Blood Gas Analysis and Pulse Oximetry

CHAPTER 15

Learning outcomes

To understand:

- **The terms used to describe the results of arterial blood gas analysis**
- **How respiration and metabolism are linked**
- **How to use the 5-step approach to analyse arterial blood gas results**
- **The principles of pulse oximetry**
- **The safe and effective use of oxygen**

Introduction

Interpreting the analysis of an arterial blood sample to determine a patient's acid-base status and respiratory gas exchange is a key component in the management of any ill patient and, in particular, in the peri-arrest situation. Although there is often a great temptation to try and analyse the numerical data in isolation, it is essential to have a system to ensure that nothing is overlooked or misinterpreted; as when reading an ECG, this starts with asking "how is the patient?" This should include any known history along with details of current oxygen therapy and medications.

There are usually four key pieces of information contained in the results of analysis of an arterial blood sample:

- pH
- $PaCO_2$ (partial pressure of carbon dioxide in arterial blood)
- Bicarbonate and base excess
- PaO_2 (partial pressure of oxygen in arterial blood)

In order to interpret these results, we first need to understand what each means. Normal ranges are given in the text; however, these will vary slightly between institutions.

pH

The acidity or alkalinity of the blood (or any solution) is determined by the concentration of hydrogen ions $[H^+]$; the greater the concentration, the more acid the solution. In the body, the concentration of hydrogen ions is extremely low, normally around 40 nanomoles per litre (nmol l^{-1}), where a nanomole is 1 billionth of a mole (a mole is the molecular weight of a substance in grams, i.e. for hydrogen it would be 2 g). To put this into perspective, sodium ions (Na^+) are present in a concentration of 135 millimoles per litre (mmol l^{-1}), i.e. 3 million times greater. In order to make dealing with such small numbers easier, we use the pH scale; this is a logarithmic scale expressing the hydrogen ion concentration between 1 and 14. The pH of a normal arterial blood sample lies between 7.35 and 7.45, or $[H^+]$ 44 - 36 nmol l^{-1}. There are two key points to remember about the pH scale:

1. The numerical value of pH changes inversely with hydrogen ion concentration. Consequently a **decrease in blood pH below 7.35** indicates an increase in $[H^+]$ above normal, a condition referred to as an **acidaemia**. Conversely, an **increase in blood pH above 7.45** indicates a reduction in $[H^+]$ below normal, a condition referred to as an **alkalaemia**. Clinicians often use the terms acidosis and alkalosis respectively to describe these situations. Strictly speaking, these terms refer to the processes that lead to the changes in pH, and it is in this context that they will be used in this manual.

2. Small changes in pH represent big changes in hydrogen ion $[H^+]$ concentration. For example, a pH change from 7.4 to 7.1 means that the hydrogen ion concentration has increased from 40 nmol l^{-1} to 80 nmol l^{-1}, i.e. it has doubled for a pH change of 0.3.

Many of the complex reactions within cells are controlled by enzymes that function only within a very narrow pH range; hence, normal pH is controlled tightly between 7.35 and 7.45. However, each day during normal activity we produce massive amounts of hydrogen ions (approximately 14 500 000 000 nmol), which if unchecked would cause a substantial decrease in pH (acidaemia) before they could be excreted. To prevent this happening the body has a series of substances known as buffers that take up hydrogen ions and thereby prevent the development of an acidaemia. The major intracellular buffers are proteins, phosphate and haemoglobin (within red blood cells) and the extracellular buffers are plasma proteins and bicarbonate (see below).

Clearly the buffering system is only a temporary solution to the production of acids; ultimately they will all be consumed and acids will start to accumulate. A system is therefore required to eliminate the acids and thereby regenerate the buffers. This is achieved by the lungs and kidneys.

Partial pressure

We normally use percentages to describe the composition of a mixture of gases, a good example being air: 21% oxygen, 78% nitrogen, 0.04% carbon dioxide. However, a better indication of the number of molecules of a gas in a mixture is better described by referring to its partial pressure. The partial pressure is the contribution each gas in a mixture makes to the total pressure. The importance of using this measure is best demonstrated by the fact that if we double the total pressure of a mixture, the partial pressures of the constituents are doubled, but the percentages remain the same. We breathe gases at atmospheric pressure or 1 atmosphere, very close to a pressure of 100 kiloPascals (kPa) or 750 mmHg (1 kPa = 7.5 mmHg). As a result, when breathing air, the contribution (partial pressure) of nitrogen is 78% of 100 kPa or 78 kPa and oxygen 21% of 100 kPa or 21 kPa. When breathing 40% oxygen, the partial pressure of the oxygen in the inspired gas is 40 kPa.

