CHAPTER 3
Acid–Base Balance

By the end of this chapter you will be able to:

- Understand how the body maintains a narrow pH
- Know the meaning of common terms used in arterial blood gas analysis
- Know the causes of acid–base abnormalities
- Use a simple system to interpret arterial blood gases
- Understand why arterial blood gases are an important test in critical illness
- Apply this to your clinical practice

Acid as a By-Product of Metabolism

The human body is continually producing acid as a by-product of metabolism. But it must also maintain a narrow pH range, necessary for normal enzyme activity and the millions of chemical reactions that take place in the body each day. Normal blood pH is 7.35–7.45 and this is maintained by:

- Intracellular buffers (e.g. proteins and phosphate)
- Extracellular buffers (e.g. plasma proteins, haemoglobin, and carbonic acid/bicarbonate)
- Finally, the excretory functions of the kidneys and lungs

A buffer is a substance that resists pH change by absorbing or releasing hydrogen ions (H^+) when acid or base is added to it. The intracellular and extracellular buffers absorb hydrogen ions and transport them to the kidneys for elimination. The carbonic acid/bicarbonate system allows hydrogen ions to react with bicarbonate to produce carbon dioxide and water and the carbon dioxide is eliminated by the lungs:

$$H^+ + HCO_3^- \xleftrightarrow{\text{carbonic anhydrase (enzyme)}} H_2CO_3 \xleftrightarrow{} CO_2 + H_2O$$

Carbonic acid (H_2CO_3) continually breaks down to form carbon dioxide and water, so this system always tends to move in a rightward direction and, unlike other buffer systems,

Essential Guide to Acute Care, Third Edition. Nicola Cooper, Paul Cramp, Kirsty Forrest and Rakesh Patel.

never gets saturated. But it is easy to see how, for example, a problem with ventilation would quickly lead to a build-up of CO_2 – a respiratory acidosis. Uniquely, the components of the carbonic acid/bicarbonate system can be adjusted independently of one another. The kidneys can regulate H^+ excretion in the urine and CO_2 levels can be adjusted by changing ventilation. The excretory functions of the lungs and kidneys are connected by carbonic acid so that if one organ becomes overwhelmed, the other can help or 'compensate'.

The lungs have a simple way of regulating CO_2 excretion, but the kidneys have three main ways of excreting H^+:

- Mainly by regulating the amount of bicarbonate (HCO_3^-) absorbed in the proximal tubule
- By the reaction $HPO_4^{2-} + H^+ \longrightarrow H_2PO_4^-$. The H^+ comes from carbonic acid, leaving HCO_3^- which passes in to the blood
- By combining ammonia with H^+ from carbonic acid. The resulting ammonium ions cannot pass back in to the cells and are excreted

The kidney produces bicarbonate (HCO_3^-) which reacts with free hydrogen ions. This is why the bicarbonate level is low when there is an excess of H^+ ions, or a metabolic acidosis.

So, in summary, the body is continually producing acid, yet at the same time must maintain a narrow pH range in order to function effectively. It does this by means of buffers and then the excretory functions of the lungs (CO_2) and kidneys (H^+). It follows therefore that acid–base disturbances occur when there is a problem with ventilation, a problem with renal function, or an overwhelming acid or base load the body cannot handle.

Some Definitions

Before moving on, it is important to understand some important definitions regarding arterial blood gases:

- Acidaemia or alkalaemia – a low pH or high pH
- Acidosis – a process which leads to acidaemia e.g. high $PaCO_2$ or excess H^+ ions (low bicarbonate)
- Alkalosis – a process which leads to alkalaemia e.g. low $PaCO_2$ or high bicarbonate
- Compensation – normal acid–base balance is a normal pH plus a normal $PaCO_2$ and normal bicarbonate. Compensation has occurred when there is a normal pH but the bicarbonate and $PaCO_2$ are abnormal
- Correction – the restoration of normal pH, $PaCO_2$, and bicarbonate
- Base excess – this measures how much extra acid or base is in the system as a result of a metabolic problem. It is calculated by measuring the amount of strong acid that has to be added to a sample to produce a pH of 7.4. A minus figure means the sample is already acidotic so no acid had to be added. A plus figure means the sample is alkalotic and acid had to be added. The normal range is −2 to +2. A minus base excess is often termed a 'base deficit'

