Blood gases, electrolytes and interpretation 2. Electrolytes

Yvonne McGrotty and Graham Bilbrough

Obtaining and interpreting blood gases and electrolytes is fundamental to the management of many critically ill veterinary patients. Metabolic and electrolyte alterations are common, and can lead to profound clinical signs affecting many organs (including the heart, skeletal muscle, intestines and the lungs); when severe, death can ensue. This is the second of two articles describing the interpretation of blood gases and electrolytes, focusing on the electrolyte parameters measured by the widely available in-clinic blood gas analysers. The first article, published in the January issue of In Practice, focused on acid-base and oxygen status.

INTERPRETATION of a blood gas profile is incomplete without consideration of electrolyte concentrations. This should focus on recognition of acid-base and electrolyte patterns. This rarely leads to a specific diagnosis; however, it allows for tailoring of fluid therapy and specific intervention with potentially life-saving fluid additives, such as potassium. Electrolyte abnormalities are often the first sign of an acid-base disorder and are required to calculate the anion gap, as discussed in the first part of this article (McGrotty and Brown 2013). Many in-clinic blood gas analysers, such as the IDEXX VetSTAT, the Epocal epoc and the Abbot i-STAT, will also measure electrolytes including sodium, potassium, chloride and ionised calcium. In addition, some blood gas analysers will also measure lactate concentration.

All in-clinic blood gas analysers use technology that differs, to a varying degree, from that typically used at reference laboratories (ion-specific potentiometry). For example, the IDEXX VetSTAT uses fluorescent dye sensors which emit light that is dependent on the concentration of the electrolyte present in the sample. A photo detector converts this fluorescence into a voltage and a numerical result is produced. The different methods provide comparable results, but care must be taken to use analyser-specific reference intervals. The clinician should be aware of the effect of interfering substances (lipids, haemoglobin, bilirubin) on the performance of the analyser.

Potassium disorders

Potassium (K⁺) is the primary intracellular cation and around 95 per cent of total body potassium is found within the cell. A sodium/potassium ATPase pump ensures that serum concentrations of potassium are low, while cellular values are high. Concentrations of intracellular and extracellular potassium are tightly

Sample haemolysis can lead to a mild increase in potassium; this effect is much more pronounced in certain dogs (eg, Akitas, English springer spaniels, neonates) which have relatively higher potassium concentrations within their red blood cells.

Inappropriate anticoagulants can have profound effects on electrolyte results. For example, ethylenediaminetetraacetic acid (EDTA), oxalate and citrate exert their anticoagulant effects by binding calcium; this results in hypocalcaemia if these anticoagulants are used for electrolyte analysis. Oxalate and EDTA are supplied chelated with potassium, which can result in an apparent hyperkalaemia if such samples are analysed. Heparin is the anticoagulant of choice when measuring electrolytes.

Particular care should be taken when samples are obtained through an intravenous catheter to ensure that the sample was not diluted by intravenous fluid therapy or ‘flush’ solution.

Sampling and artefacts

Electrolytes can be measured on both venous and arterial samples. Whole blood, serum and heparinised plasma are suitable for electrolyte analysis, although the reference intervals may vary with the sample type. For example, the plasma potassium concentration is slightly lower than the serum concentration because potassium is released from the platelets during clotting. This effect is more pronounced with thrombocytosis.

Lipaeamia and extreme hyperproteinaemia can result in pseudohyponatraemia and pseudohypokalaemia using some analysers. Sodium and potassium are measured in the aqueous fraction of plasma and when that plasma is occupied by excess quantities of lipid or protein, this leads to an apparent dilutional effect on these electrolytes.

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Sodium disorders

Sodium (Na⁺) is the major cation within the extracellular fluid (ECF). The kidney has the ability to maintain plasma sodium concentrations within narrow limits, regardless of dietary intake. Any decrease in circulating blood volume results in activation of the renin–angiotensin–aldosterone system and the production of aldosterone by the adrenal cortices. This leads to active resorption of sodium by the renal tubules.

When interpreting serum sodium values, the animal’s hydration status must be taken into consideration (ie, is the animal normovolaemic, hypovolaemic or hypervolaemic?). Retention of sodium is typically associated with water retention, while sodium loss leads to concurrent water loss.