At atmospheric pressure, the partial pressure of a gas in a mixture (in kPa) is numerically the same as the percentage (%) of the gas by volume.

When a gas is dissolved in a liquid (e.g. blood) the partial pressure within the liquid is the same as in the gas in contact with the liquid. This enables us to measure the partial pressure of oxygen and carbon dioxide in blood.

In summary, the partial pressure of a gas is a measure of the concentration of the gas in the medium it is in and is expressed as $P_{medium}Gas$, e.g. $PaCO_2$ is the partial pressure (P) of carbon dioxide (CO_2) in arterial blood (a).

$PaCO_2$

Carbon dioxide (CO_2) is an important waste product of metabolism. Under normal circumstances, it is transported in the blood to the lungs where it is excreted during expiration. For transport to the lungs, it is either combined with protein or haemoglobin, or is dissolved in plasma where it reacts with water to form hydrogen ions and bicarbonate (HCO_3^-):

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

The normal $PaCO_2$ is 5.3 kPa with a range of 4.7 - 6.0 kPa.

In the lungs, the reaction proceeds in reverse: CO_2 is generated and expired. From this reaction, we can see that CO_2 behaves as an acid: any increase in $PaCO_2$ will cause the reaction to move to the right and increase the hydrogen ion concentration with the subsequent development of an acidaemia. There will, of course, be the same increase in bicarbonate concentration but, as this is only nanomoles, it has little effect on the overall total concentration of 22 - 26 mmol l^{-1}. If the metabolic production of CO_2 is constant, the only factor that affects the amount in the blood is the rate at which it is removed by alveolar ventilation. A **decrease in alveolar ventilation** will reduce excretion of CO_2 causing an increase in $PaCO_2$ and the production of more hydrogen ions. If the pH decreases below 7.35 an acidaemia has been produced. As the primary cause of the acidaemia is a problem with the respiratory system, we call this process a **respiratory acidosis**.

Conversely, an **increase in alveolar ventilation** that removes CO_2 faster than it is generated reduces $PaCO_2$ and moves the reaction to the left, reducing the concentration of hydrogen ions. As a result the pH will increase and if it exceeds 7.45 an alkalaemia has been produced. Again, the primary cause is the respiratory system and we call this process a **respiratory alkalosis**.

It is easy to understand therefore how even brief periods of apnoea, as occurs during cardiac arrest, result in a respiratory acidosis. However, under normal circumstances, the respiratory centre in the brain stem is very sensitive to blood [H^+] and within a few minutes rapidly increases alveolar ventilation. This increases CO_2 excretion, reduces $PaCO_2$, decreases [H^+] and returns pH to normal.

The lungs are the primary mechanism by which [H^+] is adjusted by regulating $PaCO_2$.

Bicarbonate and base excess

Bicarbonate

Bicarbonate (HCO_3^-) is the most important buffer. It is generated by the kidneys and is measured easily in an arterial blood sample. It can be thought of as the opposite of an acid and as such is also called a base. When bicarbonate buffers hydrogen ions, carbon dioxide and water are produced, and it is via this route that the vast majority of acids (90%) are excreted each day. However, the acids not eliminated by the respiratory system can also be buffered as shown below. The reaction below moves to the right and bicarbonate neutralises the effect of the H^+ and prevents a decrease in plasma pH. In the kidneys, the reaction proceeds to the left, the H^+ is excreted in the urine and bicarbonate filtered and returned to the plasma. Depending on the acid load, the kidneys will excrete either acid or alkaline urine.

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

Under normal circumstances, the concentration of bicarbonate is 22 - 26 mmol l^{-1}.

If there is an acute increase in the acid load, although the respiratory system will try and increase excretion of carbon dioxide, bicarbonate will decrease as it buffers the extra H^+. Once the reserves of bicarbonate are used, H^+ will accumulate decreasing the pH. Unlike the respiratory system, the kidneys respond slowly and it takes several

days for additional bicarbonate to be produced to meet the demand to buffer the extra acid. If the kidneys fail to produce sufficient bicarbonate the resultant **metabolic acidosis** will lead to a decrease in pH below 7.35 (acidaemia).

Occasionally, there is an excess of bicarbonate. This will have the effect of excessive buffering of hydrogen ions and will produce a **metabolic alkalosis** and increase the pH above 7.45 (alkalaemia).

