Interpretation of arterial blood gases
Arterial blood gases (ABG) are obtained for two basic purposes:

1. To determine oxygenation
2. To determine acid-base status

When is an ABG required?

1. To establish a diagnosis
2. To assess illness severity
3. To guide and monitor treatment
Arterial blood gas analysis - pulmonary gas exchange: partial pressure

- Pulmonary gas exchange refers to the transfer of O₂ from the atmosphere to the bloodstream (oxygenation) and CO₂ from the bloodstream to the atmosphere (CO₂ elimination).

- Arterial blood gases help us to assess the effectiveness of gas exchange by providing measurements of the partial pressure of O₂ and CO₂ in arterial blood.

- Partial pressure – contribution of one individual gas within a gas mixture. Gases move from areas of higher partial pressure to lower partial pressure. At the alveolar-capillary membrane, air in alveoli has a higher PO₂ and lower PCO₂. O₂ molecules move from alveoli to blood and CO₂ move from blood to alveoli.

**Note**

- PO₂ = partial pressures of O₂
- PaO₂ = partial pressures of O₂ *in arterial blood*
- PCO₂ = partial pressures of CO₂
- PaCO₂ = partial pressures of CO₂ *in arterial blood*
Carbon dioxide elimination:

✓ PaCO₂ is determined by alveolar ventilation and the level of ventilation is adjusted to maintain PaCO₂ within tight limits (ref. range: PaCO₂ = 35-45 mm Hg).

✓ Increased PaCO₂ (hypercapnia) implies reduced alveolar ventilation

✓ Ventilation is regulated by an area in brainstem called respiratory center. This area contains receptors that sense PaCO₂ and connect with the muscles involved in breathing. If PaCO₂ is abnormal, the respiratory center adjusts the rate and depth of breathing accordingly.
Arterial blood gas analysis - pulmonary gas exchange

**Oxygenation:**

✓ Almost all O₂ molecules in blood are bound to Hb. The amount of O₂ in blood depends on the two factors:

1. **Hb concentration** - how much O₂ blood has the capacity to carry.
2. **Saturation of Hb with O₂ (SO₂)** - the percentage of available binding sites on Hb occupied by O₂ molecules.

**Note**

SO₂ – saturation in (any) blood
SaO₂ – saturation in arterial blood (80-100 mm Hg)

With a normal PaO₂ (80-100 mm Hg), Hb is maximally saturated (SaO₂>95%). This means blood has used up its O₂-carrying capacity and any further increase in PaO₂ will not significantly increase arterial O₂ content.
Arterial blood gas analysis – hypoxia, hypoxemia and impaired oxygenation

- The term **hypoxia** is a reduced $O_2$ delivery to tissues.
- The term **hypoxemia** is a reduced $O_2$ content ($PaO_2$) in arterial blood. It may result from impaired oxygenation, low haemoglobin (anaemia) or reduced affinity of haemoglobin for $O_2$ (e.g. carbon monoxide).
- **Impaired oxygenation** refers to hypoxaemia resulting from reduced transfer of $O_2$ from lungs to the bloodstream. It is identified by a low $PaO_2$ (<10.7 kPa; <80 mmHg).
Arterial blood gas analysis: acid-base balance

- pH - measurement of acidity or alkalinity, based on hydrogen (H+) ions present.

- Human blood normally has a pH of 7.35–7.45 (H+ = 35–45 nmol/L) and, therefore, it is slightly alkaline. If blood pH is below the normal range (<7.35), there is an acidaemia. If it is above the normal range (>7.45), there is an alkalaemia.

- An acidosis is any process that lowers blood pH, whereas an alkalosis is any process that raises blood pH.
The following equation is crucial to understanding acid–base balance:

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

First, this equation shows that CO₂, when dissolved in blood, becomes an acid.

The more CO₂ added to blood, the more $H_2CO_3$ (carbonic acid) is produced, which dissociates to release free $H^+$. Second, it predicts that blood pH depends not on the absolute amounts of CO₂ or HCO₃⁻ present but on *the ratio of CO₂ to HCO₃⁻*. Thus, a change in CO₂ will not lead to a change in pH if it is balanced by a change in HCO₃⁻ that preserves the ratio (and vice versa). Because CO₂ is controlled by respiration and HCO₃⁻ by renal excretion, this explains how compensation can prevent changes in blood pH.
Arterial blood gas analysis: maintaining acid-base balance

The respiratory buffer response

- Our lungs are responsible for removing CO₂. PaCO₂, the partial pressure of carbon dioxide in our blood, is determined by alveolar ventilation. If CO₂ production is altered, we adjust our breathing to exhale more or less CO₂, as necessary, to maintain PaCO₂ within normal limits. The bulk of the acid produced by our bodies is in the form of CO₂, so it is our lungs that excrete the vast majority of the acid load.