- Actual versus standard bicarbonate – a problem with ventilation would quickly lead to a build-up of CO_2, or a respiratory acidosis. This CO_2 reacts with water to produce H^+ and HCO_3^- and therefore causes a small and immediate rise in bicarbonate. The *standard* bicarbonate is calculated by the blood gas analyser from the actual bicarbonate, but assuming 37°C and a normal $PaCO_2$ of 5.3 kPa (40 mmHg). Standard bicarbonate therefore reflects the metabolic component of acid–base balance, as opposed to any changes in bicarbonate that have occurred as a result of a respiratory problem. Some blood gas machines only report the actual bicarbonate, in which case you should use the base excess to examine the metabolic component of acid–base balance. Otherwise, the *standard* bicarbonate and base excess are interchangeable.

  If you do not like equations, skip the box below.

pH and the Henderson–Hasselbach Equation

Everyone has heard of the Henderson–Hasselbach equation, but what is it? H^+ ions are difficult to measure as there are literally billions of them. We use pH instead, which simply put is the negative logarithm of the H^+ concentration in moles.

$$pH = -\log\left[H^+\right]$$

When carbonic (H_2CO_3) acid dissociates:

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

the product of $[H^+]$ and $[HCO_3^-]$ divided by $[H_2CO_3]$ remains constant. Put in equation form:

$$Ka = \frac{\left[H^+\right]\left[HCO_3^-\right]}{\left[H_2CO_3\right]}$$

Ka is the dissociation constant. pKa is like pH – it is the negative logarithm of Ka. The Henderson–Hasselbach equation puts the pH and the dissociation equations together and describes the relationship between pH and the molal concentrations of the dissociated and undissociated form of carbonic acid:

$$pH = pKa + \log\frac{\left[HCO_3^-\right]}{\left[H_2CO_3\right]}$$

Since $[H_2CO_3]$ is related to $PaCO_2$, a simplified version is:
pH is proportional to $[HCO_3^-]/PaCO_2$.

This simple relationship can be used to check the consistency of arterial blood gas data. If we know that pH (or the concentration of H^+ ions) is related to the ratio of HCO_3^- and $PaCO_2$, it should be easy to check whether a blood gas result is 'real' or not, or the result of lab error (see Appendix at the end of this chapter).

Common Causes of Acid–Base Disturbances

As previously mentioned, acid–base disturbances occur when there is:

- A problem with ventilation
- A problem with renal function
- An overwhelming acid or base load the body cannot handle

Respiratory Acidosis

Respiratory acidosis is caused by acute or chronic alveolar hypoventilation. The causes are described in Chapter 2 and include upper or lower airway obstruction, reduced lung compliance from infection, oedema, trauma, or obesity, and anything that causes respiratory muscle weakness, including fatigue.

In an acute respiratory acidosis, cellular buffering is effective within minutes to hours. Renal compensation takes 3–5 days to be fully effective. We know from human volunteer studies[1] by how much the standard bicarbonate rises as part of the compensatory response. Although doctors do not frequently use these figures in everyday practice, having a rough idea can be useful (see Table 3.1).

Respiratory Alkalosis

Respiratory alkalosis is caused by alveolar hyperventilation, the opposite of respiratory acidosis, and is nearly always accompanied by an increased respiratory rate. Again, renal compensation takes up to 5 days to be fully effective by excreting bicarbonate in the urine and

Table 3.1 Renal and respiratory compensation.