Base excess

This is a measure of the amount of excess acid or base is in the blood as a result of a metabolic derangement. It is calculated as the amount of strong acid or base that would have to be added to a blood sample with an abnormal pH to restore it to normal (pH 7.4). Consequently, a patient with a **base excess** of 8 mmol l^{-1} would require 8 mmol l^{-1} of **strong acid** to return their pH to normal; that is they have a metabolic alkalosis (compare with bicarbonate which would be raised, so both move in the same direction). Conversely, a patient with a **base deficit** of 8 mmol l^{-1} will require the addition of 8 mmol l^{-1} of **strong base** to normalise their pH (again, compare with bicarbonate which would be reduced). Unfortunately, the term "negative base excess" is used instead of base deficit and in the example above, the patient would have a negative base excess of -8 mmol l^{-1}.

As a result, the normal values of base excess are +2 to -2 mmol l^{-1}.

A base excess more negative than -2 mmol l^{-1} indicates a metabolic acidosis.

A base excess greater than +2 mmol l^{-1} indicates a metabolic alkalosis.

The respiratory - metabolic link

From the above we can see that the body has two systems for ensuring a stable internal environment and preventing the development of an acidosis. Additional protection is provided by the fact that the two systems are linked and can compensate for derangements in each other. This link is provided by the presence of carbonic acid (H_2CO_3), which is dependent on the presence of an enzyme called carbonic anhydrase, present in both red blood cells and the kidneys, and ideally situated to facilitate the link between the two systems.

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

Although this link exists, the ability of each system to compensate for the other is not instantaneous, but becomes more marked when the initial disturbance in one system is prolonged. A typical example demonstrating the link between the two systems is a patient with chronic obstructive pulmonary disease (COPD). This condition results in diminished capacity to excrete carbon dioxide and a respiratory acidosis. If left uncompensated, this would result in a persistent acidaemia, but the increase in carbon dioxide drives the reaction above to the right, with the production of carbonic acid (H_2CO_3). In the kidneys this has the effect of increasing H^+ ions which are excreted in the urine while at the same time increasing bicarbonate production to buffer the H^+ ions in the plasma. As a result the patient has a respiratory acidosis (increased $PaCO_2$) with a compensatory metabolic alkalosis (increased bicarbonate) and the pH will return close to normal.

A different example is a diabetic in ketoacidosis (strictly speaking ketoacidaemia). When the excess ketoacids exceed the kidney's ability for excretion, they are buffered, which consumes plasma bicarbonate. Increasing bicarbonate production takes several days. However, the reaction above can also move to the left by increasing ventilation and reducing $PaCO_2$; in effect, converting the H^+ to CO_2. Consequently, the patient has a metabolic acidosis (reduced bicarbonate) with a compensatory respiratory alkalosis (reduced $PaCO_2$).

PaO_2

The concentration of oxygen in inspired air is 21% - representing a partial pressure of 21 kPa. This is gradually reduced as the air passes down the respiratory tract, firstly because of the addition of water vapour and, in the alveoli, by the addition of carbon dioxide so that here it is normally around 13 kPa. However, the partial pressure of oxygen in arterial blood (PaO_2) is always lower than alveolar; the extent of this gradient is determined largely by the presence of any lung disease. In a healthy individual breathing air, the PaO_2 is normally higher than 11 kPa i.e. about 10 kPa lower than the inspired partial pressure. This can be used as a rule of thumb to estimate the PaO_2 for any given inspired concentration, in that it should be numerically about 10 less than the inspired concentration (%). For example, 40% inspired oxygen should result in a PaO_2 of approximately 30 kPa. With increasing lung injury, the gap between inspired concentration and PaO_2 increases. This is important to recognise because for someone breathing 50% oxygen a PaO_2 of 13 kPa is not 'normal'.

Interestingly, PaO_2 also decreases slightly with age, reaching 10 kPa at around 75 years, but then climbs again and plateaus at around 11 kPa at 85 years.

Interpreting the results

Interpretation of the result of blood gas analysis is achieved best by following strictly five steps. For clarity, only changes in base excess are discussed; however, bicarbonate will also change numerically in the same direction.

Step 1

How is the patient? This will often provide useful clues to help with interpretation of the results. For example, one might reasonably predict that analysis of arterial blood shortly after successful resuscitation would show signs of a respiratory acidosis caused by a period of inadequate ventilation and a metabolic acidosis due to the period of anaerobic respiration during the arrest producing lactic acid. Consequently, we would expect the patient to have a very low pH with changes in both $PaCO_2$ and base excess. A patient with a well-compensated, chronic condition will usually display clues about the primary cause and secondary compensation. Without the clinical history, the results of a blood gas analysis from a patient with COPD could be misinterpreted as a primary metabolic alkalosis with a compensatory respiratory acidosis.