Causes of hypernatraemia and hyponatraemia are listed in Table 2. Hypernatraemia usually results from inadequate water intake (leading to dehydration and haemoconcentration), water losses (eg, vomiting or diarrhoea) or, rarely, excessive intake of salt. Hyponatraemia may occur due to hypotonic fluid losses (eg, vomiting), renal losses (eg, hypoadrenocorticism), third spacing (eg, repeated drainage of chylous effusions) or volume overload (eg, congestive heart failure). Clinical signs of hyponatraemia and hypernatraemia are more related to the rapidity of change than the absolute value. Neurological signs (eg, disorientation, ataxia, seizures and coma) are likely when the sodium concentration is greater than 170 mmol/l or less than 120 mmol/l.

Chloride disorders

Chloride (Cl⁻) is the most prevalent anion in the ECF. Changes in the concentration of chloride generally mirror those of sodium in order to maintain electroneutrality. Therefore, accurate interpretation of the chloride status also requires concurrent measurement of sodium. Despite chloride’s importance, it is often overlooked when interpreting electrolytes and acid-base status. Plasma chloride concentration, and thus acid-base status, is regulated by the kidney by altering the amount of chloride that is reabsorbed along with sodium in the renal tubules.

The plasma concentration of chloride can be a powerful tool in characterising an underlying acid-base disorder. The ‘Law of Electroneutrality’ states that the total concentration of the positive electrolytes (the cations) must equal the total concentration of the negative electrolytes (the anions) (as shown in Table 3).

An increased ‘gap’ between the sodium (cation) and chloride (anion) implies a metabolic alkalosis (Fig 2). If the patient has a normal or decreased bicarbonate concentration, then there must be a mixed metabolic alkalosis and acidosis. Meanwhile, for the patient with a metabolic acidosis (decreased plasma bicarbonate concentration), the gap between sodium and chloride concentrations can provide information on the underlying cause (Fig 2). If the gap is decreased, it implies that there is no increase in the concentration of unmeasured anions. On the other hand, if the gap remains unchanged or increased, there is an increase in unmeasured anions. This consideration is only appropriate when plasma protein concentrations are within the reference range.

Causes of hypo- and hyperchlaema are shown in Table 4. Hyperchloraemia typically will be seen in any situation where hypernatraemia is present, or where there is a decrease in bicarbonate, as an attempt to maintain electroneutrality (Fig 2). Increased concentrations of chloride are associated with the development of acidosis, due to a decrease in strong ion...
Table 2: Causes of hyper- and hyponatraemia in dogs and cats (common causes shown in italics)

<table>
<thead>
<tr>
<th>Hypernatraemia (Na⁺ greater than 160 mmol/l)*</th>
<th>Hyponatraemia (Na⁺ less than 144 mmol/l)*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normovolaemic (pure water losses)</td>
<td>Normovolaemic</td>
</tr>
<tr>
<td>• Hypodipsia/adipsia/water deprivation</td>
<td>• Latrogenic, ie, inappropriate use of hypotonic fluid therapy or anti-diuretic drugs</td>
</tr>
<tr>
<td>• Diabetes insipidus</td>
<td>• Psychogenic polydipsia</td>
</tr>
<tr>
<td>• Pancreatitis</td>
<td>• Syndrome of inappropriate ADH secretion (rare)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Hypo- or hyperchloremia (hypotonic fluid losses)</th>
<th>Hypo- or hyperchloremia (hypotonic fluid losses)</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Vomiting and diarrhoea</td>
<td>• Vomiting and diarrhoea</td>
</tr>
<tr>
<td>• Third space losses (eg, peritonitis and pancreatitis)</td>
<td>• Third space losses, eg, peritonitis, pancreatitis</td>
</tr>
<tr>
<td>• Diuretics</td>
<td>• Diuretics</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Hypervolaemic (solute gain)</th>
<th>Hypervolaemic</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Drinking sea water or salt poisoning</td>
<td>• Congestive heart failure</td>
</tr>
<tr>
<td>• Hyperaldosteronism</td>
<td>• Severe hepatic disease causing ascites</td>
</tr>
<tr>
<td>• Iatrogenic (administration of hypertonic saline)</td>
<td>• Nephrotic syndrome/advanced renal failure</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Miscellaneous</th>
<th>Miscellaneous</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Hyperglycaemia</td>
<td>• Artefact (lipaemia)</td>
</tr>
</tbody>
</table>

