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Abnormal laboratory results

The interpretation of arterial blood gases

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Disorder of acid-base balance | Measurement of respiratory function | Blood gas analysis

Summary

Arterial blood gas analysis is used to measure the pH and the partial pressures of oxygen and carbon dioxide in arterial blood. The investigation is relatively easy to perform and yields information that can guide the management of acute and chronic illnesses.This information

indicates a patient's acid-base balance, the effectiveness of their gas exchange and the state of their ventilatory control. Interpretation of an arterial blood gas result should not be done without considering the clinical findings.The results change as the body compensates for the underlying problem. Factors relating to sampling technique, specimen processing and environment may also influence the results.

Introduction

Arterial blood gas analysis is a common investigation in emergency departments and intensive care units for monitoring patients with acute respiratory failure. It also has some application in general practice, such as assessing the need for domiciliary oxygen therapy in patients with chronic obstructive pulmonary disease. An arterial blood gas result can help in the assessment of a patient's gas exchange, ventilatory control and acid–base balance. However, the investigation does not give a diagnosis and should not be used as a screening test. It is imperative that the results are considered in the context of the patient's symptoms.

While non-invasive monitoring of pulmonary function, such as pulse oximetry, is simple, effective and increasingly widely used, pulse oximetry is no substitute for arterial blood gas analysis. Pulse oximetry is solely a measure of oxygen saturation and gives no indication about blood pH, carbon dioxide or bicarbonate concentrations.

Arterial puncture

Blood is usually withdrawn from the radial artery as it is easy to palpate and has a good collateral supply. The patient's arm is placed palm-up on a flat surface, with the wrist dorsiflexed at 45°. A towel may be placed under the wrist for support. The puncture site should be cleaned with alcohol or iodine, and a local anaesthetic (such as 2% lignocaine) should be infiltrated. Local anaesthetic makes arterial puncture less painful for the patient and does not increase the difficulty of the procedure.[1] The radial artery should be palpated for a pulse, and a pre-heparinised syringe with a 23 or 25 gauge needle should be inserted at an angle just distal to the palpated pulse (Fig. 1). A small quantity of blood is sufficient.

After the puncture, sterile gauze should be placed firmly over the site and direct pressure applied for several minutes to obtain haemostasis. If repeated arterial blood gas analysis is required, it is advisable to use a different site (such as the other radial artery) or insert an arterial line.

To ensure accuracy, it is important to deliver the sample for analysis promptly. If there is any delay in processing the sample, the blood can be stored on ice for approximately 30 minutes with little effect on the accuracy of the results.

Complications of arterial puncture are infrequent. They include prolonged bleeding, infection, thrombosis or arteriospasm.

Fig. 1

Performing an arterial puncture

arterial puncture

Interpreting a blood gas result

The automated analysers measure the pH and the partial pressures of oxygen (PaO_2) and carbon dioxide ($PaCO_2$) in arterial blood. Bicarbonate (HCO_3^-) is also calculated (Box 1). These measurements should be considered with the patient's clinical features (Table 1).

Box 1

Reference ranges for arterial blood gases

pH	7.35 – 7.45	10.6 – 13.3 kPa
PaO_2	80 – 100* mmHg	4.7 – 6.0 kPa
$PaCO_2$	35 – 45 mmHg	
HCO_3^-	22 – 26 mmol/l	

HCO_3^-	22 – 26 mmol/L	
Base excess	–2 – +2 mmol/L	

Reference ranges for venous blood gases

pH	7.32 – 7.43	
PvO_2	25 – 40 mmHg	
$PvCO_2$	41 – 50 mmHg	
HCO_3^-	23 – 27 mmol/L	

* age and altitude dependent (see text)
Kilopascals: to convert pressures to kPa, divide mmHg by 7.5

Table 1

Correlating arterial blood gas results with clinical features

	Metabolic imbalances		Respirat
	Metabolic acidosis	Metabolic alkalosis	Respiratory acidosis
pH	↓	↑	↓
$PaCO_2$	N (uncompensated) ↓ (compensated)	N (uncompensated) ↑ (compensated)	↑
HCO_3^-	↓	↑	N (uncompensate ↑ (compensated