Step 2

Is the patient hypoxaemic?
The PaO_2 while breathing room air should be 10.0 - 13.0 kPa. However, if the patient is receiving supplemental oxygen, the PaO_2 must be interpreted in light of the inspired oxygen concentration. As discussed above, the inspired partial pressure of oxygen can be regarded as the numerical equivalent of the inspired concentration (%). If there is a difference of greater than 10 between the two values, there is a defect in oxygenation, proportional to the magnitude of the difference.

Step 3

Is the patient acidaemic (pH < 7.35) or alkalaemic (pH> 7.45)? If the pH is within or very close to the normal range then this suggests normality or a chronic condition with full compensation. In principle, the body never overcompensates and this should enable the primary problem to be determined. If necessary, seek more clinical information about the patient.

Step 4

What has happened to the $PaCO_2$? In other words, is the abnormality wholly or partially due to a defect in the respiratory system?

If the pH is <7.35 (acidaemia):
4a. Is the $PaCO_2$ increased (>6.0 kPa)?
If so, there is a **respiratory acidosis** that may be accounting for all or part of the derangement. There could also be a metabolic component, see Step 5a.

If the pH is > 7.45 (alkalaemia):
4b. Is the $PaCO_2$ reduced (<4.7 kPa)?
If so, there is a **respiratory alkalosis**, but this is an unusual isolated finding in a patient breathing spontaneously, with a normal respiratory rate. It is seen more often in patients who are being mechanically ventilated with excessively high rates and/or tidal volumes. As a result, $PaCO_2$ decreases, there is a reduction in H^+ and an alkalosis develops.

Step 5

What has happened to the base excess or bicarbonate? In other words, is the abnormality wholly or partially due to a defect in the metabolic system?

If the pH is <7.35 (acidaemia):
5a. Is the base excess reduced (more negative than minus 2 mmol l^{-1}), and /or the bicarbonate reduced (<22 mmol l^{-1})? If so, there is **a metabolic acidosis** accounting for all or part of the derangement. There could be a respiratory component if the $PaCO_2$ is also increased - see Step 4a, a situation commonly seen after a cardiac arrest.

If the pH is > 7.45 (alkalaemia):
5b. Is the base excess increased (> +2 mmol l^{-1}) and/or the bicarbonate increased (>26 mmol l^{-1})?
If so, there is a **metabolic alkalosis** accounting for all or part of the derangement. There could be a respiratory component if the $PaCO_2$ is also decreased - see Step 4b, but this would be very unusual.

Example cases

Using the above, work through cases 1 - 3 at the end of this chapter. These are based on clinical cases to highlight key points.

Derangements of both $PaCO_2$ and base excess or bicarbonate - compensation

In addition to the combined changes seen in case 3, the results may show changes in both the respiratory and metabolic components, but with minimal disturbance of the pH. This is the result of compensation; both the respiratory and metabolic systems are capable of reacting to changes in the other, the aim being to minimise long term changes in pH. Four examples follow:

Example 1
pH <7.40, with a increased $PaCO_2$ (> 6.0 kPa) and increased base excess (>+2 mmol l^{-1}) or bicarbonate (>26 mmol l^{-1}).

The tendency towards an acidaemia indicates that this is the primary problem and the increased $PaCO_2$ indicates that it is a **respiratory acidosis**. The increased base excess/bicarbonate represents a **compensatory metabolic alkalosis,** bringing the pH back towards normality.

Example 2
pH <7.40, with a decreased base excess (<-2 mmol l^{-1}) or bicarbonate (<22 mmol l^{-1}) and decreased $PaCO_2$ (<4.7 kPa).

The tendency towards an acidaemia indicates that this is the primary problem and the decreased base excess/bicarbonate suggests that it is a **metabolic acidosis**. The decrease in $PaCO_2$ represents a **compensatory respiratory alkalosis,** bringing the pH back towards normality.

Example 3
pH > 7.40, with increased base excess (>+2 mmol l^{-1}) or bicarbonate (>26 mmol l^{-1}) and increased $PaCO_2$ (>6.0 kPa).

The tendency towards an alkalaemia indicates that this is the primary problem and the increase in base excess/bicarbonate suggests that it is primarily a **metabolic alkalosis**. The increased $PaCO_2$ is **respiratory compensation** bringing the pH back towards normality. This picture may be seen where there is loss of acid from the body e.g. prolonged vomiting of gastric contents (hydrochloric acid) and also occurs in chronic hypokalaemia. In this case, the body compensates by moving potassium from intracellular to extracellular in exchange for hydrogen ions. The pH increases and CO_2 is retained to try and compensate.