- CO₂ is carried in the blood to the lungs. In blood CO₂ combines with water to form carbonic acid (H₂CO₃). The more CO₂ is added to blood, the more H₂CO₃ is produced, which dissociates to release H⁺.

\[
\begin{align*}
\text{H₂O} + \text{CO₂} & \leftrightarrow \text{H₂CO₃} \leftrightarrow \text{H⁺} + \text{HCO₃⁻}
\end{align*}
\]

- Activation of the lungs to compensate for an imbalance starts to occur within 1-3 minutes.
Arterial blood gas analysis: acid-base balance

The renal buffer response

\[ H_2O + CO_2 \leftrightarrow H_2CO_3 \leftrightarrow H^+ + HCO_3^- \]

✓ The kidneys are responsible for excreting metabolic acids. They secrete H⁺ ions into urine and reabsorb HCO₃⁻ from urine. HCO₃⁻ is a base (and therefore accepts H⁺ ions), so it reduces the concentration of H⁺ ions in blood. The kidneys can adjust urinary H⁺ and HCO₃⁻ excretion in response to changes in metabolic acid production.

↓ blood pH
kidneys will compensate by retaining HCO3
↑ HCO3 level

↑ blood pH
kidneys will compensate by excreting HCO3
↓ HCO3 level

✓ The system may take hours to days to correct the imbalance.
Disturbances of acid-base balance:

1. Metabolic
2. Respiratory

Ad.1

• Metabolic processes are those that primarily alter the HCO$_3^-$ concentration in the blood. A decrease in serum HCO$_3^-$ (base) leads to a metabolic acidosis, while an increase in serum HCO$_3^-$ leads to a metabolic alkalosis.

Ad.2

• Respiratory processes alter the pH by changing the CO$_2$ levels.
• CO$_2$ is a respiratory acid. CO$_2$ accumulation causes an acid state in the blood (through carbonic acid), and as respirations (respiratory rate and/or tidal volume) increase, the body eliminates more CO$_2$ (acid) and is left with a respiratory alkalosis. A decrease in ventilation leads to retention and increased levels of CO$_2$, and thus a respiratory acidosis.
Steps to arterial blood gas interpretation: uncompensated acid-base balance

**Step 1**
Assess the pH to determine if the blood gas is within normal range (7.35-7.45) or alkalotic (>7.45) or acidotic (<7.35)

**Step 2**
If the blood is alkalotic or acidotic, we need to determine if it is caused primarily by a respiratory or metabolic problem. To do this, access the PaCO2 level. With the respiratory problem, as the pH decreases below 7.35, the PaCO2 should rise. If the pH rises above 7.45, the PaCO2 should decrease. Compare the pH and PaCO2 values. If pH and PaCO2 are moving in the opposite directions, then the problem is primarily respiratory in nature.

**Step 3**
Access the HCO3 value. With the metabolic problem, normally as the pH increases, the HCO3 should also increase. As the pH decreases, the HCO3 should also decrease. Compare the pH and HCO3 values. If pH and HCO3 are moving in the same directions, then the problem is primarily metabolic in nature.

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<th>pH</th>
<th>PaCO2</th>
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Arterial blood gas analysis: maintaining acid-base balance

Compensation

When a patient develops an acid–base imbalance, the body attempts to compensate. Lungs and kidneys are the primary buffer response system in the body. The body tries to overcome either a respiratory or metabolic dysfunction in an attempt to return pH into normal value.

The patient can be

1. uncompensated
   - pH outside normal range
2. partially compensated
3. fully compensated
   - pH within normal range
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<th>pH</th>
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<th>PaCO2</th>
<th>HCO3</th>
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<tr>
<td>Respiratory alkalosis</td>
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Arterial blood gas interpretation: mixed acid – base disturbances

When a primary respiratory disturbance and primary metabolic disturbance occur simultaneously, there is said to be a mixed acid–base disturbance.

If these two processes oppose each other, the pattern will be similar to a compensated acid–base disturbance and the resulting pH derangement will be minimised.

