Second Edition

Understanding ABGs & Lung Function Tests

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Recognising abnormal results

chapter 3

The ability to recognise abnormal patterns in lung function and arterial blood gas abnormalities (ABGs) allows for a rapid narrowing of the differential diagnosis. For example, an obstructive lung spirometry pattern in a person with long-standing shortness of breath can point towards a particular type of respiratory disease. Likewise, a blood gas abnormality that can be easily identified as primarily metabolic or respiratory in nature can help rapidly narrow down the cause of an acute illness.

3.1 Arterial blood gas abnormalities

ABGs are one of the first investigations performed for a critically ill patient because they provide rapid and vital information on blood oxygenation, alveolar ventilation and tissue perfusion. In interpreting ABG results, it is useful to group abnormalities as primarily a disturbance of:

- acid–base balance
- ventilation and oxygenation

Identification of critical ABG results

The major ABG changes that suggest that a patient is in a critical condition are shown in **Table 3.1**.

Acid–base disturbances

ABG results can show whether there is an acidotic or alkalotic disturbance of the blood, and whether the cause of the imbalance is primarily respiratory (i.e. related to either breathing or alveolar blood diffusion) or metabolic (i.e. concerning acid and base metabolism in the blood or cells). Therefore, four patterns of acid–base disorders

Guiding principle

Definitions:

- **Acidaemia:** blood pH is <7.35
- **Acidosis:** the process causing acid to accumulate, usually causing acidaemia
- **Alkalaemia:** blood pH is >7.45
- **Alkalosis:** the process causing alkali to accumulate, usually causing alkalaemia

Note: the terms acidosis and alkalosis are commonly used in place of acidaemia and alkalaemia, respectively.

ABG measure	Indication of severity
pH	Low pH indicates acidosis that is damaging to tissues and organs
PCO_2	Rises if patient becomes more drowsy, indicating assisted ventilation
PO_2	Falls if patient becomes tired and cannot compensate for metabolic acidosis, indicating assisted ventilation
Lactate	High lactate indicates acidosis that is damaging to tissues and organs
Potassium	High potassium damages tissues (especially cardiomyocytes) and may necessitate emergency dialysis

Table 3.1 Major shifts in arterial blood gas (ABG) values which suggest the need for critical care support

	pH	$Paco_2$ (kPa)	Standard bicarbonate (mmol/L)	Compensation
Respiratory acidosis	↓ (<7.35)	↑ (>6)	Normal/ ↑ (>22)	Renal
Respiratory alkalosis	↑ (>7.45)	↓ (<4.7)	Normal/ ↑ (>22)	Renal
Metabolic acidosis	↓ (<7.35)	Normal/ ↓ (<4.7)	↓ (<20)	Respiratory
Metabolic alkalosis	↑ (>7.45)	Normal	↑ (>33)	Respiratory

Table 3.2 Arterial blood gas (ABG) values in the four different acid–base disturbances

are classified (**Table 3.2**). These primary disorders are usually also seen with compensatory changes in blood gases, as the system attempts to retain homeostasis. In chronic disturbances of acid–base balance, more compensation is usually seen, as the system has adapted to compensate to better protect the pH.

Figure 3.1 shows how to identify quickly which acid–base disturbance is present. Some cases of mixed acid–base

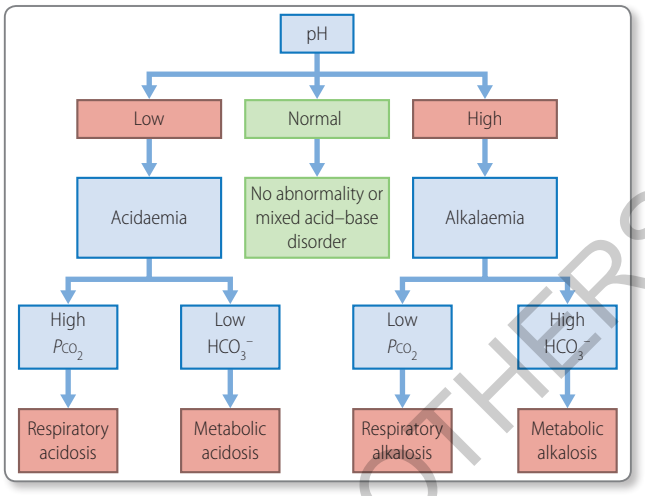

Figure 3.1 Identifying an acid–base disorder.

disturbance also occur, showing different elements of the four patterns.

