

Acid- Base Balance + Arterial Blood Gases

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Clinical Skills 2 Teaching

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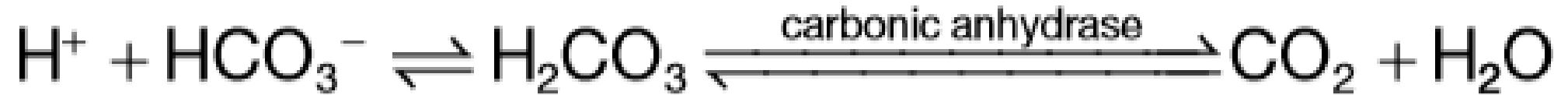
Learning Objectives for ABGs

- Understand the physiology behind acid-base regulation in the body
- Use of ABGs for assessment and monitoring
- Being able to interpret Arterial Blood Gas results
- Understanding how to measure the anion gap and its importance
- Practice examples

Physiological background of acid-base balance

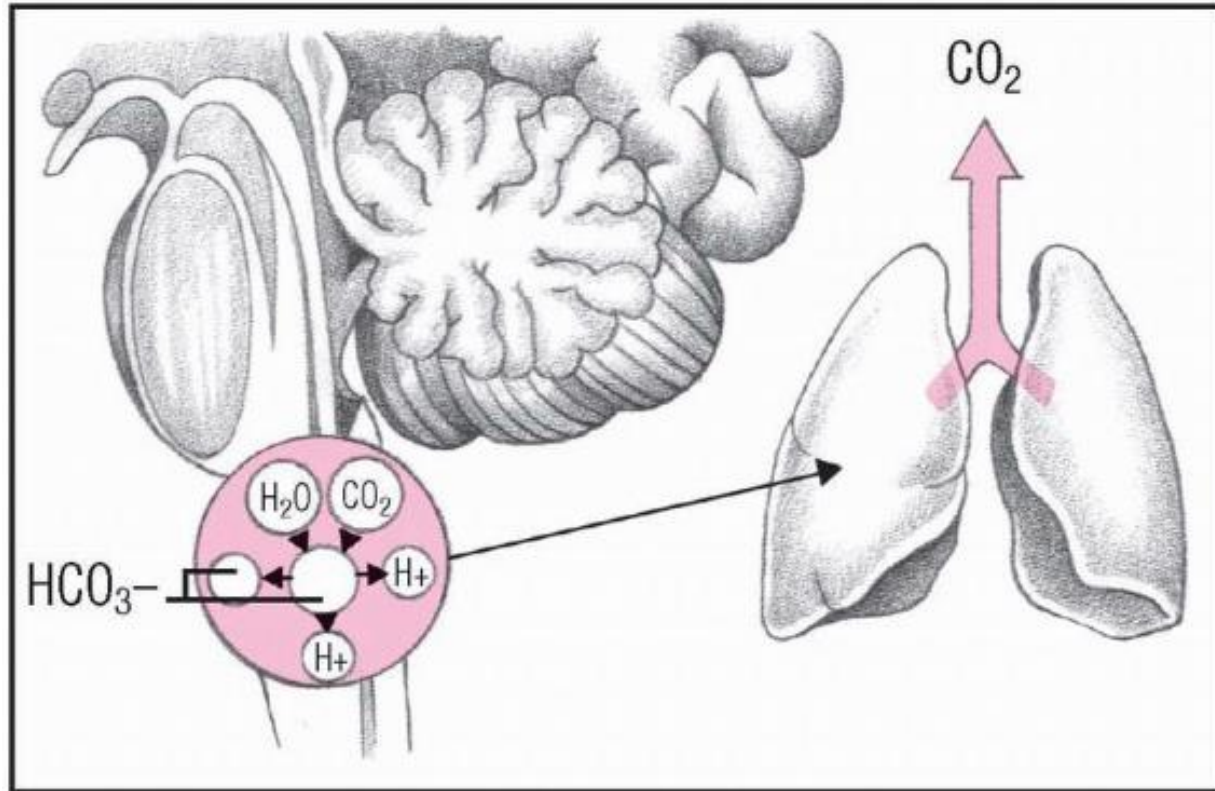
- You eat 70-100mmol of acid a day in your normal diet!
- Your pH in your blood is tightly controlled between 7.38-7.42 and relies on the H⁺ ions in the extracellular and intracellular fluid
- Acidosis is a pH below 7.38 (an increase in H⁺ ions)
- Alkalosis is a pH above 7.42 (a decrease in H⁺ ions)
- There are buffers in the body to maintain a tight blood pH such as haemoglobin (intracellular), calcium carbonate and phosphate (in your bone tissue) and hydration to carbon dioxide (bicarbonate-carbonic acid buffer pair)