By contrast, if the two processes cause pH to move in the same direction (metabolic acidosis and respiratory acidosis or metabolic alkalosis and respiratory alkalosis), a profound acidaemia or alkalaemia may result.
Lactate - basic principles

Biochemistry of Lactate

✓ In a normal steady state with adequate tissue resources and oxygenation, more cellular energy can be extracted aerobically by means of the citric acid cycle and the electron-transport chain. In this case, cells convert pyruvate to acetyl CoA through oxidative decarboxylation.

Pyruvate + NAD$^+$ + CoA $\rightarrow$ Acetyl CoA + CO$_2$ + NADH

✓ In contrast, when the body experiences inadequate tissue perfusion, it undergoes anaerobic metabolism to create energy. In this case, pyruvate metabolizes to lactate ultimately generating fewer ATPs (2 vs. 36) than through the normal, aerobic mechanism.
Lactic acidosis

- Lactic acidosis is the most common cause of metabolic acidosis in hospitalised patients.
- It is defined by a low HCO₃ in association with plasma lactate concentration > 4mmol/L.
- This can occur due to a problem with local blood supply (e.g. ischemic intestine or limb) or as a generalised failure of tissue oxygenation (e.g. profound hypoxemia, shock, cardiac arrest).
- The extent of lactic acidosis is an indicator of disease severity.
H⁺ (35–45 nmol/L) < 35 = alkalaemia, > 45 = acidaemia

Concentration of free hydrogen ions (H⁺): this is a measure of how acidic or alkaline a solution is.

pH (7.35–7.45) < 7.35 = acidaemia, > 7.45 = alkalaemia

Negative log of the H⁺ concentration; this is a common representation of the H⁺ concentration. Because of the logarithmic nature of the scale, small changes in the pH actually represent large changes in the H⁺ concentration.

Pₒ₂ (> 10.6 kPa or > 80 mmHg in arterial blood on room air)

Partial pressure of O₂ can be thought of as the drive for O₂ molecules to move from one place to another. Pₒ₂ is not a measure of O₂ content but it does determine the extent to which haemoglobin is saturated with O₂. Pₒ₂ refers specifically to the partial pressure of O₂ in arterial blood.

Pₛₒ₂ (4.7–6.0 kPa or 35–45 mmHg in arterial blood)

Partial pressure of CO₂ can be thought of as the drive for CO₂ molecules to move from one place to another and (unlike Pₒ₂) is directly proportional to the amount of CO₂ in blood. Pₛₒ₂ refers specifically to the partial pressure of CO₂ in arterial blood.

Sₒ₂ (> 96% on room air)

O₂ saturation of haemoglobin: the percentage of O₂-binding sites on haemoglobin proteins occupied by O₂ molecules. This is a measure of how much of the blood’s O₂-carrying capacity is being used. Sₒ₂ refers specifically to the O₂ saturation of arterial blood.

HCO₃⁻ act (22–28 mmol/L)

Actual bicarbonate: the plasma bicarbonate concentration calculated from the actual Pₗ₃O₂ and pH measurements in the arterial blood sample. High bicarbonate levels signify a metabolic alkalosis and low levels signify a metabolic acidosis.

HCO₃⁻ std (22–28 mmol/L)

Standard bicarbonate: the plasma bicarbonate concentration calculated from the P₂O₂ and pH measurements in the arterial blood sample after the P₂O₂ in the sample has been corrected to 5.3 kPa (40 mmHg). The authors recommend using this measurement of bicarbonate in ABG analysis.

BE (−2 to +2)

Base excess (BE): a calculation of the amount of base that needs to be added to, or removed from, a sample of blood to achieve a neutral pH, at 37°C, after P₂O₂ has been corrected to 5.3 kPa (40 mmHg). A positive BE indicates that there is more base than normal (metabolic alkalosis) and a negative BE indicates that there is less base than normal (metabolic acidosis).

Lactate (0.4–1.5 mmol/L)

An indirect measure of lactic acid: high levels of lactic acid are usually a sign of tissue hypoxia.

Hb (13–18 g/dL for men, 11.5–16 g/dL for women)

Plasma haemoglobin concentration: this effectively determines the blood’s capacity to carry O₂.

Na (135–145 mmol/L)

Plasma sodium concentration.

K (3.5–5 mmol/L)

Plasma potassium concentration.

Cl (95–105 mmol/L)

Plasma chloride concentration.

iCa (1.0–1.25 mmol/L)

Plasma ionised calcium concentration.

Glucose (3.5–5.5 mmol/L if fasting)

Plasma glucose concentration.