂ production (VCO₂) in the body and the degree of alveolar minute ventilation (MV_a): $pCO_2 = VCO_2/MV_a$

(ii) $MV_a = RR \times TV_a$

Where RR is respiratory rate and TV_a refers to the tidal volume, which is tidal volume (TV) – dead space (VD).

pO₂. This is the partial pressure of oxygen in the blood. *This is not the same as SaO₂ which tells you how much of the hemoglobin is saturated with oxygen.* pO₂ is an indicator how tightly bound the oxygen molecules are to the hemoglobin and of oxygen delivery to the tissues.

HCO₃⁻. This is the concentration of bicarbonate ion in the blood.

Base excess/deficit. *This is a calculated value.* This is the difference between the normal serum HCO₃⁻ of 24 and the patient's measured serum HCO₃⁻, multiplied by 1.2 .

$$\text{Base excess/deficit} = -1.2(24 - \text{measured HCO}_3^-)$$

This formula takes into account the fact that only 75% of the buffer action of the blood is due to HCO₃⁻, and that hemoglobin, phosphate, and other proteins buffer the rest.

NORMAL RANGES (ON ROOM AIR)	ABG	VBG
pH	7.35-7.45	7.25-7.35
pCO ₂	35-45	41-51
pO ₂	80-100	35-40
HCO ₃	22-26	22-26
Base excess/deficit	+/- 2	+/- 2

INTERPRETATION: First, does the patient have an acidosis or an alkalosis? Second, is the primary problem metabolic or respiratory? Third, is there any compensation by the patient? (And don't forget that respiratory compensation is immediate, but it takes the kidneys a while to kick in...) After determining these things, then you can evaluate how effectively the patient is oxygenating. *For the rest this chapter, when referring to blood gas interpretation, we will be using ABGs.*

1) LOOK AT THE pH! This will determine the primary problem.

pH < 7.35 Acidosis (metabolic and/or respiratory)

pH > 7.45 Alkalosis (metabolic and/or respiratory)

2) LOOK AT THE pCO₂ (high or low)!

paCO₂ > 45mmHg Respiratory Acidosis (alveolar hypoventilation)

paCO₂ < 35mmHg Respiratory Alkalosis (alveolar hyperventilation)

3) LOOK AT THE HCO₃⁻ (high or low)!

HCO₃⁻ < 22mEq/L Metabolic Acidosis

HCO₃⁻ > 26mEq/L Metabolic Alkalosis

PUTTING IT TOGETHER—RESPIRATORY

paCO₂ > 45 with a pH < 7.35 represents a respiratory acidosis.

paCO₂ < 36 with a pH > 7.45 represents a respiratory alkalosis.

For a primary respiratory problem, pH and paco₂ will move in OPPOSITE directions.

--For an acute change in paco₂ of 10 mmHg, the pH will change by 0.08 units.

If the pH and paco₂ do not agree according to these rules, then there must be a metabolic component.

PUTTING IT TOGETHER—METABOLIC

HCO₃⁻ > 26 with a pH > 7.45 represents a metabolic alkalosis.

For a primary metabolic problem, pH, paco₂ and HCO₃⁻ are all moving in the SAME direction.

--For each deviation in the HCO₃⁻ by 10meq/L, the pH will change in the same direction by 0.15 units.

The only way to have a “normal” blood gas is to have a normal pH and a normal paco₂, PaO₂, and HCO₃⁻. If any of those are off, then there must be some sort of...

COMPENSATION: The body’s attempt to return the acid/base status to normal.

Primary Problem

respiratory acidosis
respiratory alkalosis
metabolic acidosis
metabolic alkalosis

Compensation

metabolic alkalosis
metabolic acidosis
respiratory alkalosis
respiratory acidosis

EXPECTED COMPENSATORY MECHANISMS

For a RESPIRATORY ACIDOSIS:

--ACUTELY, for each increase in paco₂ of 10 mmHg, the pH will decrease by 0.08 units. The HCO₃⁻ will increase by 0.1-1 mEq/L for every increase in 10 mmHg paco₂.

--CHRONICALLY, for each increase in paco₂ of 10 mmHg, the pH will decrease by 0.03 units. The HCO₃⁻ will increase by 1-3.5 mEq/L for every increase in 10 mmHg paco₂.

For a RESPIRATORY ALKALOSIS:

--ACUTELY, for each decrease in paco₂ of 10 mmHg, the pH will increase by 0.08 units. The HCO₃⁻ will decrease by 0-2 mEq/L for every decrease in 10 mmHg paco₂.

--CHRONICALLY, for each decrease in paco₂ of 10 mmHg, the pH will decrease by 0.17 units. The HCO₃⁻ will decrease by 2-5 mEq/L for every decrease in 10 mmHg paco₂.

For a METABOLIC ACIDOSIS:

--For every 1mEq/L decrease in HCO₃, the paCO₂ will decrease by 1-1.5 mmHg.

OR: $paCO_2 = 1.5(HCO_3) + 8 (+/- 2)$

For a METABOLIC ALKALOSIS:

--For every 1mEq/L increase in HCO₃, the paCO₂ will increase by 0.5-1 mmHg.

OR: $paCO_2 = 0.7(HCO_3) + 20 (+/- 1.5)$

Acute Changes in Acid-Base Homeostasis

		pH	paCO ₂	HCO ₃
Acidosis	Acute Respiratory	low	high	normal
	Acute Metabolic	low	normal	low
Alkalosis	Acute Respiratory	high	low	normal
	Acute Metabolic	high	normal	high

Chronic Changes in Acid-Base Homeostasis

		pH	paCO ₂	HCO ₃
Acidosis	Chronic Respiratory	normal	high	high
	Chronic Metabolic	normal	low	low
Alkalosis	Chronic Respiratory	normal	low	low
	Chronic Metabolic	normal	high	high

ACUTE RESPIRATORY ACIDOSIS

--paCO₂ is elevated and pH is acidotic; the decrease in pH is entirely due to the increase in paCO₂.

--HCO₃ will be in the normal range because the kidneys have not had adequate time to establish effective compensatory mechanisms.

CAUSES (anything that acutely causes alveolar hypoventilation)

--Respiratory causes: airway obstruction, severe pneumonia, chest trauma/pneumothorax

--Acute drug intoxication (narcotics, sedatives)

--Residual neuromuscular blockade

--CNS disease (head trauma)

CHRONIC RESPIRATORY ACIDOSIS

- paCO₂ is elevated with a normal pH; this is secondary to an increase in HCO₃.
- Renal mechanisms increase the excretion of H⁺ w/in 24 hours and may correct the resulting acidosis caused by chronic CO₂ retention (up to a point).

CAUSES (anything that chronically causes alveolar hypoventilation)

- Chronic lung disease (BPD, COPD)
- Neuromuscular disease (SMA, Muscular dystrophy)
- Extreme obesity
- Obstructive sleep apnea
- Chest wall deformity

ACUTE RESPIRATORY ALKALOSIS

- paCO₂ is low and the pH is alkalotic; this is secondary to the decrease in paCO₂.
- HCO₃ will be in the normal range because the kidneys have not had sufficient time to establish effective compensatory mechanisms.

CAUSES (anything that causes alveolar hyperventilation)

- Pain, anxiety, hypoxemia, restrictive lung disease, severe congestive failure, pulmonary emboli, drugs, sepsis, fever, thyrotoxicosis, pregnancy, overaggressive mechanical ventilation, hepatic failure.

METABOLIC ACIDOSIS (ELEVATED ANION GAP)

CAUSES: REMEMBER MUDPILES

- Methanol
- Uremia
- Diabetic ketoacidosis, also other forms of ketoacidosis: alcoholic, starving
- Paraldehyde
- Iron, isoniazid (INH)
- Lactic acidosis—hypoxia, shock, sepsis, seizures
- Ethanol, ethylene glycol (also isopropyl alcohol)
- Salicylates

METABOLIC ACIDOSIS (NONGAP)

- CAUSES: RTA, post-respiratory alkalosis, hypoaldosteronism, potassium sparing diuretics, pancreatic loss of bicarbonate, diarrhea, carbonic anhydrase inhibitors, ureteral diversions, administration of acid (HCl, NH₄Cl, arginine HCl)**

C. ASSESSING OXYGENATION

--**HYPOXEMIA.** This is when the oxygen content of blood is decreased, i.e. $paO_2 < 60$ mmHg and $SaO_2 < 90\%$.

--**HYPOXIA.** This is when there is an inadequate amount of oxygen available to be used by the tissues and metabolic needs are not met.

MECHANISMS OF HYPOXEMIA

1) Alveolar hypoventilation

2) Altered gas exchange

--R→L shunt

--Ventilation-perfusion mismatch

--Impaired diffusion

ASSESSMENT OF GAS EXCHANGE

1) Alveolar-arterial Oxygen Tension Difference. This is otherwise known as the A-a gradient, where "A" refers to alveolar and "a" refers to arterial. A gradient of 10-20 is considered normal.

$$PAO_2 = FIO_2 (P_{atm} - PH_2O) - PaCO_2 / RQ$$

Where: PAO_2 is the partial pressure of oxygen in the alveoli

FIO_2 is the fraction of inspired oxygen

P_{atm} is the atmospheric pressure (760 mmHg) at sea level

PH_2O is the water vapor pressure (47 mmHg)

RQ is the respiratory quotient (0.8)

2) Arterial-inspired O₂ ratio or P/F ratio
 PaO_2 / FIO_2