	Primary change	Compensatory response
Metabolic acidosis	↓ $[HCO_3^-]$	For every 1 mmol/L fall in $[HCO_3^-]$, $PaCO_2$ falls by 0.15 kPa (1.2 mmHg)
Metabolic alkalosis	↑ $[HCO_3^-]$	For every 1 mmol/L rise in $[HCO_3^-]$, $PaCO_2$ rises by 0.01 kPa (0.7 mmHg)
Acute respiratory acidosis	↑ $PaCO_2$	For every 1.3 kPa (10 mmHg) rise in $PaCO_2$, $[HCO_3^-]$ rises by 1 mmol/L
Chronic respiratory acidosis	↑ $PaCO_2$	For every 1.3 kPa (10 mmHg) rise in $PaCO_2$, $[HCO_3^-]$ rises by 3.5 mmol/L
Acute respiratory alkalosis	↓ $PaCO_2$	For every 1.3 kPa (10 mmHg) fall in $PaCO_2$, $[HCO_3^-]$ falls by 2 mmol/L
Chronic respiratory alkalosis	↓ $PaCO_2$	For every 1.3 kPa (10 mmHg) fall in $PaCO_2$, $[HCO_3^-]$ falls by 4 mmol/L

retaining hydrogen ions. When asked what causes hyperventilation, junior doctors often reply 'anxiety'. In fact, hyperventilation is a sign, not a diagnosis and it has many causes:

- Lung causes: bronchospasm, hypoxaemia, pulmonary embolism, pneumonia, pneumothorax, and pulmonary oedema
- Central nervous system causes: stroke, cerebral haemorrhage, and raised intracranial pressure
- Metabolic causes: fever, hyperthyroidism
- Drugs (e.g. salicylate poisoning)
- Psychogenic causes: pain, anxiety

Metabolic Acidosis

Metabolic acidosis most commonly arises from an overwhelming acid load. Respiratory compensation occurs within minutes. Maximal compensation occurs within 12–24 hours, but respiratory compensation is limited by the work involved in breathing and the systemic effects of a low carbon dioxide (mainly cerebral vasoconstriction). It is unusual for the body to be able to fully compensate for a metabolic acidosis.

There are many potential causes of a metabolic acidosis, so it is important to subdivide these in to metabolic acidosis with an increased anion gap and metabolic acidosis with a normal anion gap. In general, a metabolic acidosis with an increased anion gap is caused by the body gaining acid, whereas a metabolic acidosis with a normal anion gap is caused by the body losing base.

The Anion Gap

Blood tests measure most cations (positively charged molecules) but only a few anions (negatively charged molecules). Anions and cations are equal in the human body, but if all the *measured* cations and anions are added together there would be a gap – this reflects the concentration of those anions not measured, mainly plasma proteins. This is called the anion gap and is calculated from a blood sample:

$$(\text{Sodium} + \text{potassium}) - (\text{chloride} + \text{bicarbonate})$$

The normal range for the anion gap is 15–20 mmol/L, but this varies from lab to lab and should be adjusted downwards in patients with a low albumin (by 2.5 mmol/L for every 1 g/dL fall in plasma albumin). Similarly, a fall in any unmeasured cations (e.g. calcium or magnesium) may produce a spurious increase in the anion gap.

Some patients may have more than one reason to have a metabolic acidosis (e.g. diarrhoea leading to loss of bicarbonate plus sepsis and hypoperfusion). Many blood gas machines calculate the anion gap but if not, it should always be calculated when there is a metabolic acidosis as this helps to narrow down the cause. The base deficit is known to correlate with mortality.[2] A severe metabolic acidosis usually indicates critical illness.

Metabolic Acidosis with an Increased Anion Gap

In a metabolic acidosis with an increased anion gap, the body has gained acid through:

- Ingestion
- The body's own production
- An inability to excrete it

Common clinical causes are:

- Ingestion: salicylate, methanol, ethylene glycol, and tricyclic antidepressant poisoning
- Lactic acidosis type A (anaerobic tissue metabolism): any condition causing tissue hypoperfusion, either global (e.g. shock, cardiac arrest) or local (e.g. intra-abdominal ischaemia)
- Lactic acidosis type B (liver dysfunction): reduced lactate metabolism in liver failure, metformin
- Ketoacidosis: insulin deficiency (diabetic ketoacidosis), starvation
- Impaired renal function
- Severe rhabdomyolysis (damaged cells release H^+ and organic anions)

Metabolic Acidosis with a Normal Anion Gap

In a metabolic acidosis with a normal anion gap, bicarbonate is lost via the kidneys or the gastrointestinal tract. Occasionally, reduced renal H^+ excretion is the cause. A normal anion gap metabolic acidosis is sometimes also called 'hyperchloraemic acidosis'. Common clinical causes are:

- Renal tubular acidosis
- Diarrhoea, fistula, or ileostomy
- Acetazolomide therapy

Overall, the most common causes of a metabolic acidosis in hospital are those that have an increased anion gap.