Base excess	↓	↑	N/↑
Clinical features	Kussmaul-type breathing (deeper, faster respiration), shock, coma	Paraesthesia, tetany, weakness	Acute: air hunge disorientation Chronic: hypoventilation, hypoxia, cyanosi
Common causes	With raised anion gap: diabetic ketoacidosis, lactic acidosis, poisons (e.g. ethylene glycol), drug overdoses (paracetamol, aspirin, isoniazid, alcohol) With normal anion gap: diarrhoea, secretory adenomas, ammonium chloride poisoning, interstitial nephritis, renal tubular acidosis, acetazolamide administration	Vomiting, prolonged therapy with potassium-wasting diuretics or steroids, Cushing's disease, ingestion/overdose of sodium bicarbonate (e.g. antacids)	Hypoventilation chronic lung disease with CO_2retention, e.g chronic obstructive pulmonary disease, respiratory depression from drugs (e.g. opioids, sedatives), sever asthma, pulmonary oedema

N = within normal range ↑ = increased ↓ = decreased

pH

The pH determines the presence of acidaemia or alkalaemia. If the body has compensated for the disorder, the pH may be in the normal range.

$PaCO_2$

The $PaCO_2$reflects the state of alveolar ventilation. An elevated $PaCO_2$ reflects alveolar hypoventilation, whereas a decreased

$PaCO_2$ reflects alveolar hyperventilation. Acute changes in $PaCO_2$ will alter the pH. As a general rule, a low pH with a high $PaCO_2$ suggests a respiratory acidosis, while a low pH with a low $PaCO_2$ suggests a metabolic acidosis.

There is a delayed response of $PaCO_2$ to an acute change. Increases in $PaCO_2$ occur relatively slowly, as the body's overall CO_2 stores are very large (approximately 20 L) and the volume of CO_2 generated by metabolism (200 mL/min) makes little overall difference. For instance, during a breath-hold, the $PaCO_2$ rises at a rate of only 2–3 mmHg per minute, hence patients with a very high $PaCO_2$ usually have a long-standing disorder. Accordingly, even when treated the $PaCO_2$ may take a long time to return to normal.

The state of arterial blood oxygenation is determined by the PaO_2. This reflects gas exchange in the lungs and normally the PaO_2 decreases with age. This is due to decreased elastic recoil in the lungs in the elderly, thereby yielding a greater ventilation-perfusion mismatch. The expected PaO_2 when breathing air at sea level can be calculated with the equation $PaO_2 = 100 – (age \times 0.25)$. Consequently, a PaO_2 of 75 mmHg, which may be of concern in a young person, is usually unremarkable in an 85-year-old.

PaO_2

A PaO_2 that is less than expected indicates hypoxaemia. This can result from hypoventilation or a mismatch of ventilation and perfusion. If alveolar ventilation is adequate (that is, $PaCO_2$ is normal), then the hypoxaemia is almost certainly caused by a ventilation-perfusion disturbance. The nature of the hypoxaemia can be further assessed by the difference between the alveolar and arterial oxygen tensions.

The alveolar–arterial oxygen tension difference

If an arterial blood gas result shows hypoxaemia (low PaO_2) and inadequate alveolar ventilation (high $PaCO_2$), it must be determined whether the hypoxaemia is related to hypoventilation, or is secondary to a disturbance in ventilation-perfusion, or both. This is assessed by calculating the difference between the alveolar (PAO_2) and arterial (PaO_2) oxygen tensions (see Box 2).

The alveolar–arterial difference, or gradient, can be estimated only if the oxygen fraction of inspired air (FiO_2, usually 0.21 on room air), barometric pressure and water vapour pressure are known. A normal reference range is 5–15 mmHg. The difference, expressed as $P(A–a)O_2$, increases with age, cigarette smoking and increasing FiO_2. An expected $P(A–a)O_2$ can be calculated using the formula $P(A–a)O_2 = 3 + (0.21 \times \text{patient's age})$.

All causes of hypoxaemia, apart from hypoventilation, increase the alveolar-arterial difference. In a patient breathing room air, a $P(A–a)O_2$ greater than 15 mmHg suggests a ventilation-perfusion mismatch related to disease of the airways, lung parenchyma or pulmonary vasculature. However, the result is non-specific in defining the actual pathology and again the patient's clinical features are essential for diagnosis.