*Reference intervals may vary with species, sample type and analyser

ADH Antidiuretic hormone

Lactate

Lactate measurement may be available on some blood gas analysers. Lactate is an organic acid synthesised

Ionised calcium

Many blood gas analysers can also reliably measure ionised calcium ([iCa²⁺]), which is the biologically active fraction of total calcium. Around 50 per cent of calcium is found in the ionised form, with the remainder being bound to proteins (mainly albumin) or chelated with anions. Protein-bound and chelated calcium are biologically inactive. Unlike total calcium measurements, ionised calcium is unaffected by alterations in the serum albumin concentration. Ionised and total calcium measurements are often poorly correlated.

Measurement of ionised calcium is superior to that of total calcium in many settings, but samples have strict handling requirements unless they can be analysed immediately because changes in pH, refrigeration, freezing and exposure to oxygen can have a significant effect on [iCa²⁺] results. Rapid patient-side analysis of [iCa²⁺] using a blood gas analyser helps to reduce the effects that may occur with delays in testing (where the sample is exposed to air, resulting in increased pH and decreased [iCa²⁺]). Heparinised plasma, serum or whole blood can be used for ionised calcium analysis; however, the authors recommend the use of a dry, electrolyte-balanced heparin, followed by immediate centrifugation and direct analysis of the resultant plasma. If a liquid lithium heparin is the only available option, particular care should be taken to observe the correct ratio of anticoagulant to blood.

Measurement of ionised calcium can help to differentiate various causes of hypercalcaemia (Table 5); ionised calcium is generally elevated in hypercalcaemia of malignancy and in hyperparathyroidism. Only a small proportion of animals with renal failure or hypoadrenocorticism will have increased ionised calcium values, even though total calcium may be increased.

Ionised hypocalcaemia may occur in hypoparathyroidism, ethylene glycol toxicity and pancreatitis.

Lactate

Lactate measurement may be available on some blood gas analysers. Lactate is an organic acid synthesised

**Fig 2:** Differences in the plasma cations and anions in alkalosis, normal and acidosis patients. The major cations in the plasma are sodium (Na⁺) and potassium (K⁺). Also, there are small concentrations of unmeasured cations (UA⁻). The major anions are chloride (Cl⁻) and bicarbonate (bicarb); there are also anions that cannot be measured by today’s analysers. The anion gap is a calculated value (using the concentrations of sodium, potassium, chloride and bicarbonate) and an elevated value can indicate an increased concentration of unmeasured anions. In alkalosis, there is an increase in the plasma bicarbonate concentration. In metabolic acidosis, there is a decrease in the plasma bicarbonate concentration. Metabolic acidosis is frequently divided into two types: high anion gap acidosis (there is an increase in the unmeasured anions) and hyperchloremic metabolic acidosis (normal anion gap; chloride anion increases to replace the lost bicarbonate and maintain electroneutrality).
from pyruvate. It is synthesised in high quantities (mostly by the skeletal muscle and gut) when anaerobic metabolism occurs and is, therefore, an indicator of systemic hypoperfusion and tissue hypoxia. When lactate production exceeds metabolism, lactic acidosis occurs. Two isomeric forms exist: L-lactate and D-lactate, but only L-lactate is measured by blood gas analysers.

Two types of lactic acidosis are recognised (Table 6):

- Type A: due to systemic or local hypoxia
- Type B: non-hypoxic form which occurs due to a variety of metabolic disorders and toxins.

Type A lactic acidosis is the most likely form to be identified in veterinary patients.

Either venous or arterial samples can be used to measure lactate and measurements should be made within 30 minutes of collecting the sample (preferably within five minutes) to prevent false elevations resulting from glycolytic activity by red blood cells. If this is not possible, the authors recommend the use of fluoride oxalate as an anticoagulant and immediate separation of the red blood cells and analysis within eight hours. A consistent sample handling technique is needed for a meaningful interpretation of the trend in lactate concentration.

Prolonged venous stasis (longer than one minute) and exertion can result in elevated plasma lactate concentrations. Care should be taken when interpreting samples obtained from peripheral veins (eg, cephalic vein) as prolonged venous occlusion can lead to local venous stasis that may affect lactate concentration.