Example 4
pH > 7.40, with a decreased $PaCO_2$ (<4.7 kPa) and decreased base excess (<-2 mmol l^{-1}) or bicarbonate (<22 mmol l^{-1}).

The tendency towards an alkalaemia indicates that this is the primary problem and the decrease in $PaCO_2$ suggests that this is primarily a **respiratory alkalosis**. The decrease in base excess/bicarbonate is the **metabolic compensation** bringing the pH back towards normality. This is not a common finding, but may be seen after a few days when hyperventilation is used to help control intracranial pressure in patients with brain injury.

Using the above, work through cases 4 and 5 at the end of this chapter.

There is one final situation that deserves mention and is important to identify: an ill patient with a pH <7.4, a normal $PaCO_2$ (4 - 6.0 kPa) and a decreased base excess (<-2 mmol l^{-1}) or bicarbonate (< 22 mmol l^{-1}).

This is most likely to represent the situation of a metabolic acidosis in a patient with chronic carbon dioxide retention. The patient is trying to compensate by lowering their carbon dioxide (to cause a compensatory respiratory alkalosis), but they are starting from a higher $PaCO_2$. Their lung disease will limit the amount of CO_2 they can excrete, thereby preventing it decreasing any further. Once again it illustrates the importance of having information about the patient as identified at the beginning.

Practical aspects of blood gas analysis during resuscitation

During cardiac arrest, arterial blood gas values are of limited use because they correlate poorly with the severity of hypoxaemia, hypercarbia and acidosis in the tissues. Indeed, during cardiac arrest, venous blood gases may reflect more accurately the acid-base state of the tissues. These are interpreted using the same 5-step approach, however, the normal range of values will be different to arterial blood and they should be interpreted cautiously.

Once return of spontaneous circulation (ROSC) is achieved, arterial blood gas analysis will provide a useful guide to post cardiac arrest treatment, such as the optimal fractional inspired oxygen (FiO_2) and minute ventilation. Arterial lactate concentration can also be used to indicate adequacy of tissue oxygenation, normal arterial blood lactate concentration being 0.7 - 1.8 mmol l^{-1}. Immediately after cardiac arrest, concentrations are high, reflecting the lactic acidosis that has been caused by inadequate oxygenation of the tissues during the period of cardiac arrest. After ROSC a progressively decreasing lactate value is another indicator of adequate tissue oxygenation.

In the peri-arrest setting, it may be easiest to obtain a sample of arterial blood (into a heparinised syringe) from the femoral artery. The radial artery may be preferable once the patient has an adequate cardiac output and blood pressure. The radial artery is also the best site for insertion of an arterial cannula; this enables continuous monitoring of blood pressure and frequent blood sampling in the post cardiac arrest period.

Pulse oximetry

Role

Pulse oximetry is a vital adjunct to the assessment of hypoxaemia. Clinical recognition of decreased arterial oxygen saturation of haemoglobin (SaO_2) is subjective and unreliable, with the classic sign of cyanosis appearing late when arterial oxygen saturation is between 80 - 85%. Pulse oximetry is simple to use, relatively cheap, non-invasive and provides an immediate, objective measure of arterial blood oxygen saturation. It is now used widely in all in-hospital settings and increasingly in both primary care and the prehospital environment. Oxygen saturation, 'the fifth vital sign', now also forms a component of many early warning systems to identify the deteriorating patient.

Principles

The pulse oximeter probe containing light-emitting diodes (LEDs) and a photoreceptor situated opposite, is placed across tissue, usually a finger or earlobe. Some of the light is transmitted through the tissues while some is absorbed. The ratio of transmitted to absorbed light is used to generate the peripheral arterial oxygen saturation (SpO_2) displayed as a digital reading, waveform, or both. As a result of rapid sampling of the light signal, the displayed reading will alter every 0.5 - 1 s, displaying the average SpO_2 over the preceding 5 - 10 s. Most pulse oximeters are accurate to within +/- 2% above an SaO_2 of 90%.

Tissue thickness should be optimally between 5 - 10 mm. Poor readings may be improved by trying different sites, warming sites or applying local vasodilators.

Pulse oximeters often provide an audible tone related to the SpO_2, with a decreasing tone reflecting increasing degrees of hypoxaemia. Information about pulse rate and waveform (plethysmographic waveform) may also be provided. A poor signal may indicate a low blood pressure or poor tissue perfusion - reassess the patient.