The anion gap

The anion gap is a calculated estimate of the negatively charged particles in blood that are not directly measured by common blood tests. These unmeasured anions include proteins and organic acids, and the gap is based on the difference between the main measured cations (Na^+ and sometimes K^+) and anions (Cl^- and HCO_3^-). It is important to calculate and consider this value because it helps to differentiate the cause of a metabolic acidosis. A normal anion gap is 10–18 mmol/L. Metabolic acidosis with an increased anion gap is caused by excess acid, whereas a normal anion gap metabolic acidosis is from loss of base.

The anion gap is calculated by including or, more commonly, ignoring the level of potassium (because this is usually negligible):

$$\text{Anion gap} = ([Na^+]) - ([Cl^-] + [HCO_3^-])$$

$$\text{or Anion gap} = ([Na^+] + [K^+]) - ([Cl^-] + [HCO_3^-])$$

Interpreting the ABG report in five steps The basic rules for interpreting abnormal blood gas results are shown in **Table 3.3.** Interpreting ABG results can be done in five steps:

1. pH: is the primary acid–base abnormality acidaemia (<7.35) or alkalaemia (>7.45)?
2. $PaCO_2$ (arterial carbon dioxide tension) and bicarbonate: does it indicate a metabolic or respiratory cause?
3. Compensation: is it present? If not, or it is to an inappropriate level, there may be a secondary problem
4. Anion gap: this is a calculated estimate of unmeasured blood anions and is important in the context of metabolic acidosis, as it can narrow down where the acid is coming from
5. PaO_2: how well is the alveolar O_2 diffusing into the arteries, i.e. what is the alveolar–arterial gradient ($P_{A-a}O_2$)? Compare PaO_2 to the inspired O_2 concentration that the patient is breathing and the $PaCO_2$

Compensation Compensatory changes seen in ABG results include increased CO_2 blow-off (hyperventilation) in metabolic acidosis and increased renal H^+ excretion in respiratory acidosis. If hypoventilation occurs there is an increase in blood CO_2 concentration and pressure, and secondary changes in the bicarbonate concentration due to renal compensation. This renal compensatory mechanism is affected by the retention of sodium ions and the excretion of hydrogen ions as ammonium chloride; the sodium is available to form more sodium bicarbonate, thus increasing the bicarbonate and attempting to maintain a HCO_3^-: $PaCO_2$ 20:1 ratio.

However, renal compensation is rarely complete and the pH tends to drop. In respiratory acidosis, therefore, the blood shows an increase in $PaCO_2$, total CO_2 content and bicarbonate (HCO_3^-), and a decrease in pH and serum chloride concentration. In respiratory alkalosis, where a high pH results from hyperventilation, the opposite findings are observed.

Respiratory acidosis

In respiratory acidosis, there is a decrease in respiratory exchange (of O_2 with CO_2), causing retention of $PaCO_2$ in the

Measurement	Interpretation
pH	High pH indicates alkalosis, respiratory or metabolic Low pH indicates acidosis, respiratory or metabolic
PaO_2	High PaO_2 may be due to hyperventilation causing a respiratory alkalosis Low PaO_2 indicates hypoxia and supplemental O_2 may be needed
$PaCO_2$	High $PaCO_2$ indicates hypoventilation, leading to a respiratory acidosis Low $PaCO_2$ may be due to hyperventilation, leading to respiratory alkalosis
HCO_3^-	High bicarbonate indicates a metabolic alkalosis Low bicarbonate is seen in metabolic acidosis (e.g. diabetic ketoacidosis)
Base excess	A more positive base excess (> +2) indicates metabolic alkalosis A more negative base excess (< –2) indicates metabolic acidosis
Anion gap	High anion gap indicates loss of bicarbonate in metabolic acidosis Low anion gap indicates hypoalbuminaemia

Table 3.3 Causes of changes in blood gas measurements

blood. This results in a high $PaCO_2$ which leads to renal retention of bicarbonate in order to buffer the excess H^+. The kidneys compensate by increasing secretion of H^+ over a period of 3–5 days which results in increased plasma HCO_3^-.