Box 2

The alveolar–arterial oxygen gradient

$P(A\text{-}a)O_2 = PAO_2 – PaO_2$

PaO_2 = arterial oxygen tension

PAO_2 = alveolar oxygen tension

$$PAO_2 = FiO_2(P_B - P_{H2O}) - 1.2(PaCO_2)$$

FiO_2 = oxygen fraction in inspired air

P_B = barometric pressure (760 mmHg at sea level)

P_{H2O} = water vapour tension (47 mmHg at 37° C)

Normal value

Bicarbonate

Bicarbonate is a weak base that is regulated by the kidneys as part of acid–base homeostasis. The HCO_3^- measured in arterial blood reflects the metabolic component of arterial blood. Together, $CO_2$and HCO_3^- act as metabolic and respiratory buffers respectively. They are related via the equation:

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

Compensatory changes

For any disturbance of gas tensions in arterial blood, a compensatory system exists to maintain homeostasis. In a metabolic disorder, where HCO_3^- may be retained or excreted by the kidneys, respiratory compensation can occur almost immediately to alter the rate and depth of ventilation to retain or remove CO_2. This occurs due to the exquisite sensitivity of chemoreceptors in the medulla to carbonic acid (H_2CO_3) or H^+. Renal compensation in response to a respiratory disorder takes much longer, sometimes between three and five days, to retain or remove HCO_3^- as required.

As a general rule, when compensation is present the arterial blood gas result shows two imbalances – derangement of both HCO_3^- and $PaCO_2$. A clue to which imbalance is the primary disturbance is obtained from the pH. If pH is leaning toward acidosis or alkalosis, then the parameter that matches the pH trend (that is, is increased or decreased corresponding to pH) is the primary problem and the other is due to compensation.

The base excess

The metabolic component of the acid–base balance is reflected in the base excess. This is a calculated value derived from blood pH and $PaCO_2$. It is defined as the amount of acid required to restore a litre of blood to its normal pH at a $PaCO_2$ of 40 mmHg. The base excess increases in metabolic alkalosis and decreases (or becomes more negative) in metabolic acidosis, but its utility in interpreting blood gas results is controversial.

While the base excess may give some idea of the metabolic nature of a disorder, it may also confuse the interpretation. The alkalaemia or acidaemia may be primary or secondary to respiratory acidosis or

alkalosis. The base excess does not take into account the appropriateness of the metabolic response for any given disorder, thus limiting its utility when interpreting results.

Anion gap

The anion gap assists with the diagnosis of metabolic acidosis (Box 3). This difference between the concentrations of measured anions and cations increases with dehydration and decreases with hypoalbuminaemia. The gap also widens if there is an increase in the concentration of unmeasured anions such as ketones and lactate.

Box 3
The anion gap concept

- the anion gap is an artificial concept that may indicate the cause of a metabolic acidosis
- it represents the disparity between the major measured plasma cations (sodium and potassium) and the anions (chloride and bicarbonate)
- when calculating the anion gap, potassium is usually omitted from the calculation thus: Gap = $Na^+ + (Cl^- + HCO_3^-)$
- the anion gap is normally between 8 and 16 mmol/L
- a raised anion gap indicates an increased concentration of lactate, ketones or renal acids and is seen in starvation and uraemia
- a raised anion gap is seen in overdoses of paracetamol, salicylates, methanol or ethylene glycol
- a normal anion gap is seen if a metabolic acidosis is due to diarrhoea or urinary loss of bicarbonate

Factors influencing blood gas results

A number of sampling and environmental factors may affect the result of the analysis. Delayed processing of the sample may yield a falsely low PaO_2 as the delay allows leucocytes to consume oxygen. This can be

PaO_2, as the delay allows leucocytes to consume oxygen. This can be avoided by prompt transport of the sample on ice.

Air bubbles introduced when performing the arterial puncture can also cause a falsely high PaO_2 and a falsely low $PaCO_2$.[2] This can be avoided by gently removing air bubbles within the specimen immediately after collection without agitating the sample.