Mini-Tutorial: The Use of Intravenous Sodium Bicarbonate in Metabolic Acidosis

HCO_3^- as sodium bicarbonate is sometimes administered intravenously to raise blood pH in a severe metabolic acidosis but this poses several problems. It increases the formation of CO_2 which passes readily in to cells (unlike HCO_3^-) and this can worsen intracellular acidosis. It is hypertonic and contains a significant sodium load (see Chapter 5). Some patients with airway or ventilation problems may need mechanical ventilation to counter the increased CO_2 production caused by an infusion of sodium bicarbonate. Tissue necrosis can result from extravasation from the cannula. Many of the causes of metabolic acidosis respond to restoration of intravascular volume and tissue perfusion with oxygen, intravenous fluids, and treatment of the underlying cause. For these reasons, *routine* intravenous sodium bicarbonate is not recommended to treat a metabolic acidosis. It tends to be reserved for specific conditions e.g. tricyclic poisoning (when it acts like an 'antidote') or treatment of a severe metabolic acidosis with acute kidney injury (see mini-tutorial in Chapter 7). A solution of 8.4% sodium bicarbonate contains 1 mmol/mL of sodium or bicarbonate.

Table 3.2 Changes in pH, $PaCO_2$, and standard bicarbonate in different acid–base disturbances.

	pH	$PaCO_2$	St bicarbonate/BE	Compensatory response
Respiratory acidosis	Low	High	Normal	st bicarbonate rises
Metabolic acidosis	Low	Normal	Low	$PaCO_2$ falls
Respiratory alkalosis	High	Low	Normal	st bicarbonate falls
Metabolic alkalosis	High	Normal	High	$PaCO_2$ rises

Metabolic Alkalosis

Metabolic alkalosis is the least well known of the acid–base disturbances. It can be divided in to two groups: saline responsive and saline unresponsive. Saline responsive metabolic alkalosis is the most common and occurs with volume depletion (e.g. vomiting or diuretic use). Gastric outflow obstruction is a well-known cause of 'hypokalaemic hypochloraemic metabolic alkalosis'. Excessive vomiting or nasogastric suction leads to the loss of hydrochloric acid, but the decline in glomerular filtration rate which often accompanies these conditions perpetuates the metabolic alkalosis. The kidneys try to reabsorb chloride (hence urine chloride levels are low), but there is less of it from loss of hydrochloric acid, so the only available anion to be reabsorbed is bicarbonate. Metabolic alkalosis is often associated with hypokalaemia due to secondary hyperaldosteronism from volume depletion as well as the vomiting.

Another cause of saline-responsive metabolic alkalosis is when hypercapnia is corrected quickly by mechanical ventilation. Post-hypercapnia alkalosis occurs because a high $PaCO_2$ directly affects the proximal tubules and decreases sodium chloride reabsorption leading to volume depletion. If chronic hypercapnia is corrected rapidly with mechanical ventilation, a metabolic alkalosis can ensue because there is already a high bicarbonate and the kidney needs time to excrete it. The pH change causes a shift in potassium which can result in hypokalaemia and sometimes cardiac arrhythmias.

Saline unresponsive metabolic alkalosis occurs due to renal problems:

- With high blood pressure: excess mineralocorticoid (exogenous or endogenous)
- With normal blood pressure: severe hypokalaemia, hypercalcaemia
- High-dose penicillin therapy
- Ingestion of exogenous alkali with a low glomerular filtration rate

A summary of the changes in pH, $PaCO_2$, and standard bicarbonate in different acid–base disturbances is shown in Table 3.2.