Causes of respiratory acidosis are listed in **Table 3.4**.

Respiratory alkalosis

Respiratory alkalosis is caused by alveolar hyperventilation, so that excessive CO_2 is exhaled and a low $PaCO_2$ results. Renal compensation is by decreasing ammonium (NH_4^+) excretion, leading to a fall in HCO_3^-.

Causes of respiratory alkalosis are listed in **Table 3.5**.

Type of cause	Cause
Central	Drugs, e.g. morphine and sedatives Stroke Infection
Airway obstruction	Asthma Chronic obstructive pulmonary disease (COPD)
Parenchymal emphysema	Pneumoconiosis Bronchitis Acute respiratory distress syndrome Barotrauma
Neuromuscular	Poliomyelitis Kyphoscoliosis Myasthenia gravis Muscular dystrophies
Miscellaneous	Obesity Hypoventilation

Table 3.4 Causes of respiratory acidosis

Metabolic acidosis

Metabolic acidosis results from the body producing too much acid or from the kidneys failing to excrete enough. This acid accumulation causes a primary decrease in HCO_3^- as carbonic acid is produced to buffer the acid. The lungs compensate by hyperventilation, which decreases $PaCO_2$ as CO_2 is blown off.

Guiding principle

The anion gap estimates the level of serum ions that aren't measured in routine analysis because they are difficult to measure. It can be considered a measure of organic acids such as phosphate, ketones and lactate. It is calculated by subtracting the total serum anion concentration (Cl^- and HCO_3^-) from serum cations (Na^+ and K^+). In practice, potassium is often left out because it is usually negligible. Normally, the anion gap is 10–18 mmol/L.

The anion gap calculation is important to differentiate the cause of a metabolic acidosis. If the anion gap is normal, this means that bicarbonate is being lost, either in the gastrointestinal tract (e.g. diarrhoea) or by renal disease letting it leak out. A high anion gap results from increased production of organic acids (e.g. lactic acid, urate or diabetic

Type of cause	Cause
Central nervous system stimulation	Pain Anxiety, psychosis Fever Cerebrovascular accident Meningitis Encephalitis Trauma
Hypoxaemia or tissue hypoxia	High altitude Pneumonia Pulmonary oedema Aspiration Severe anaemia
Drugs or hormonal causes	Pregnancy Progesterone Salicylates Nikethamide
Stimulation of thoracic neural receptors	Haemothorax Flail chest Cardiac failure Pulmonary embolism
Miscellaneous	Septicaemia Mechanical hyperventilation Hepatic failure Heat exposure Recovery from metabolic acidosis

Table 3.5 Causes of respiratory alkalosis. Most mechanisms increase pH by hyperventilation and CO_2 blow-off

ketoacidosis) or ingestion of large amounts of acid (e.g. ethylene glycol poisoning).

Causes of metabolic acidosis are listed in **Table 3.6**.

Metabolic alkalosis

Metabolic alkalosis is the result of an increase of HCO_3^- due to either a decreased H^+ concentration (e.g. by copious vomiting) or a direct increase in HCO_3^-. The latter can occur from bicarbonate retention, an intracellular shift of H^+ or by ingestion of large amounts of alkali (e.g. antacids). The lungs compensate

