# The interpretation of arterial blood gases

***Abhishek K Verma**, Resident, General Medicine, and **Paul Roach**, Consultant Respiratory and Sleep Medicine Physician, Gosford Hospital, New South Wales*

## 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.**

Key words: acid–base disorders, pulmonary function tests.

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## 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Blood is usually withdrawn from the radial artery as it is easy to palpate and has a good collateral supply.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**

## 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).

### *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.

*Box 1*

**Reference ranges for arterial blood gases**

| | | |
|---|---|---|
| pH | 7.35 – 7.45 | |
| $PaO_2$ | 80 – 100* mmHg | 10.6 – 13.3 kPa |
| $PaCO_2$ | 35 – 45 mmHg | 4.7 – 6.0 kPa |
| $HCO_3^-$ | 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 | | Respiratory imbalances | |
|---|---|---|---|---|
| | **Metabolic acidosis** | **Metabolic alkalosis** | **Respiratory acidosis** | **Respiratory alkalosis** |
| pH | ↓ | ↑ | ↓ | ↑ |
| $PaCO_2$ | N (uncompensated)<br>↓ (compensated) | N (uncompensated)<br>↑ (compensated) | ↑ | ↓ |
| $HCO_3^-$ | ↓ | ↑ | N (uncompensated)<br>↑ (compensated) | N (uncompensated)<br>↓ (compensated) |
| Base excess | ↓ | ↑ | N / ↑ | N / ↓ |
| Clinical features | Kussmaul-type breathing (deeper, faster respiration), shock, coma | Paraesthesia, tetany, weakness | Acute: air hunger, disorientation<br>Chronic: hypoventilation, hypoxia, cyanosis | Acute: hyperventilation, paraesthesia, light-headedness<br>Chronic: hyperventilation, latent tetany |
| Common causes | With raised anion gap: diabetic ketoacidosis, lactic acidosis, poisons (e.g. ethylene glycol), drug overdoses (paracetamol, aspirin, isoniazid, alcohol)<br>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 $CO_2$ retention, e.g. chronic obstructive pulmonary disease, respiratory depression from drugs (e.g. opioids, sedatives), severe asthma, pulmonary oedema | Hyperventilation – anxiety, pain, febrile illness, hypoxia, pulmonary embolism, pregnancy, sepsis |

N = within normal range ↑ = increased ↓ = decreased

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.

## $PaO_2$

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.

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**

$P(A–a)O_2 = PAO_2 – PaO_2$

$PaO_2$ = 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 <15 mmHg

## 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.

## 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 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]

*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

### *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

*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<br>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 alkalosis and metabolic alkalosis | A patient with hepatic cirrhosis who is given diuretics<br>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<br>Mechanism: long-standing air flow limitation may cause chronic hypercapnia and respiratory acidosis via impaired $CO_2$ excretion, 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<br>Mechanism: chronic renal failure causes a metabolic acidosis by uraemia and failure to excrete acids while the respiratory alkalosis results from blowing off excess $CO_2$ due to alveolar hyperventilation |
| Metabolic acidosis and metabolic alkalosis | A patient with chronic renal failure who suffers from severe intractable vomiting<br>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 $Cl^-$ through vomiting |