Interpreting an Arterial Blood Gas Report

There are a few simple rules when looking at an arterial blood gas report:

- Always consider the clinical situation
- An abnormal pH indicates the primary acid–base problem

- The body never overcompensates
- Mixed acid–base disturbances are common in clinical practice

Any test has to be interpreted in the light of the clinical situation. A normal blood gas result might be reassuring, but not if the patient has acute severe asthma when a 'normal' $PaCO_2$ would be extremely worrying. The body's compensatory mechanisms only aim to bring the pH towards normal and never swing like a pendulum in the opposite direction. So a low pH with a high $PaCO_2$ and high standard bicarbonate is always a respiratory acidosis and never an 'overcompensated' metabolic alkalosis. These principles will be easily seen as you work through the case histories at the end of this chapter. Many doctors miss vital information when interpreting arterial blood gas results because they do not use a systematic method.

There are five steps to follow when interpreting an arterial blood gas result:

1) Look at the pH first
2) Look at the $PaCO_2$ and the standard bicarbonate (or base excess) to see whether this is a respiratory or a metabolic problem, or both
3) Check the appropriateness of any compensation. For example, in a metabolic acidosis, you would expect the $PaCO_2$ to be low. If the $PaCO_2$ is normal, this indicates a 'hidden' respiratory acidosis as well. (If you like, you can use Winter's formula as a shortcut to calculate the expected $PaCO_2$: Expected $PaCO_2 = [(1.5 \times HCO_3^-) + 8 \pm 2] \times 0.133$)
4) Check or calculate the anion gap if there is a metabolic acidosis.
5) Finally, look at the PaO_2 and compare it to the inspired oxygen concentration (more on this in Chapter 4)

Why Arterial Blood Gas Analysis is Important in Critical Illness

Arterial blood gas analysis can be performed quickly and gives the following useful information:

- A measure of oxygenation (PaO_2)
- A measure of ventilation ($PaCO_2$)
- A measure of perfusion (standard bicarbonate or base excess)

In other words, a measure of A, B, and C – which is why it is an extremely useful test in the management of a critically ill patient.

Key Points – Acid–Base Balance

- The body maintains a narrow pH range using buffers and then the excretory functions of the lungs and kidneys
- Acid–base disturbances occur when there is a problem with ventilation, a problem with renal function, or an overwhelming acid or base load the body cannot handle
- Use the five steps described here to interpret an arterial blood gas result so that important information is not missed
- Arterial blood gases are an important test in critical illness

Self-Assessment: Case Histories

Normal values: pH 7.35–7.45, $PaCO_2$ 4.5–6.0 kPa (35–46 mmHg), PaO_2 11–14.5 kPa (83–108 mmHg), BE −2 to +2, and st bicarbonate 22–28 mmol/L.

1 A 65-year-old man with COPD comes to the emergency department complaining of breathlessness. His arterial blood gases on air show: pH 7.29, $PaCO_2$ 8.5 kPa (65.3 mmHg), st bicarbonate 30.5 mmol/L, BE +4, and PaO_2 8.0 kPa (62 mmHg). What is the acid–base disturbance and what is your management?

2 A 60-year-old ex-miner with severe COPD is admitted with breathlessness. His arterial blood gases on air show: pH 7.36, $PaCO_2$ 9.0 kPa (65.3 mmHg), st bicarbonate 35 mmol/L, BE +6, and PaO_2 6.0 kPa (46.1 mmHg). What is the acid–base disturbance and what is your management?

3 A 24-year-old man with epilepsy is admitted to hospital in tonic-clonic status epilepticus. This is terminated by the administration of intravenous lorazepam. Arterial blood gases on 10 L/min oxygen via reservoir bag mask show: pH 7.05, $PaCO_2$ 8.0 kPa (61.5 mmHg), st bicarbonate 16 mmol/L, BE −8, and PaO_2 15 kPa (115 mmHg). His other results are sodium 140 mmol/L, potassium 4 mmol/L, and chloride 98 mmol/L. What is his acid–base status and why? What is your management?

4 A 44-year-old man comes to the emergency department with pleuritic chest pain and shortness of breath which he has had for a few days. A moderate-sized left-sided pneumothorax is seen on his chest X-ray. His arterial blood gases on 10 L/min oxygen via reservoir bag mask show: pH 7.44, $PaCO_2$ 3.0 kPa (23 mmHg), st bicarbonate 16 mmol/L, BE −8, and PaO_2 30.5 kPa (234.6 mmHg). Is there a problem with acid–base balance?