The reference range for lactate in dogs is accepted as being less than 2.5 mmol/l, and less than 1.4 mmol/l in cats. Levels of hyperlactataemia are proportional to the severity of the hypoperfusion. Values below the reference interval are not considered to be clinically significant. Neonates typically have higher lactate concentrations than adults. Generally, lactate values greater than 5 mmol/l will lead to acidaemia. Serial measurements of lactate are of much more use than a single value, and trends over time can be used to determine whether therapy has been effective. Pronounced elevations of lactate are not necessarily associated with a poorer prognosis as long as values decrease rapidly with appropriate therapy. However, sustained (type A) hyperlactataemia is a very serious sign and is associated with a poor prognosis.

### Table 4: Causes of hyper- and hypochloraemia in dogs and cats

<table>
<thead>
<tr>
<th>Hyperchloraemia (Cl⁻ greater than 122 mmol/l)*</th>
<th>Hypochloraemia (Cl⁻ less than 109 mmol/l)*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Artifactual (potassium bromide therapy)</td>
<td>Drug-induced (loop diuretics, sodium bicarbonate)</td>
</tr>
<tr>
<td>Iatrogenic (administration of potassium chloride, physiological saline [0.9 per cent sodium chloride], hypertonic saline)</td>
<td>Gastrointestinal losses (vomiting, diarrhoea)</td>
</tr>
<tr>
<td>Diarrhoea</td>
<td>Metabolic alkalosis</td>
</tr>
<tr>
<td>Hypodipsia/adipsia</td>
<td></td>
</tr>
<tr>
<td>Hyperaldosteronism (rare)</td>
<td></td>
</tr>
<tr>
<td>Salt poisoning (rare)</td>
<td></td>
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<tr>
<td>Respiratory alkalosis</td>
<td></td>
</tr>
</tbody>
</table>

*Reference intervals may vary with species, sample type and analyser

### Table 5: Causes of ionised hyper- and hypocalcaemia in dogs and cats (conditions generally affecting only total calcium values are shown in brackets)

<table>
<thead>
<tr>
<th>Hypercalcaemia (iCa²⁺ greater than 1.5 mmol/l)*</th>
<th>Hypocalcaemia (iCa²⁺ less than 1.25 mmol/l)*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lymphoma</td>
<td>Primary hyperparathyroidism</td>
</tr>
<tr>
<td>Multiple myeloma</td>
<td>Ethylene glycol toxicity</td>
</tr>
<tr>
<td>Apocrine sac adenocarcinoma</td>
<td>Pancreatitis</td>
</tr>
<tr>
<td>Primary hyperparathyroidism</td>
<td>Artifactual (EDTA, citrate or oxalate contamination; excessive lithium heparin)</td>
</tr>
<tr>
<td>(Chronic renal failure)</td>
<td></td>
</tr>
<tr>
<td>Hypoadrenocorticism</td>
<td></td>
</tr>
<tr>
<td>Vitamin D toxicity</td>
<td></td>
</tr>
<tr>
<td>Granulomatous disease</td>
<td></td>
</tr>
<tr>
<td>Idiopathic in cats</td>
<td></td>
</tr>
</tbody>
</table>

*Reference intervals may vary with species, sample type and analyser

### Table 6: Causes of hyperlactaemia/lactic acidosis

<table>
<thead>
<tr>
<th>Type A lactic acidosis</th>
<th>Type B lactic acidosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Systemic hypoperfusion</td>
<td>Toxins, eg, ethylene glycol, ethanol</td>
</tr>
<tr>
<td>• Cardiogenic shock</td>
<td>Drugs, eg, salicylates, propylene glycol</td>
</tr>
<tr>
<td>• Hypovolaemic shock</td>
<td>Diabetes mellitus</td>
</tr>
<tr>
<td>• Septic shock</td>
<td>Severe liver disease</td>
</tr>
<tr>
<td>Local hypoperfusion, eg, gastric necrosis</td>
<td>Neoplasia</td>
</tr>
<tr>
<td>Increased glycolysis, eg, exercise, trembling</td>
<td>Septis/systemic inflammatory response</td>
</tr>
<tr>
<td></td>
<td>syndrome</td>
</tr>
<tr>
<td></td>
<td>Hypoglycaemia</td>
</tr>
</tbody>
</table>

*Reference intervals may vary with species, sample type and analyser

### Treatment of electrolyte disorders

Most electrolyte and lactate disorders will resolve after correction of the underlying problem. For example, lactate concentration will quickly return to normal as tissue perfusion is restored. However, judicious use of intravenous fluid therapy may expedite the return to normality. In the case of gastric vomiting leading to a hyperchloraemic metabolic alkalosis, the use of 0.9 per cent sodium chloride (Cl⁻ 150 mmol/l) may be preferable to Hartmann’s solution (Cl⁻ 111 mmol/l) as a resuscitative fluid to restore the acid-base balance.