*Fig. 2*

**Interpreting acidaemia on an arterial blood gas result**

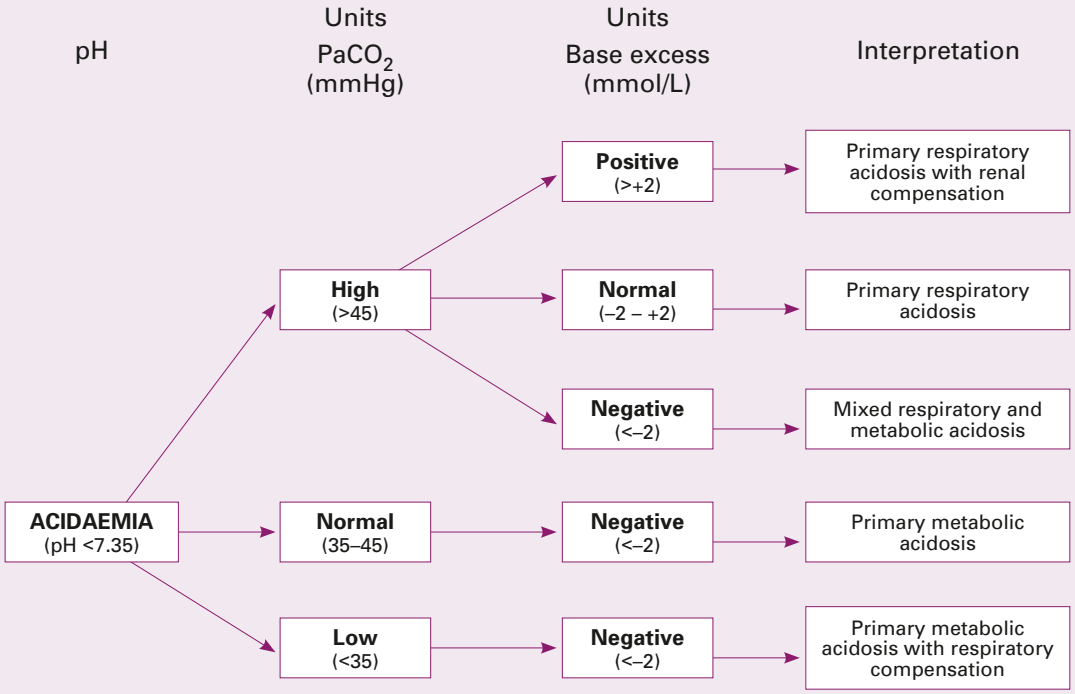


Image adapted from reference 4 with permission

*Fig. 3*

**Interpreting alkalaemia on an arterial blood gas result**

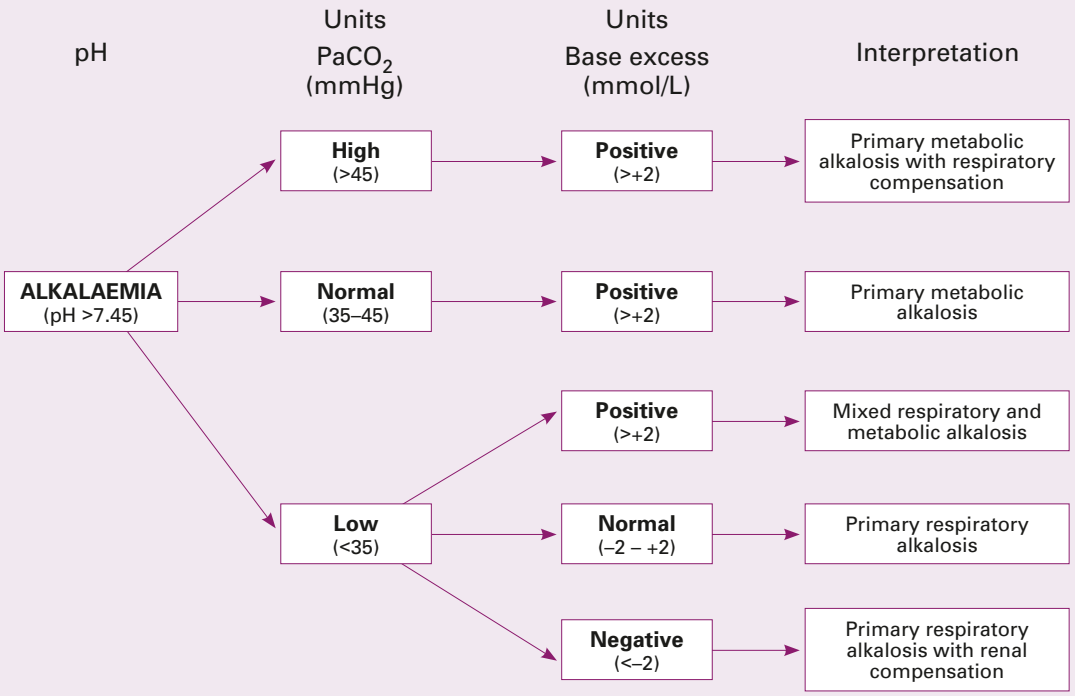


Image adapted from reference 4 with permission

Fig. 2 and Fig. 3 are algorithms for the consideration of primary and mixed acid–base disorders.[4]

## 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.

## References


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## Further reading


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*Conflict of interest: none declared*

### Self-test questions

*The following statements are either true or false (answers on page 131)*

7. The partial pressure of carbon dioxide in arterial blood ($PaCO_2$) is inversely related to alveolar ventilation.
8. The partial pressure of oxygen in arterial blood ($PaO_2$) reflects the gas exchange function of the lungs.

# New drugs

Some of the views expressed in the following notes on newly approved products should be regarded as tentative, as there may be limited published data and little experience in Australia of their safety or efficacy. However, the Editorial Executive Committee believes that comments made in good faith at an early stage may still be of value. As a result of fuller experience, initial comments may need to be modified. The Committee is prepared to do this. Before new drugs are prescribed, the Committee believes it is important that full information is obtained either from the manufacturer's approved product information, a drug information centre or some other appropriate source.

## Certolizumab

Cimzia (UCB)

pre-filled syringe containing 200 mg in 1 mL of liquid

Approved indication: rheumatoid arthritis

Australian Medicines Handbook section 15.2.1

Certolizumab, like adalimumab, etanercept and infliximab, is a tumour necrosis factor inhibitor indicated for rheumatoid arthritis. It is a recombinant humanised antibody Fab' fragment which has been pegylated to extend its plasma half-life to that of the whole antibody.

Peak plasma concentrations are reached between 54 and 171 hours after subcutaneous administration and its bioavailability is approximately 80%. The terminal elimination half-life is around 14 days. However, the presence of antibodies to certolizumab increases its clearance and appears to correlate with reduced patient responses. Giving methotrexate concomitantly with certolizumab reduces the formation of anti-certolizumab antibodies.