5 A patient is admitted to hospital with breathlessness and the arterial blood gases on air show: pH 7.2, $PaCO_2$ 4.1 kPa (31.5 mmHg), st bicarbonate 36 mmol/L, BE +10, and PaO_2 7.8 kPa (60 mmHg). Can you explain this?

6 An 80-year-old woman is admitted with abdominal pain. She has cool peripheries and a tachycardia. Her respiratory rate is 24/ min and her oxygen saturations are 95% on air. Her arterial blood gases on air show: pH 7.1, $PaCO_2$ 3.5 kPa (30 mmHg), st bicarbonate 8 mmol/L, BE −20, and PaO_2 12 kPa (92 mmHg). You review the clinical situation – she has generalised tenderness in the abdomen but it is soft. There is no fever. Her blood glucose is 6.0 mmol/L (100 mg/dL) and her creatinine and liver tests are normal. The chest X-ray is normal. There are reduced bowel sounds. The ECG shows atrial fibrillation. What is the most likely reason for the acid–base disturbance? What is your management?

7 A 30-year-old woman who is 36 weeks pregnant has arterial blood gases taken on air because of pleuritic chest pain and breathlessness. The results are as follows: pH 7.48, $PaCO_2$ 3.4 kPa (26 mmHg), st bicarbonate 19 mmol/L, BE −4, and PaO_2 14 kPa (108 mmHg). What do these blood gases show? Could this indicate a pulmonary embolism?

8 A 45-year-old woman with a history of peptic ulcer disease reports 6 days of persistent vomiting. On examination, she has a blood pressure of 100/60 mmHg and looks dehydrated and unwell. Her blood results are as follows: sodium 140 mmol/L, potassium 2.2 mmol/L, chloride 86 mmol/L, actual bicarbonate 40 mmol/L, urea 29 mmol/L (BUN 80 mg/dL), pH 7.5, $PaCO_2$ 6.2 kPa (53 mmHg), PaO_2 14 kPa (107 mmHg), urine pH 5.0, urine sodium 2 mmol/L, urine potassium 21 mmol/L, and urine chloride 3 mmol/L. What is the acid–base disturbance? How would you treat this patient? Twenty-four hours after appropriate therapy, the venous bicarbonate is 30 mmol/L and the following urine values are obtained: pH 7.8, sodium 100 mmol/L, potassium 20 mmol/L, and chloride 3 mmol/L. How do you account for the high urinary sodium but low urinary chloride concentration?

9 A 50-year-old man is recovering on a surgical ward 5 days after a total colectomy for bowel obstruction. He has type 1 diabetes and is on intravenous insulin. His ileostomy is working normally. His vital signs are: blood pressure 150/70 mmHg, respiratory rate 16/min, SpO_2 98% on air, urine output 1200 mL/day, temperature 36.7°C, and he is well perfused. The surgical team are concerned about his persistently high potassium (which was noted preoperatively as well) and metabolic acidosis. His blood results are: sodium 130 mmol/L, potassium 6.5 mmol/L, urea 14 mmol/L (BUN 39 mg/dL), creatinine 180 µmol/L (2.16 mg/dL), chloride 109 mmol/L, normal synacthen test, and albumin. He is known to have diabetic nephropathy and is on ramipril. His usual creatinine is 180 µmol/L. His arterial blood gases on air show: pH 7.31, $PaCO_2$ 4.0 kPa (27 mmHg), st bicarbonate 15 mmol/L, BE −8, and PaO_2 14 kPa (108 mmHg). The surgical team are wondering whether this persisting metabolic acidosis means that there is an intra-abdominal problem, although a recent abdominal CT scan was normal. What is your advice?