Pulse oximetry provides only a measure of oxygen saturation, not content, and thus gives no indication of actual tissue oxygenation. Furthermore, it provides no information on the partial pressure of carbon dioxide in the body ($PaCO_2$) and is not a measure of adequacy of ventilation. In cases of critical illness, or when type II respiratory failure (see below) is suspected (e.g. known COPD, congenital heart disease) arterial blood gas sampling must be performed. Readings from a pulse oximeter must not be used in isolation: it is vital to interpret them in light of the clinical picture and alongside other investigations, and potential sources of error.

Limitations

The relationship between oxygen saturation and arterial oxygen partial pressure (PaO_2) is demonstrated by the oxyhaemoglobin dissociation curve (Figure 15.1). The sinusoid shape of the curve means that an initial decrease from a normal PaO_2 is not accompanied by a drop of similar magnitude in the oxygen saturation of the blood, and early hypoxaemia may be masked. At the point where the SpO_2 reaches 90-92%, the PaO_2 will have decreased to around 8 kPa. In other words, the partial pressure of oxygen in the arterial blood will have decreased by almost 50% despite a reduction in oxygen saturation of only 6-8%.

The output from a pulse oximeter relies on a comparison between current signal output and standardised reference data derived from healthy volunteers. Readings provided are thus limited by the scope of the population included in these studies, and become increasingly unreliable with increasing hypoxaemia. Below 70% the displayed values are highly unreliable.

There are several acknowledged sources of error with pulse oximetry:

- Presence of other haemoglobins: carboxyhaemoglobin (carbon monoxide poisoning), methaemoglobin (congenital or acquired), fetal haemoglobins and sickling red cells (sickle cell disease)
- Surgical and imaging dyes: methylene blue, indocyanine green and indigo carmine cause falsely low saturation readings

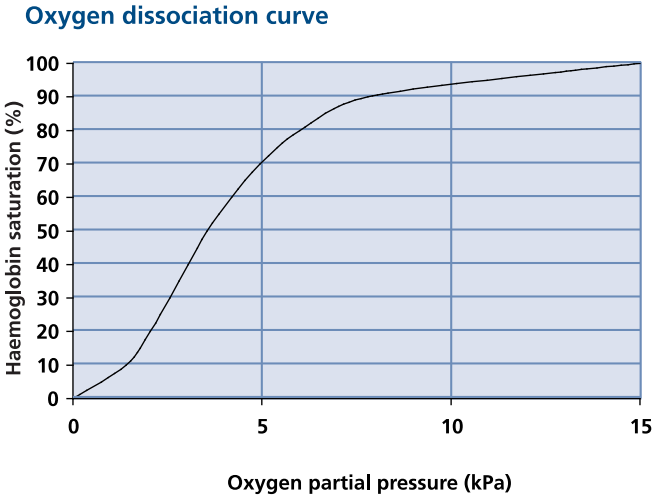

Figure 15.1 Oxyhaemoglobin dissociation curve

- Nail varnish (especially blue, black and green)
- High-ambient light levels (fluorescent and xenon lamps)
- Motion artefact
- Reduced pulse volume:
  - Hypotension
  - Low cardiac output
  - Vasoconstriction
  - Hypothermia

Pulse oximeters are not affected by:

- Anaemia (reduced haemoglobin)
- Jaundice (hyperbilirubinaemia)
- Skin pigmentation

Pulse oximetry does not provide a reliable signal during CPR.

Uses

Pulse oximetry has four main uses:

1. detection of/screening for hypoxaemia;
2. targeting oxygen therapy;
3. routine monitoring during anaesthesia;
4. diagnostic (e.g. sleep apnoea).

Targeted oxygen therapy

In critically ill patients, those presenting with acute hypoxaemia (initial SpO_2 < 85%), or in the peri-arrest situation, give high-concentration oxygen immediately. Give this initially with an oxygen mask and reservoir ('non-rebreathing' mask) and an oxygen flow of 15 l min^{-1}. During cardiac arrest use 100% inspired oxygen concentration to maximise arterial oxygen content and delivery to the tissues.

Once ROSC has been achieved and the oxygen saturation of arterial blood can be monitored reliably, adjust the inspired oxygen concentration to maintain a SpO_2 of 94 - 98%. If pulse oximetry (with a reliable reading) is unavailable, continue oxygen via a reservoir mask until definitive monitoring or assessment of oxygenation is available. All critically ill patients will need arterial blood gas sampling and analysis as soon as possible. Evidence suggests both hypoxaemia and hyperoxaemia (PaO_2 > 20 kPa) in the post-resuscitation phase may lead to worse outcomes than those in whom normoxaemia is maintained.

Special clinical situations

Patients with respiratory failure can be divided into two groups:

- Type I: low PaO_2 (< 8 kPa), normal $PaCO_2$ (< 6 - 7 kPa). In these patients it is safe to give a high concentration of oxygen initially with the aim of returning their PaO_2 to normal and then once clinically stable, adjusting the inspired oxygen concentration to maintain an SpO_2 of 94 - 98%.