When severe hyper- or hyponatraemia is present, caution should be taken that the maximum rate of change is 0.5 mmol/l sodium per hour (either direction). An inappropriate selection of resuscitative fluid, such as 0.18 per cent sodium chloride solution to treat hypernatraemia, can be fatal.

An increased ionised calcium concentration should always prompt further diagnostic procedures and treatment. Numerous options include: fluid diuresis, furosemide, glucocorticoids, bisphosphonates and others. Treatment should be more aggressive if the
patient has clinical symptoms. The use of glucocorticoids should be avoided in hypercalcaemic patients until the underlying diagnosis has been confirmed. With hypocalcaemia, when tetany is present, intravenous calcium should be administered cautiously (electrocardiogram monitoring is recommended).

### Summary

Blood gas, pH and electrolyte analyses have more immediacy and potential impact on critical patient care than any other laboratory determination. The interpretation of blood gases is incomplete without consideration of the electrolyte concentrations, and vice versa. Careful sample handling is essential to avoid artefacts that can confuse interpretation.

Electrolyte disorders are common in veterinary practice and result from many different diseases. Typically, the results do not provide a specific diagnosis; rather, the results allow timely intervention with tailored fluid therapy.

### Self-assessment test: Electrolyte examples

In the three cases below, what abnormalities are present and what are the possible causes?

#### Case 1

A six-month-old entire male mixed breed dog is presented by the owner. The dog was found in a semi-comatose state. Venous blood gases were obtained:

- **pH**: 7.137 (7.31 – 7.42)
- **HCO₃⁻**: 3 mmol/l (20 – 29)
- **pCO₂**: 32 mmHg (32 – 49)
- **Na⁺**: 154 mmol/l (144 – 160)
- **K⁺**: 6.5 mmol/l (3.5 – 5.8)
- **Cl⁻**: 111 mmol/l (109 – 122)
- **Anion Gap**: 47 (12 – 24)
- **iCa⁺⁺**: 0.6 mmol/l (1.25 – 1.5)

What abnormalities are present? Suggest a possible cause and what you might anticipate finding from urinalysis.

#### Case 2

A two-year-old neutered female Labrador retriever presents with a one-week history of profuse vomiting. She is 10 per cent dehydrated.

- **pH**: 7.502 (7.31 – 7.42)
- **HCO₃⁻**: 31.1 mmol/l (20 – 29)
- **pCO₂**: 39.3 mmHg (32 – 49)
- **Na⁺**: 119 mmol/l (144 – 160)
- **K⁺**: 2.7 mmol/l (3.5 – 5.8)
- **Cl⁻**: 69 mmol/l (109 – 122)
- **Anion gap**: 46 (12 – 24)
- **Glucose**: 32 mmol/l (1.25 – 1.5)

What abnormalities are present and what would be your approach to intravenous fluid therapy?

#### Case 3

A nine-year-old neutered male mixed breed dog presents collapsed. In the preceding months, the owner had become aware of polydipsia, but had not sought veterinary attention. The owner states that no other signs were observed in the previous days. The dog has a poor body condition. Venous blood gases were obtained:

- **pH**: 7.195 (7.31 – 7.42)
- **HCO₃⁻**: 3 mmol/l (20 – 29)
- **pCO₂**: 27.1 mmHg (32 – 49)
- **Na⁺**: 132.4 mmol/l (144 – 160)
- **K⁺**: 4.3 mmol/l (3.5 – 5.8)
- **Cl⁻**: 98 mmol/l (109 – 122)
- **Anion gap**: 46 (12 – 24)
- **Glucose**: 32 mmol/l (1.25 – 1.5)

What abnormalities are present? Suggest a possible cause and what you might anticipate finding at urinalysis.