Type of metabolic acidosis	Cause
High anion gap	Lactic acidosis Ketoacidosis, e.g. diabetes, alcohol abuse, starvation Renal failure: acute and chronic. Toxins, e.g. methanol, ethylene glycol, salicylates
Normal anion gap	
Bicarbonate loss from gastrointestinal tract	Diarrhoea Extrarenal pancreatic or small bowel drainage Ureterosigmoidostomy Drugs, e.g. calcium chloride (acidifying agents), magnesium sulphate
Renal acidosis	Proximal renal tubular acidosis Distal renal tubular acidosis Tubulointerstitial disease
Drug induced	Potassium-sparing diuretics, e.g. amiloride, spironolactone Trimethoprim Pentamidine Angiotensin-converting enzyme (ACE) inhibitors Non-steroidal anti-inflammatory drugs (NSAIDs)
Others	Rapid saline infusion Loss of potential bicarbonate (i.e. anion loss in urine)

Table 3.6 Causes of metabolic acidosis

by slower breathing (hypoventilation) to retain CO_2, showing as a rise in $PaCO_2$.

Causes of metabolic alkalosis are listed in **Table 3.7**.

Mixed acid–base disturbances

Mixed acid–base disturbances occur frequently in hospitalised patients, especially when they are critically ill. This is because many patients, especially in a critical condition, can have mixed pathologies affecting pH homeostasis. They can be difficult to interpret, and a good understanding of compensatory mechanisms and its proportionality is needed. Four signs that suggest a mixed acid–base disorder is present are:

Type of cause	Cause
Acute alkali administration	E.g. milk alkali syndrome
Gastrointestinal	Vomiting Nasogastric suction Villous adenoma
Renal diuretics	Hypercalcaemia Recovery from lactic acidosis/ketoacidosis Hypokalaemia
Effective extracellular volume expansion	High renin: renal artery stenosis Accelerated hypertension Aldosteronism Cushing's syndrome Steroids

Table 3.7 Causes of metabolic alkalosis

1. Compensation is not occurring, or is too little or too much
2. pH is normal but $PaCO_2$ or HCO_3^- is abnormal
3. $PaCO_2$ and HCO_3^- are abnormal in opposite directions
4. In metabolic acidosis, the change in HCO_3^- concentration is not proportional to the change in the anion gap

One commonly seen pattern is an increased $PaCO_2$ with alkaline pH. This results from an attempt to reduce the $PaCO_2$ in a patient with hypercapnic respiratory failure by hyperventilation on a mechanical ventilator. Hypercapnic alkalaemia is also seen in patients with lactic acidosis or diabetic keto acidosis (DKA) who are treated with excess bicarbonate, i.e. an anion gap metabolic acidosis with metabolic alkalosis.

If $PaCO_2$ or HCO_3^- is abnormal in opposite directions, so that one is raised and the other reduced, it is likely that a mixed respiratory and metabolic acid–base disorder is present. For example, combined respiratory and metabolic acidosis presents with elevated $PaCO_2$ and reduced HCO_3^-. The decrease in pH is also much more than expected from the rise in $PaCO_2$ values.

In combined respiratory and metabolic alkalosis, on the other hand, the $PaCO_2$ is reduced and the HCO_3^- is elevated.

Similarly, the pH rise is more than you would expect when looking at the fall in $PaCO_2$.

Mixed respiratory and metabolic disturbances can occur in patients with chronic obstructive pulmonary disease (COPD) who are hypercapnic with respiratory acidosis and develop a metabolic alkalosis as a result of therapy with steroids and diuretics. The pH is generally near normal.

3.2 Respiratory failure

Most of the causes of hypoxia (if sustained and severe enough) lead to respiratory failure (a $PaO_2 < 8$ kPa). Disturbed pulmonary gas exchange leads to low PaO_2 with or without raised $PaCO_2$. The mechanisms leading to respiratory failure are similar to those leading to hypoxia (discussed in more detail below).