- Type II: low PaO_2 (< 8 kPa), increased $PaCO_2$ (> 6 - 7 kPa). This is often described as hypercapnic respiratory failure and is usually caused by COPD. If given excessive oxygen, these patients may develop worsening respiratory failure with further increases in $PaCO_2$ and the development of a respiratory acidosis. If unchecked, this will eventually lead to unconsciousness, and respiratory and cardiac arrest. **The target oxygen saturation in this at risk population should be 88 - 92%.** However, when critically ill, give these patients high-flow oxygen initially; then analyse the arterial blood gases and use the results to adjust the inspired oxygen concentration. When clinically stable and a reliable pulse oximetry reading is obtained, adjust the inspired oxygen concentration to maintain an SpO_2 of 88 - 92%.

In patients with a myocardial infarction or an acute coronary syndrome, and who are not critically or seriously ill, aim to maintain an SpO_2 of 94 - 98% (or 88 - 92% if the patient is at risk of hypercapnic respiratory failure). This may be achievable without supplementary oxygen, and represents a change from previously accepted practice.

Key learning points

- The results of arterial blood gas analysis should be interpreted systematically using the 5-step approach.
- Pulse oximetry enables arterial blood oxygen saturation to be monitored continuously.
- During CPR use an inspired oxygen concentration of 100% until return of spontaneous circulation (ROSC) is achieved.
- After ROSC is achieved, and once the SpO_2 can be monitored reliably, titrate the inspired oxygen concentration to keep the SpO_2 in the range 94 - 98% (or 88 - 92% in patients at risk of hypercapnic respiratory failure).

Further reading

A Simple Guide to Blood Gas Analysis. Eds. Driscoll P, Brown T, Gwinnutt C, Wardle T. BMJ Publishing Group. London 1997.

O'Driscoll BR, Howard LS, Davison AG. BTS guideline for emergency oxygen use in adult patients. Thorax 2008;63 Suppl 6:vi1-68.

Example cases

Case 1:

21 year old woman, thrown from her horse at a local event. On the way to hospital, she has become increasingly drowsy and the paramedics have inserted an oropharyngeal airway and given high-flow oxygen via a face mask with a reservoir. On arrival at hospital, an arterial blood sample shows:

PaO_2	18.8 kPa (FiO_2 85%)
pH	7.19
$PaCO_2$	10.2 kPa
Bicarbonate	23.6 mmol l^{-1}
Base excess	-2.4 mmol l^{-1}

Step 1: From the history we would predict the reduction in level of consciousness to impair ventilation, decreasing oxygenation and increasing $PaCO_2$, causing a respiratory acidosis. There is unlikely to be much compensation because the situation is acute.

Step 2: Although the PaO_2 is just above the normal range, breathing 85% oxygen we would expect a PaO_2 around 75 kPa. Therefore there is a significant impairment in oxygenation.

Step 3: The patient clearly has an acidaemia with a pH well below normal.

Step 4: The $PaCO_2$ is increased, consistent with the low pH and the patient has a respiratory acidosis.

Step 5: The base excess is just below the normal limit and the bicarbonate is within normal limits. This confirms that there is no significant metabolic contribution or compensation.

In summary, the patient has an acute respiratory acidosis with impaired oxygenation.

Case 2:

A 19 year old man with asthma is bought to the emergency department (ED) by his parents. Over the past 4 h he has become increasingly wheezy with no response to his inhalers. He is now very distressed, tachypnoeic and has audible wheeze. He is receiving oxygen at 15 l min^{-1} via a face mask with reservoir and analysis of an arterial blood sample shows:

PaO_2	23.6 kPa (FiO_2 85%)
pH	7.57
$PaCO_2$	3.4 kPa
Bicarbonate	23.1 mmol l^{-1}
Base excess	+1.8 mmol l^{-1}

Step 1: From the history we would predict the bronchospasm to impair oxygenation and the hyperventilation to reduce his $PaCO_2$ causing a respiratory alkalosis. There is unlikely to be much compensation because the situation is acute.

Step 2: Although the PaO_2 is above the normal range, breathing 85% oxygen we would expect a PaO_2 around 75 kPa. Therefore there is a significant impairment in oxygenation.

Step 3: The patient clearly has an alkalaemia with a pH above the normal range.

Step 4: The $PaCO_2$ is decreased, consistent with the raised pH and the patient has a respiratory alkalosis.

Step 5: The base excess and bicarbonate are within normal limits. This confirms that there is no significant metabolic contribution or compensation.