(Answers overleaf)

### Reference


### Further reading


Case 1
Abnormalities
The blood pH is decreased: acidaemia.

The HCO$_3^-$ concentration is decreased: metabolic acidosis.

The pCO$_2$ is within the reference interval: the expected compensatory respiratory alkalosis is absent (the patient is semi-comatose).

There is a moderate elevation in the potassium concentration, which is presumably due to the acidaemia.

There is a marked increase in the anion gap (elsewhere the concentration of plasma proteins were normal): increase in concentration of unmeasured anions.

Possible causes include lactic acidosis, uraemic acidosis, diabetic ketoacidosis and ingestion of ethylene glycol. Given the presenting history, ingestion of ethylene glycol (as antifreeze) is the most likely cause.

Possible cause
Hypocalcaemia. Calcium binding to oxalic acid (a metabolite of ethylene glycol) in the renal tubules leads to calcium oxalate crystalluria and increased renal loss of calcium.

Urinalysis findings
Calcium oxalate crystalluria (six-sided elongated crystals) appears 3 to 6 hours postingestion of ethylene glycol and, although not consistently present, if found, they are highly suggestive of ethylene glycol toxicity.

Case 2
The blood pH is increased: alkalaemia.

The concentration of bicarbonate is increased: metabolic alkalosis.

Hyponatraemia.

Hyponatraemia and increased respiratory alkalosis.

There is an increase in the ratio between the concentration of bicarbonate and the pCO$_2$. The compensation for the metabolic alkalosis is inadequate.

The blood pH is decreased; acidemia.

Case 3
The blood pH is decreased: acidaemia.

The HCO$_3^-$ concentration is decreased: metabolic acidosis.

The pCO$_2$ is decreased: respiratory alkalosis (the compensation for the metabolic acidosis is incomplete).

Hyponatraemia and parallel hypochloraemia.

There is a marked increase in the anion gap (elsewhere the concentration of plasma proteins were normal): increase in concentration of unmeasured anions.

Possible causes include lactic acidosis, uraemic acidosis, diabetic ketoacidosis and ingestion of ethylene glycol. Given the presenting history and blood glucose concentration, diabetic ketoacidosis is likely.

Dilutional hyponatraemia occurs due to a shift in water, by osmosis, from the extracellular to the intracellular compartment. This occurs by transfer of water to the intracellular compartment, driven by osmotic pressure gradient. The osmotic pull is due to increase in plasma glucose.

Ingestion of ethylene glycol (or similar) is the most likely cause.

Urinary findings
Hydroxybutyric acid, ketoclastic acid, and excess renal loss of calcium.

Ethylene glycol (or similar) in the renal tubules leads to calcium oxalate crystalluria.

The kidneys are affected by the ethylene glycol toxicity.

Evidence of renal impairment includes increased urine specific gravity, increased urine osmolality, increased urine volume, and decreased urine pH.

Case 1
The presentation in Case 1 is consistent with diabetic ketoacidosis (DKA).

The patient was found in a semi-comatose state with laboured breathing, dehydrated, with characteristic ketones in the urine. The patient was hypoglycaemic, with a low blood glucose concentration. The dog was presented for x-ray and a foreign body was found in the proximal small intestine.

The diagnosis is made with measurement of blood glucose concentration. This is critical in the management of patients with DKA, as hypoglycaemia is common and can lead to severe neurological complications.

Case 2
The presentation in Case 2 is consistent with metabolic alkalosis (MA).

The patient was presented with signs of hyperventilation and increased respiratory rate. The blood pH was elevated, with a decreased concentration of bicarbonate. The patient was normotensive and had increased respiratory rate.

The diagnosis is made with measurement of blood pH and bicarbonate concentration. This is critical in the management of patients with MA, as hyperventilation can lead to increased respiratory rate and decreased blood pH.

Case 3
The presentation in Case 3 is consistent with diabetic ketoacidosis (DKA) and hypovolaemic shock.

The patient was presented with signs of hyperglycaemia, dehydration, and hypovolaemic shock. The blood glucose concentration was elevated, with a decreased concentration of bicarbonate. The patient was hypotensive and had increased respiratory rate.

The diagnosis is made with measurement of blood glucose concentration and blood pressure. This is critical in the management of patients with DKA, as hyperglycaemia and hypovolaemic shock can lead to severe neurological and cardiovascular complications.