Respiratory failure is due to one of three mechanisms, each presenting different patterns on ABGs (**Table 3.8**):

- Inadequate ventilation of gases into alveoli
- Inadequate diffusion of gases across the respiratory membrane
- Inadequate blood perfusion

Type of failure	Causes
Ventilatory	Upper airway pathology, e.g. obstructive sleep apnoea, foreign body aspiration Lower airway pathology, e.g. cancers, obstructive airway disease Muscle pathology, e.g. myopathy, trauma Neurological disease, e.g. motor neuron disease, central respiratory depressant drugs
Diffusion	Pleural causes, e.g. pneumothorax, pleural effusions Parenchymal causes, e.g. bronchiectasis, obstructive airway disease
Perfusion	Pulmonary embolism Intracardiac shunting

Table 3.8 Causes of respiratory failure

Type I and type II respiratory failure

Patients are divided into type I and type II respiratory failure (**Table 3.9**) based on key differences in how they respond to supplemental O_2 treatment.

- Type I is hypoxia ($PaO_2 < 8$ kPa) with a normal/low $PaCO_2$ and is caused by $\dot{V}/\dot{Q}$ mismatch
- Type II is hypoxia and hypercapnia ($PaCO_2 > 6.0$ kPa) and is caused by hypoventilation with or without a $\dot{V}/\dot{Q}$ mismatch

The treatment in for both is O_2 and respiratory support. Caution is required when treating type II respiratory failure, because in certain patients excessive oxygen may lead to a worsening of hypercapnia (see Chapter 4, page 103).

Type of failure	ABG results	Causes	Response to oxygen therapy
Type I	• PaO_2 low (<8.0 kPa) • $PaCO_2$ normal or low • P_A–aO_2 increased	Asthma, pneumonia and pulmonary embolism	Usually good response
Type II	• PaO_2 decreased (<8.0 kPa) • $PaCO_2$ increased (>6.0 kPa) • P_A–aO_2 normal • pH decreased	Pulmonary: asthma, COPD, pneumonia, fibrosis Neuromuscular: myasthenia gravis, Guillain–Barré syndrome, poliomyelitis, Cervical spine injury Reduced respiratory drive: sedatives, CNS disease Anatomical: kyphoscoliosis	At risk of hypercapnia due to loss of central respiratory drive. Give controlled O_2 and monitor serial ABGs

Table 3.9 Types of respiratory failure

Hypoxia

Normal PaO_2 is 10.5–13.5 kPa. Hypoxia is therefore defined as <10.5 kPa and considered severe (i.e. respiratory failure) if <8 kPa. Hypoxia and/or hypercapnia ($PaCO_2$ > 6 kPa) is due to one of the following mechanisms:

1. **Low inspired oxygen** (FiO_2), e.g. when at high altitude
2. **Alveolar hypoventilation** due to a reduction in ventilation at the level of the alveoli. Causes include physical defects of the alveoli, such as pneumonia, or a depressed respiratory centre, as seen in CNS disease or obesity–hypoventilation syndromes. The alveolar–arterial O_2 gradient (P_A–aO_2) is usually normal
3. **Abnormal diffusion** occurs when the respiratory membrane disrupts O_2 (and CO_2) diffusion across it, leading to an increased P_A–aO_2 gradient. Thickened, scarred basement membranes interrupt diffusion, and are seen in diseases such as idiopathic pulmonary fibrosis, sarcoidosis and other interstitial lung diseases. Diffusion of O_2 is affected to a greater extent than that of CO_2 because the latter diffuses at 20 times the rate
4. **Ventilation–perfusion mismatch** is when ventilation is not matching blood perfusion through the alveolar capillaries, the most common reason for hypoxia. An example is a pulmonary embolism. The ventilation–perfusion ratio ($\dot{V}/\dot{Q}$) represents the ratio between alveolar ventilation and capillary perfusion. A ventilation–perfusion mismatch can also occur in other conditions mentioned above, such as if pneumonia causes alveolar hypoventilation in an area of lung which remains perfused
5. **Shunts** are when blood meant for the pulmonary capillary bed bypasses it. There are various shunt mechanisms. For example, gravity exerts a physiological shunt on lung blood supply, and a normal anatomical shunt occurs due to collateral veins (e.g. the veins draining the myocardium). A right-to-left cardiac shunt is when blood moves from the right circulation to the left due to openings between atria, ventricles or great vessels, and right heart pressure is greater than left

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ISBN 978-1-909836-85-3

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