In summary, the patient has an acute respiratory alkalosis with impaired oxygenation.

Case 3:

A 52 year old man, complaining of crushing central chest pain is bought to the ED by his wife. He is attached to an ECG monitor, given oxygen 40% by face mask, and sublingual GTN; an intravenous cannua is inserted and he is given aspirin and morphine. After about 5 minutes he suddenly has a cardiac arrest. After 4 minutes of resuscitation he has a palpable pulse and starts to breathe spontaneously. Analysis of an arterial blood sample shows:

PaO_2	8.9 kPa (FiO_2 40%)
pH	7.11
$PaCO_2$	7.2 kPa
Bicarbonate	14 mmol l^{-1}
Base excess	-10.6 mmol l^{-1}

Step 1: From the history we would predict the impaired ventilation to result in hypoxaemia, an increased $PaCO_2$ and respiratory acidosis. The impaired circulation will cause an increase in anaerobic respiration, production of lactate and a metabolic acidosis that will consume bicarbonate. The failure of circulation is likely to prevent any degree of compensation.

Step 2: The patient is hypoxaemic and breathing 40% oxygen. We would expect a PaO_2 around 30 kPa. Therefore there is a significant impairment in oxygenation.

Step 3: The patient clearly has a severe acidaemia, with a very low pH.

Step 4: The $PaCO_2$ is increased, consistent with the low pH and the patient has a respiratory acidosis.

Step 5: The base excess and bicarbonate are both reduced. This is consistent with a metabolic acidosis.

In summary, the patient has a mixed respiratory and metabolic acidosis with impaired oxygenation.

Case 4:

A 68 year old man with a long history of COPD is reviewed on the medical ward before discharge. Analysis of an arterial blood sample shows:

PaO_2	8.9 kPa (FiO_2 40%)
pH	7.34
$PaCO_2$	7.3 kPa
Bicarbonate	30.2 mmol l^{-1}
Base excess	5.3 mmol l^{-1}

Step 1: From the history we would predict the patient to have a chronically raised $PaCO_2$ causing a respiratory acidosis. However, there is likely to be significant compensation in the form of a metabolic alkalosis. Oxygenation is likely to be impaired.

Step 2: The PaO_2 is significantly reduced. Breathing 40% oxygen we would expect a PaO_2 around 30 kPa. Therefore there is a significant impairment in oxygenation.

Step 3: The patient has a borderline acidaemia with a pH just below the normal range.

Step 4: The $PaCO_2$ is increased, causing a respiratory acidosis. However, the increase is probably greater than we would expect from the minimal reduction in pH.

Step 5: The base excess and bicarbonate are both increased confirming that there is a metabolic alkalosis. This compensation has helped minimise or compensate for the pH disturbance caused by the respiratory acidosis.

In summary, the patient has a chronic respiratory acidosis with a compensatory metabolic alkalosis, with significantly impaired oxygenation.

Case 5:

A 22 year old male, recently diagnosed with insulin dependent diabetes mellitus presents to the ED having been unwell for 48 h and with a gradually increasing blood sugar concentration, despite taking his insulin. He is notably tachypnoeic and tachycardic and a point-of-care measurement of his blood glucose is 23 mmol l^{-1}. Analysis of an arterial blood sample while breathing oxygen, 6 l min^{-1} via a facemask shows:

PaO_2	22.2 kPa (FiO_2 40%)
pH	7.34
$PaCO_2$	3.8 kPa
Bicarbonate	19.1 mmol l^{-1}
Base excess	-7.9 mmol l^{-1}

Step 1: From the history the most likely problem is that the patient is developing a diabetic ketoacidosis i.e. a metabolic acidosis. However, the fact that he is tachypnoeic suggests that he is trying to compensate by reducing his $PaCO_2$. This will cause a respiratory alkalosis. If there are no abnormal signs in his chest, oxygenation should be relatively normal.

Step 2: Breathing 40% oxygen we would expect a PaO_2 around 30 kPa. However, with a tachypnoea, the facemask is probably delivering less than 40% oxygen and so his oxygenation is unimpaired.

Step 3: The patient has a borderline acidaemia with a pH just below the normal range.

Step 4: The $PaCO_2$ is decreased, causing a respiratory alkalosis and therefore not the cause of the primary disturbance.

Step 5: The base excess and bicarbonate are both decreased confirming that there is a metabolic acidosis. However, the pH is not as low as would be expected for this degree of change.

In summary, the patient has a metabolic acidosis (as a result of impaired glucose metabolism and the production of ketoacids) with a compensatory respiratory alkalosis.