Henderson-Hasselbalch equation



- An increase in H^+ ions (decreasing pH) drives the reaction to the right and so there is a drop in plasma HCO_3^- and an increase in the arterial pressure of CO_2 (PaCO_2 - measured in kilopascals)
- Acidosis (a drop in pH due to increase of H^+ ions OR drop in HCO_3^- ions) stimulates increased ventilation which blows off excess CO_2 generated

Exhalation of acidic CO_2 gas through the respiratory system



1) If respiratory rate increases what happens to the blood pH?

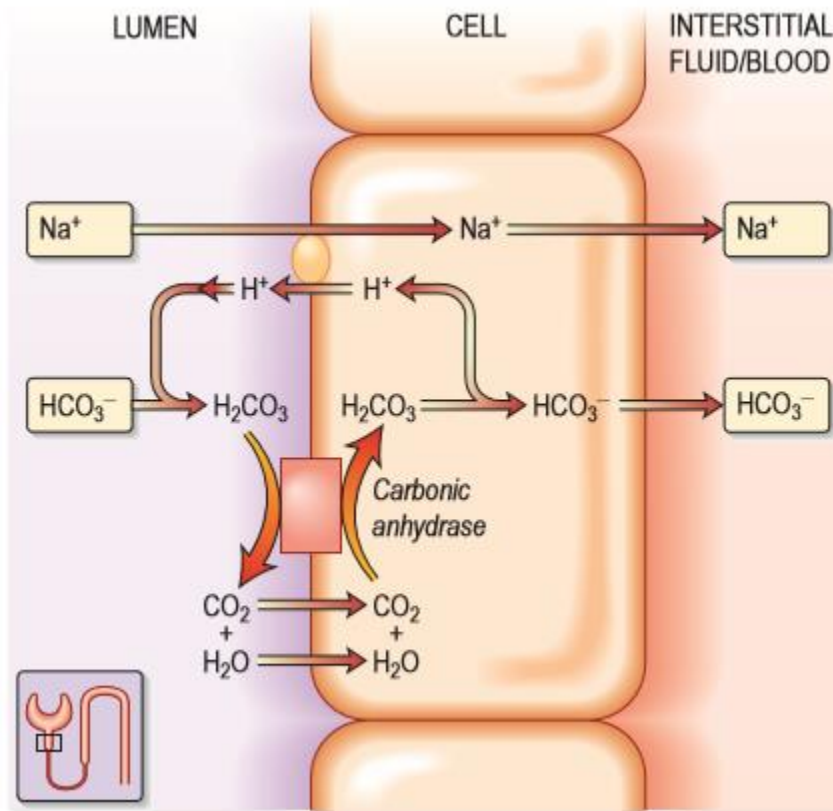
2a) If breathing is ineffective (slow or shallow) what happens to the blood CO₂?

b) Thus what happens to the blood pH?

Plasma HCO_3^- needs to be maintained at 25 mmol/L and increased ventilation doesn't replace the HCO_3^- ions used in the buffering process so....