10 Match the clinical history with the appropriate arterial blood gas values:

	pH	$PaCO_2$	St bicarbonate (mmol/L)
a	7.39	8.45 kPa (65 mmHg)	37
b	7.27	7.8 kPa (60 mmHg)	26
c	7.35	7.8 kPa (60 mmHg)	32

- A severely obese 24-year-old man
- A 56-year-old woman with COPD who has been started on a diuretic for peripheral oedema, resulting in a 3 kg weight loss
- A 16-year-old girl with a severe asthma attack

Self-Assessment: Discussion

1 There is an acidaemia (low pH) due to a high $PaCO_2$ – a respiratory acidosis. The standard bicarbonate is just above normal. The PaO_2 is low. Management starts with assessment and treatment of airway, breathing, and circulation. Medical treatment of an

exacerbation of COPD includes controlled oxygen therapy, nebulised/inhaled bronchodilators, steroids, antibiotics if necessary, and non-invasive ventilation if the respiratory acidosis does not resolve quickly.[3]

2 There is a normal pH with a high $PaCO_2$ (respiratory acidosis) and a high st bicarbonate (metabolic alkalosis). Which came first? The history points towards this being a chronic respiratory acidosis, compensated for by a rise in st bicarbonate (renal compensation). If the pH fell due to a further rise in $PaCO_2$, you would call this an 'acute on chronic respiratory acidosis' which would look like this: pH 7.17, $PaCO_2$ 14.6 kPa (109 mmHg), st bicarbonate 39 mmol/L, BE +7.6, and PaO_2 6.0 kPa (46.1 mmHg). Management would be the same as for case number 1. Note that non-invasive ventilation is only indicated when the pH falls below 7.35 due to a rise in $PaCO_2$.

3 There is an acidaemia (low pH) due to a high $PaCO_2$ and a low standard bicarbonate – a mixed respiratory and metabolic acidosis. The PaO_2 is low in relation to the inspired oxygen concentration. The high $PaCO_2$ is likely to be due to airway obstruction and the respiratory depressant effects of intravenous lorazepam. This can be deduced because there is such a large difference between the inspired oxygen concentration (FiO_2) and the PaO_2. Aspiration pneumonia is another possibility. Persistent tonic-clonic seizures cause a lactic acidosis because of anaerobic muscle metabolism. Management in this case starts with assessment and treatment of airway, breathing, and circulation, followed by disability and examination/planning. A benzodiazepine aborts 80% of seizures in status epilepticus. Lorazepam is the drug of choice because seizures are less likely to relapse compared with diazepam (55% at 24 hours compared with 50% at 2 hours). Additional therapy is then required to keep seizures at bay – 15–20 mg/kg of intravenous phenytoin as a slow infusion with cardiac monitoring is an example of initial treatment. If this fails, consider other diagnoses and sedation with propofol or barbiturates on the intensive care unit.[4]

4 There is a normal pH with a low $PaCO_2$ (respiratory alkalosis) and a low st bicarbonate (metabolic acidosis). Which came first? The history points towards this being a respiratory alkalosis, compensated for by a fall in st bicarbonate (a compensated respiratory alkalosis) over the last few days. On the other hand, if you saw a similar arterial blood gas in a patient presenting with hyperglycaemia and ketonaemia, it could be a patient heading towards diabetic ketoacidosis.

5 As you may have guessed, this is an impossible blood gas – the answer is lab error!

6 There is an acidaemia (low pH) due to a very low st bicarbonate (metabolic acidosis). The $PaCO_2$ is appropriately low, although it should be lower than this – approximately 2.5 kPa, possibly indicating that the patient is tiring. The PaO_2 is normal. The anion gap is not given, but the presence of atrial fibrillation is a clue to the diagnosis of ischaemic bowel. Intra-abdominal catastrophes are associated with a metabolic acidosis. This case also illustrates what was described in Chapter 1 – an acute abdomen is usually soft in older people. They have fewer signs that are more diffuse, less well

localised, and often atypical despite the presence of serious intra-abdominal pathology.[5] Management starts with assessment and treatment of airway, breathing, and circulation followed by disability and examination/planning – call the surgeon.

7 There is a high pH (alkalaemia) due to a low $PaCO_2$ (respiratory alkalosis). The st bicarbonate is just below normal. The PaO_2 is normal. A respiratory alkalosis is a normal finding in advanced pregnancy.[6] The A–a gradient is not affected by pregnancy and is normal in this case (see Chapter 4). Arterial blood gases (including the A–a gradient) can be normal in pulmonary embolism and therefore do not help in making the diagnosis.