Body temperature can also affect arterial blood gas tensions. This is relevant in febrile or hypothermic patients, so body temperature should be recorded at the time of collection.[3]

Mixed acid–base disorders

It is possible to have a mixed respiratory and metabolic disorder that makes interpretation of an arterial blood gas result difficult. As a general rule, when a normal pH is accompanied by an abnormal $PaCO_2$ or HCO_3^- then a mixed metabolic-respiratory disorder exists. Table 2 provides some common clinical examples of mixed respiratory and metabolic disturbances, and Fig. 2 and Fig. 3 are algorithms for the consideration of primary and mixed acid–base disorders.[4]

Table 2

Examples of mixed acid–base disorders

Mixed metabolic/respiratory disturbance	Example
Respiratory acidosis and metabolic acidosis	A patient with acute pulmonary oedema after an acute myocardial infarct Mechanism: poor cardiac circulation (causing a lactic acidosis – metabolic acidosis) with concurrent poor alveolar ventilation (due to pulmonary oedema) – causing CO_2 retention and a concomitant respiratory acidosis
Respiratory	A patient with hepatic cirrhosis who is given

alkalosis and metabolic alkalosis	diuretics Mechanism: patients with hepatic cirrhosis can experience the phenomenon of the hepatopulmonary syndrome where the major symptom is dyspnoea (causing a respiratory alkalosis), while diuretics can cause a decrease in blood volume, which stimulates the renin-angiotensin-aldosterone system, increasing the exchange between Na^+ and K^+ or H^+ at the distal tubule, resulting in an increase in bicarbonate concentration and a metabolic alkalosis
Respiratory acidosis and metabolic alkalosis	A patient with long-standing chronic obstructive pulmonary disease who is given diuretics for concomitant heart failure Mechanism: long-standing air flow limitation may cause chronic hypercapnia and respiratory acidosis via impaired CO_2excretion, while diuretics can cause a decrease in blood volume, which stimulates the renin-angiotensin-aldosterone system, increasing the exchange between Na^+ and K^+ or H^+ at the distal tubule, resulting in an increase in bicarbonate concentration and a metabolic alkalosis
Respiratory alkalosis and metabolic acidosis	A patient with chronic renal failure who begins to hyperventilate secondary to anxiety Mechanism: chronic renal failure causes a metabolic acidosis by uraemia and failure to excrete acids while the respiratory alkalosis results from blowing off excess CO_2due to alveolar hyperventilation
Metabolic acidosis and metabolic alkalosis	A patient with chronic renal failure who suffers from severe intractable vomiting Mechanism: chronic renal failure causes a metabolic acidosis by uraemia and failure to excrete acids while a concurrent metabolic alkalosis results from the depletion in the body stores of H^+ and Cl^2 through vomiting

Fig. 2

Interpreting acidaemia on an arterial blood gas result

Image adapted from reference 4 with permission

Fig. 3

Interpreting alkalaemia on an arterial blood gas result

Image adapted from reference 4 with permission

Limitations of blood gas analysis

The blood gas analysis cannot yield a specific diagnosis. A patient with asthma may have similar values to another patient with pneumonia. Alternatively, a patient with chronic obstructive pulmonary disease and respiratory failure may have similar results to a patient with pulmonary oedema.

The analysis does not reflect the degree to which an abnormality actually affects a patient. A low PaO_2 does not necessarily indicate tissue hypoxia, nor does a normal PaO_2 indicate adequate tissue oxygenation. Oxygen utilisation is influenced by other factors such as regional blood flow, haemoglobin affinity for oxygen and cardiac output.

Blood gas analysis cannot be used as a screening test for early pulmonary disease. Severe disease may be present before significant changes are seen in blood gases.

Venous blood gases

It is easier to obtain a venous sample than an arterial sample. In some situations analysis of venous blood can provide enough information to assist in clinical decisions. In general, the pH, $CO_2$and HCO_3^- values are similar in venous and arterial blood (Box 1). The main difference is the partial pressure of oxygen in venous blood is less than half that of arterial blood. Venous blood should not therefore be used to assess oxygenation.

Conclusion

Measuring arterial blood gases can be a useful adjunct to the assessment of patients with either acute or chronic diseases. The results show if the patient is acidaemic or alkalaemic and whether the cause is likely to have a respiratory or metabolic component. The $PaCO_2$ reflects alveolar ventilation and the PaO_2 reflects the oxygenation of arterial blood. When combined with a patient's clinical features, blood gas analysis can facilitate diagnosis and management.

Conflict of interest: none declared

Further Reading

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