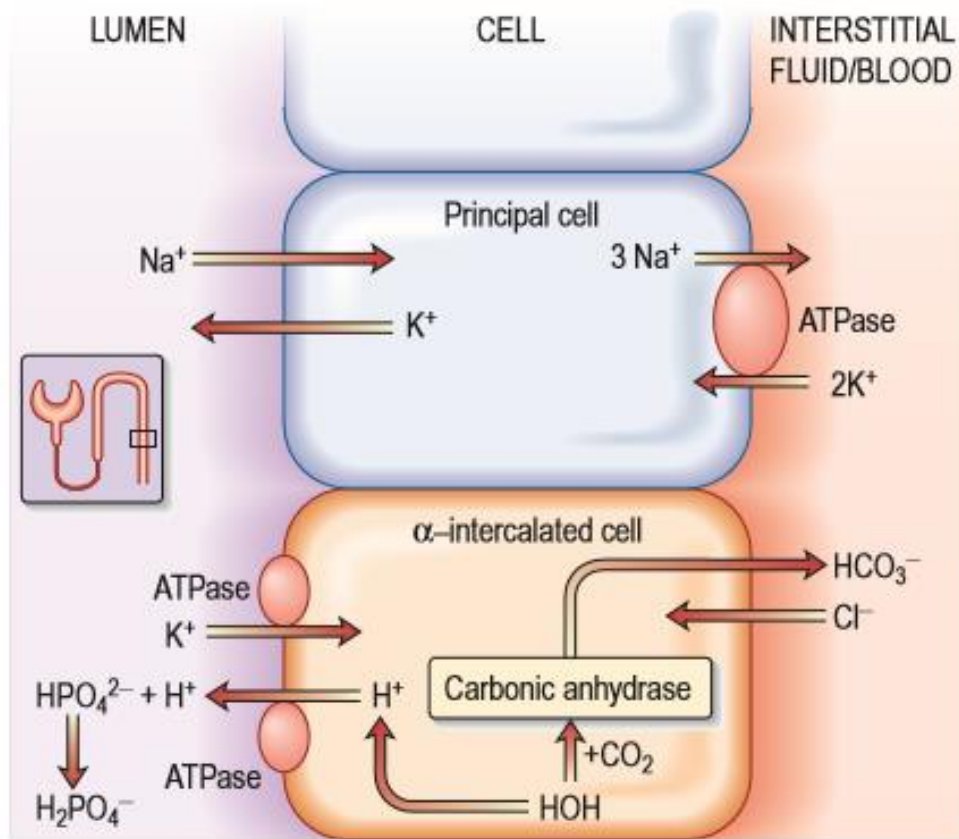
So.... which other organ is involved in regulating acid-base balance excreting excess H^+ ions and reabsorbing HCO_3^- ions?

Reabsorption of Sodium Bicarbonate in the kidney



- Exchange of sodium (Na^+) with hydrogen (H^+) on luminal wall in proximal tubule
- Hydrogen (H^+) and bicarbonate (HCO_3^-) form H_2CO_3 in the lumen
- Carbonic Acid (H_2CO_3) is broken down again by the carbonic anhydrase enzyme in the luminal wall into water (H_2O) and carbon dioxide (CO_2) which is absorbed intracellularly
- Once again the carbonic anhydrase enzyme in the luminal wall drives the reverse reaction to H_2CO_3 which breaks down to hydrogen (H^+) which is excreted and bicarbonate (HCO_3^-) which is reabsorbed into the blood

Excretion of Hydrogen in the kidney



- Hydrogen (H^+) ions from cortical collecting ducts is indirectly linked to the absorption of sodium (Na^+)
- Aldosterone enters 'Principal cells' in collecting duct and opens the Na^+ channel (increases activity of the Na^+/K^+ ATPase enzyme)
- Aldosterone also directly stimulates H^+ ATPase enzyme in the alpha-intercalated cell that enhances H^+ excretion

