

Data 1

The medical registrar shows you some bloods from a patient who has a long-standing ileal conduit and presented with abdominal pain and vomiting to the surgeons.

		Reference range
Sodium	143 mmol/L	132–144 mmol/L
Potassium	4.4 mmol/L	3.5–5.5 mmol/L
Urea	9.8 mmol/L	3.5–7.4 mmol/L
Creatinine	162 µmol/L	62–106 µmol/L
Calcium	1.89 mmol/L	2.10–2.55 mmol/L
Corrected calcium	2.37 mmol/L	2.15–2.65 mmol/L
Glucose	5.1 mmol/L	4.0–5.9 mmol/L
Alkaline phosphatase	48 IU/L	40–129 IU/L
Albumin	16 g/L	34–48 g/L
Phosphate	1.69 mmol/L	0.7–1.4 mmol/L
Haemoglobin	7.2 g/dL	11.5–16.5 g/dL
Haematocrit	0.22	
FiO ₂	0.21	
pH	7.3	7.35–7.45
pO ₂	11.9 kPa	10.0–14 kPa
pCO ₂	4.6 kPa	4.4–5.9 kPa
BE	–8.4 mEq/L	–2–+2 mEq/L
Lactate	2.9 mmol/L	0–2 mmol/L
Chloride	114 mmol/L	95–105 mmol/L
Bicarbonate	17.6 mmol/L	22–28 mmol/L

1. What do you make of these blood gases? What is the anion gap (AG)?
This is an example of metabolic acidosis. The chloride and lactate levels are elevated. [1]
You need to calculate the anion gap (AG), the principle of electroneutrality:
 $(\text{Na}^+ + \text{K}^+) - (\text{Cl}^- + \text{HCO}_3^-)$ is $(143 + 4.4) - (114 + 17.6) = 15.8$ (normal AG acidosis).
[1]
The normal value is 12–16 mEq/L – the difference is mainly due to the unmeasured negative charge on the proteins, sulphates and phosphates.

2. Is the albumin level significant?

Albumin is the major unmeasured anion and contributes almost the whole of the value of the AG. Low albumin will reduce the 'normal' gap. This should be commented on as it is relevant here. (Hypoproteinaemia is common in critical illness, albumin has a lot of negative charge.) A high AG acidosis in a patient with hypoalbuminaemia may appear as a normal AG acidosis if the low albumin is not corrected for. This albumin gap needs to be calculated as follows: [1]

The albumin gap = $40 - \text{albumin}$

The AG corrected value = $\text{AG} + (\text{albumin gap}/4)$.

It is generally accepted that the AG should be corrected upwards by 2.5 for every 10 g/L fall in the serum albumin. [1]

For this case, the albumin gap is $40 - 16 = 24$, making corrected AG $15.8 + (24/4) = 21.8$. This has changed an apparently normal AG acidosis into an increased AG acidosis.

3. Discuss the various causes of a normal and increased AG acidosis.

- Normal AG acidosis: [2 for most]
 - Disorders of bicarbonate homeostasis
 - Hyperchloraemia causes the acidosis
 - GI losses, vomiting, diarrhoea, renal losses, renal tubular acidosis, acetazolamide, iatrogenic (sodium chloride)
- Increased AG acidosis – increased 'unmeasured' anions: [2 for most]
 - Lactate, ketones, ethanol, aspirin, cyanide, methanol, ethylene glycol
- Reduced AG acidosis – increased 'unmeasured' cations (for completeness):
 - Rare
 - Hypermagnesaemia, lithium toxicity, excess protein, myeloma
 - Waldenström's macroglobulinaemia (immunoglobulins are strong cations)

4. What is the cause of this acidosis in this patient? How would we clarify this?

This is a mixed picture. There is hypoalbuminaemia which complicates things.

You need to look for a cause of the raised AG, probably related to under-resuscitation, i.e. lactate, but it's worth checking ketones if there is prolonged starvation, and serum vs. calculated osmolality if there is possible alcohol intoxication [2]

There is also hyperchloraemia. This may be iatrogenic following resuscitation but there will be an element of pre-existing derangement from the urinary diversion. [1]

5. How does the ileal conduit affect serum chloride?

The ileal conduit secretes bicarbonate into the lumen of the bowel in exchange for chloride. This results in bicarbonate loss from the body and excess chloride re-absorption. [2]

6. What is the strong ion difference (SID)? Explain.

The $\text{SID} = [\text{Na}^+] + [\text{K}^+] - [\text{Cl}^-]$; the normal value is 40 mmol/L. [1]

The pH, i.e. $[\text{H}^+]$, depends on the SID.

If you alter the value of SID, more or less water dissociates to maintain electroneutrality, hence altering $[\text{H}^+]$. Na^+ and K^+ are regulated strictly by other systems. The main 'metabolic regulator' is therefore Cl^- . [2]

In this case, $\text{SID} = 143 + 4.4 - 114 = 33.4$.

This would suggest that $(40 - 33.4 = 6.6)$ mmol/L of the base excess is attributable to the strong ion changes, namely a change in the Na:Cl ratio or hyperchloraemia.

7. What treatment would you suggest based on the arterial blood gases?

You need to ensure that tissue hypoperfusion is treated and corrected by seeing the base excess and lactate and urine output, etc. improve.

You may need to use flow haemodynamic monitoring to ensure good cardiac output in the face of hypotension. [2]

8. Would you administer bicarbonate? What are the potential problems?

As a proportion of this problem is due to electrolyte problems, then correction of this may be justified in the form of bicarbonate. Ensure that any hypoperfusion is addressed first as this will just mask some of the 'perfusion markers'. Sodium bicarbonate necessitates a large sodium load and is said to cause a paradoxical intracellular acidosis through increased CO₂ generation. [2]