8 There is a high pH (alkalaemia) due to a high bicarbonate (metabolic alkalosis). The $PaCO_2$ is just above normal. The PaO_2 is normal, assuming the patient is breathing room air. The potassium and chloride levels are both low. This is the hypokalaemic hypochloraemic metabolic alkalosis seen in severe vomiting due to gastric outflow obstruction. The physical findings and low urinary chloride point towards volume depletion. The patient requires intravenous sodium chloride 0.9% with potassium. During therapy, volume expansion reduces the need for sodium reabsorption, hence the high levels in the urine. The discrepancy between urinary sodium and chloride is primarily due to urinary bicarbonate excretion. Further sodium chloride replacement is necessary for as long as the low urinary chloride persists, since it indicates ongoing chloride and volume depletion.

9 There is a low pH (acidaemia) due to a low st bicarbonate – a metabolic acidosis. The $PaCO_2$ is appropriately low. The anion gap may be calculated as $(130+6.5)-(12+109) = 15.5$ mmol/L, which is normal. The PaO_2 is normal. Common causes of a normal anion gap metabolic acidosis include renal tubular acidosis, diarrhoea, fistula, ileostomy, and acetazolamide therapy. In this case, excessive gastrointestinal losses and acetazolomide can be excluded, which leaves a possible renal cause. Renal tubular acidosis is a collection of disorders in which the kidneys either cannot excrete H^+ or generate bicarbonate. Only one of the renal tubular acidoses is associated with a high serum potassium – type 4, or 'hyporeninaemic hypoaldosteronism' which is seen in patients with diabetic or hypertensive nephropathy and is often exacerbated by ACE inhibitors, aldosterone blockers (e.g. spironolactone), and non-steroidal anti-inflammatory drugs. The metabolic acidosis seen in this condition is usually mild, with the st bicarbonate concentration remaining around 15 mmol/L. Treatment usually consists of dietary restriction of potassium, stopping exacerbating medication, and oral sodium bicarbonate.

10 Severe obesity suggests chronic hypercapnia (c). COPD and diuretic therapy suggest chronic hypercapnia with a superimposed metabolic alkalosis (a). A severe asthma attack suggests an acute respiratory acidosis (b).

Appendix – Checking the Consistency of Arterial Blood Gas Data

H^+ concentration is sometimes used instead of pH. A simple conversion between pH 7.2 and 7.5 is that $[H^+] = 80$ minus the two digits after the decimal point. So if the pH is 7.35, then $[H^+]$ is $80-35 = 45$ nmol/L (see Table 3.3).

Table 3.3 pH and equivalent [H^+].

pH	[H^+] nmol/L
7.6	26
7.5	32
7.4	40
7.3	50
7.2	63
7.1	80
7.0	100
6.9	125
6.8	160

Earlier in this chapter, the box on the Henderson–Hasselbach equation explained that pH is proportional to $[HCO_3^-]/PaCO_2$. Another way of writing this is as follows:

$$\left[H^+\right] = Ka\frac{PaCO_2}{HCO_3^-} \quad \text{where Ka is the dissociation constant}$$

$$\text{or} \left[H^+\right] = 181 \times \frac{PaCO_2}{HCO_3^-} \quad \text{in kPa (or } 24 \times PaCO_2 \text{ in mmHg)}$$

The following arterial blood gas: pH 7.25, $PaCO_2$ 4.5 kPa (35 mmHg), st bicarbonate 14.8 mmol/L, and PaO_2 8.0 kPa (61 mmHg), shows a metabolic acidosis. A pH of 7.25 = [H+] of 55 nmol/L. Do the figures add up?

$$181 \times \frac{4.5}{14.8} = 55$$

Yes, a $PaCO_2$ of 4.5 kPa with a st bicarbonate of 14.8 would give a [H^+] of 55 nmol/L. You may find this simple calculation useful when checking for lab error, or making up arterial blood gases for teaching material.

References

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Further Resources

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