In Summary

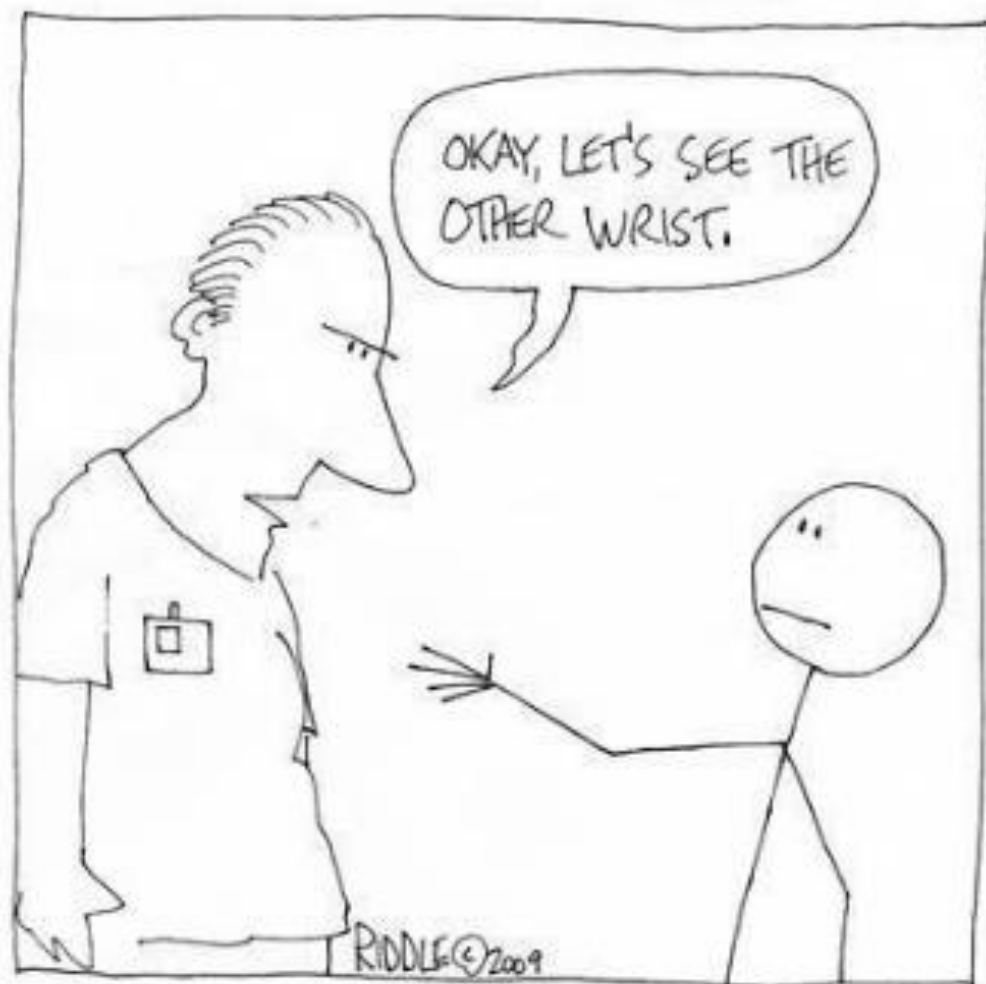
- Carbonic acid (H_2CO_3) links the respiratory and metabolic systems
- Advantage of this is that the components can vary independently of each other e.g. if respiratory system falters the metabolic system can compensate and vice versa
- Respiratory system controls carbon dioxide (CO_2) in the blood (which is an acidic gas)
- The kidneys (metabolic system) control level of sodium bicarbonate ($\text{Na}^+\text{HCO}_3^-$) in the blood (which is a base and buffer of hydrogen ions)
- Kidneys can further regulate the pH by excretion of hydrogen (H^+) ions

When to take an arterial blood gas?

Patients who are in:

1. Respiratory failure
2. Critically ill or likely to deteriorate quickly
3. Sepsis
4. Multiorgan failure
5. Uncontrolled diabetes
6. Taken an overdose or toxin

DON'T TAKE AN ABG UNLESS IT WILL CHANGE YOUR MANAGEMENT



THIS WAS GOING TO BE A TOUGH ABG.

Normal Arterial Blood Gases

| | |
|-------------------------------|---------------------------|
| pH | 7.35-7.45 |
| PaCO ₂ | 4.7-6.0 kPa (35-45 mmHg) |
| PaO ₂ | 11-13.5 kPa (83-102 mmHg) |
| HCO ₃ ⁻ | 22-28 mmol/L |
| Anion gap | 10-16 mmol/L |

Scenario 1 – A breathless man

A 60 year old man with a history of chronic obstructive pulmonary disease presents to the emergency department with increasing shortness of breath, fever, and a productive cough of yellow-green sputum. He is unable to speak in full sentences. His wife says he has been unwell for two days.

Measurement of arterial blood gas shows:

pH 7.2 (Normal range 7.35-7.45)
O₂ 7.9 kPa (59 mmHg) (Normal range 11-13.5kPa/ 83-102mmHg)
PaCO₂ 9.3 kPa (70 mmHg) (Normal range 4.7-6.0kPa / 35-45mmHg)
HCO₃⁻ 27 mmol/L (Normal range 22-28 mmol/L)

How would you interpret this ABG?

Methodology to interpret ABG results

Step 1: Is there an acid-base abnormality—that is, is the pH outside the normal range?

Step 2: Is the respiratory (PaCO_2) or the metabolic (HCO_3^-) component abnormal, and is it the primary cause of the acid-base abnormality—that is, does it tell the same story as the pH?

Step 2a: If there is metabolic acidosis—that is, a low pH and a low HCO_3^- , it may be useful to take an additional step and calculate the anion gap.

Step 3: Is there compensation?

Step 4: What does the PaO_2 tell you?

Step 4 - What does the PaO₂ tell you?

- Remember PaO₂ does NOT affect acid-base balance
- If a patient has hypoxaemia if PaO₂ less than 85mmHg on room air (although not clinically important unless below 65mmHg) classified as respiratory failure
- Type I respiratory failure is when patient has hypoxaemia in absence of hypercapnia (no raised PaCO₂)
- **Type II respiratory failure** is when patient has hypoxaemia in presence of hypercapnia (**raised PaCO₂**) – this indicates *hypoventilation*

Anion gap and its use in Metabolic Acidosis

$$\text{Anion gap} = (\text{Na}^+ + \text{K}^+) - (\text{Cl}^- + \text{HCO}_3^-) = 10-16 \text{ mmol/L}$$

- The number of anions and cations should be equal, but a standard blood analysis does not measure them all, resulting in a difference of 10-16 mmol/L. Some causes of metabolic acidosis result in an increased anion gap and others result in a normal anion gap.

There is *no increase in anion gap* when there is a *loss of bicarbonate*. It is usually associated with a concomitant rise in plasma chloride including:

- Renal tubular acidosis
- Severe diarrhoea (intestinal secretions below the stomach contain a large amount of bicarbonate)

Causes of Metabolic Acidosis and an *Increased* Anion Gap

An increase in anion gap (**over 16 mmol/L**) occurs when there is increased production of organic acids, such as ketones and lactic acid, or reduced excretion of them including:

- Lactic acidosis—shock, infection, tissue ischaemia
- Ketoacidosis—diabetes mellitus, alcohol abuse
- Urate—renal failure
- Poisoning— aspirin, metformin, methanol

Scenario 2

A six year old boy is taken to the ER department with vomiting and a decreased level of consciousness. His breathing is slow and deep (Kussmaul breathing), and he is lethargic and irritable in response to stimulation. He appears to be dehydrated—his eyes are sunken and mucous membranes are dry—and he has a two week history of polydipsia, polyuria, and weight loss.

Scenario 2 – ABG result

Measurement of arterial blood gas (ABG) shows:

| | |
|---|-----------------------------|
| pH 7.2 | (Normal range 7.35-7.45) |
| PaO ₂ 13.3 kPa (100 mm Hg) | (Normal range 11-13.5 kPa) |
| PaCO ₂ 3.3 kPa (25 mm Hg) | (Normal range 4.7-6.0 kPa) |
| HCO ₃ ⁻ 10 mmol/L | (Normal range 22-28 mmol/L) |

Other results are Na⁺ 126 mmol/L, K⁺ 5 mmol/L, and Cl⁻ 95 mmol/L.

- 1) What is your assessment of the ABG?
- 2) What is the anion gap?

$$\text{Anion gap} = (\text{Na}^+ + \text{K}^+) - (\text{Cl}^- + \text{HCO}_3^-) = 10-16 \text{ mmol/L (normal range)}$$
$$\text{Anion gap} = (126 + 5) - (95 + 10) = 26$$

Scenario 3

A 12 year old girl attends the emergency department after falling and hurting her arm. In triage she is noted to be tachycardic and tachypnoeic. She is given some pain killers. While waiting to be seen by the doctor, she becomes increasingly hysterical, complaining that she is still in pain and now experiencing muscle cramps, tingling, and paraesthesia.

Scenario 3 – ABG result

Measurement of arterial blood gas shows

| | |
|---|------------------------------|
| pH 7.5 | (Normal range 7.35-7.45) |
| PaO ₂ 15.3 kPa (115 mm Hg) | (Normal range 11.0-13.5 kPa) |
| PaCO ₂ 3.9 kPa (29 mm Hg) | (Normal range 4.7-6.0 kPa) |
| HCO ₃ ⁻ 24 mmol/L | (Normal range 22-28 mmol/L) |

What does this result show?

Summary of pH, PaCO₂ and Bicarbonate

| Primary condition | pH | PaCO ₂ | Bicarbonate |
|-----------------------|----|-------------------|-------------|
| Respiratory acidosis | ↓ | ↑ | (↑) |
| Respiratory alkalosis | ↑ | ