

Interpreting an *ABG* is a crucial skill for health care professionals, and it is especially important in critically ill patients.

This guide will focus on a commonly used seven-step process, which helps ensure a **complete interpretation** of every ABG.

In addition, provided is a list of **commonly encountered** acid-base disorders.

Many guides exist to aid the interpretation of the *ABG*, some may vary in how they have ordered, or combined their steps. We have put this guide together in a method which appears clinically appropriate and logical to us, however a knowledge of other techniques may still be helpful.

7 Step Approach

STEP 1

Review the patient, including taking a brief history and examination

Before thinking about analysing the *ABG*, we need some context about the patient's current clinical status. For instance:

- 1. Are they on **supplementary** oxygen?
- Although the ABG may look normal, if they are on high-flow oxygen, you may expect their P_aO_2 to be much higher.
- 2. Are they hyper- or hypoventilating?
 - A normal P_aCO₂ in someone who is hyperventilating is not normal.
- 3. Are there any **previous** ABG results?
- A chronic problem such as COPD may require a different response to compared to a patient with acute hypoxia.

STEP 2

Is this patient hypoxic?

When first approaching an ABG result, first check the **oxygenation status** of the patient. This is assessed via the P_aO_2 .

This is because **hypoxia** is likely to kill you in **minutes**, where as a acid-base imbalance is more likely to take **hours**.

A good example of this is an **asthma attack**, which presents with a low P_aO_2 , but a relatively normal acid-base balance, however clearly needs **rapid action**.

The normal range for P_aO_2 is: 10 - 14kPa.

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<10kPa = hypoxia
<8kPa = severe hypoxia, and respiratory failure (type 1: P<sub>a</sub>CO<sub>2</sub><6kP<sub>a</sub>; type 2: P<sub>a</sub>CO<sub>2</sub>>6kP<sub>a</sub>)
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NOTE: If the patient is receiving supplemental oxygen, their P_aO_2 , as a general rule, should be approximately 10kPa less than the % inspired concentration / F_iO_2 .

So a patient on 40% oxygen would be expected to have a P_aO₂ of approximately 30kPa.

STEP 3

Is there acidemia or alkalemia present?

pH < 7.35 acidemia

7.35 - 7.45 **normal**

pH > 7.45 alkalemia

Even if the pH is **normal** however, it does not **rule out** a pathology.

NOTE: The term **acidemia/alkalemia** describes the state of low/high blood pH, while **acidosis/alkalosis** is used to describe the processes leading to these states. However, the terms are sometimes used interchangeably clinically.

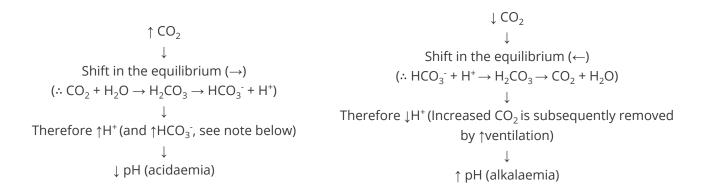
STEP 4

Is there a respiratory component? (pCO₂)

 The respiratory system alters pH by changing how much CO₂ it retains or expels, by changing the bodies ventilation rate.

$$CO_2 + H_2O \rightleftharpoons H_2CO_3 \rightleftharpoons HCO_3^- + H^+$$

Carbon Dioxide + Water ≠ Carbonic Acid ≠ Bicarbonate + Hydrogen Ion



In more simplistic terms, CO_2 forms **carbonic acid** (and then H^+), therefore a **high CO_2** with lead to an **acidemia**, and a **low CO_2**, due to a reduction of carbonic acid (and H^+) will lead to an **alkalemia**.

CO₂ ≈ Acidotic

So to determine if the cause is respiratory in origin, the pH and P_aCO₂ must change in **opposite** directions.

$$\begin{array}{ccc} & pH & P_aCO_2 \\ \text{Respiratory acidosis} & \downarrow & \uparrow \\ \text{Respiratory alkalosis} & \uparrow & \downarrow \\ \end{array}$$

If they change in the same directions (so that an increase in the acidotic P_aCO_2 is associated with alkalotic high pH, it may be a **compensatory** response, explained in step 6)

The normal range for P_aCO_2 is: 4.7 - 6kPa.

NOTE: There are very large quantities of HCO_3^- in the body compared to H^+ , this means that a **large change** in the concentration of H^+ can occur, with only a very **small change** in the concentration of HCO_3^- . So even though the **dissociation** of H_2CO_3 , leads to an equal number of HCO_3^- and H^+ ions being generated, the change in pH may be **significant**, whereas the change in HCO_3^- concentration may be **negligible**.

Is there a metabolic component? (HCO₃-)

• The metabolic system (namely the kidneys) alters pH by retaining or excreting bicarbonate (HCO₃-)

$$CO_2 + H_2O \rightleftharpoons H_2CO_3 \rightleftharpoons HCO_3^- + H^+$$

Carbon Dioxide + Water ≠ Carbonic Acid ≠ Bicarbonate + Hydrogen Ion

$$\uparrow HCO_3^{-1}$$
↓
Shift in the equilibrium (←) (∴ $HCO_3^{-1} + H^+ \rightarrow H_2CO_3 \rightarrow CO_2 + H_2O$)
↓
Therefore $\downarrow H^+$ (as gets converted into $CO_2 + H_2O$ to be 'blown off' through ventilation)
↓
$$\uparrow pH \text{ (alkalaemia)}$$
↓
$$\downarrow HCO_3^{-1}$$
↓
Shift in the equilibrium (→) (∴ $CO_2 + H_2O \rightarrow H_2CO_3 \rightarrow HCO_3^{-1} + H^+$)
↓
Therefore $\uparrow H^+$
↓
$$\downarrow pH \text{ (acidaemia)}$$

 HCO_3^- can be thought to **"mop up"** H^+ , increasing pH, therefore a **high** bicarbonate will lead to an **alkalemia**, and a **low** bicarbonate an **acidemia**.

HCO₃⁻≈ Alkalotic

So to determine if the cause is metabolic in origin, pH and P_aCO₂ must change in the **same** direction.

$$\begin{array}{ccc} & & \text{pH} & \text{HCO}_3^{-1} \\ \text{Metabolic acidosis} & \downarrow & \downarrow \\ \text{Metabolic alkalosis} & \uparrow & \uparrow \end{array}$$

If they change in the opposite directions (so that an increase in the alkalotic HCO_3^- is associated with a acidaemic low pH, it may be a **compensatory** response, explained in step 6)

The normal range for HCO_3^- is: 22 - 26 mmol/L.

Which component is the primary disruption, and is there compensation present?

There may be some cases where both **respiratory** and **metabolic** components are present. In this case, commonly one is the **primary** component, or cause of the disruption, and the other one is a **compensatory** process, which attempts to **correct** the pH.

If the cause of the pH imbalance is from the **respiratory** system, **metabolic** compensation can either **renally excrete or retain HCO₃-**, to balance the pH and bring it back closer to the normal range.

If the cause of the pH imbalance is **metabolic**, the **respiratory** system can compensate by either **retaining or**

expelling ('blowing off') **CO**₂ to balance the pH (via increasing or decreasing **ventilation**).

When coming to approach if compensation is taking place, assess which value is **contradicting** the pH. For instance a high HCO₃⁻ (**alkalotic**) and a low pH (**acidemia**) suggests **metabolic** compensation; a high CO₂ (**acidotic**) and a high pH (**alkalemia**) suggests **respiratory** compensation. (You may also note that the compensation moves in the **same direction** as the primary disruption i.e. high P_aCO₂ and high HCO₃⁻.)

pH P _a CO ₂ HCO ₃	-
Acidosis Respiratory	with metabolic compensation
\downarrow \uparrow \uparrow	
Acidosis Metabolic	with respiratory compensation
\ \	
Alkalosis Respiratory	with metabolic compensation
\uparrow \downarrow \downarrow	
Alkalosis Metabolic	with respiratory compensation
\uparrow \uparrow \uparrow	

Partially compensated = pH still out of normal limits; Fully compensated = pH returned to normal limits

Usually, compensation does not return the pH to normal (7.35 – 7.45), and **over compensation** never occurs.

NOTE: Respiratory compensation can happen over the course of **minutes**; metabolic compensation however takes **days**, therefore if **metabolic** compensation is present, it can be assumed that the **respiratory** disruption has been going on for at least a **few days**.

NOTE: It is possible to have a **mixed** picture in terms of causes i.e. respiratory & metabolic acidosis / respiratory & metabolic alkalosis. In which case both pathologies are leading to a **combined** acidemia or alkalemia, and therefore both causes need **correcting**.

Are there any other deranged values?

Ensure that you look at **all other figures** on the analysis, and **comment** on any abnormalities.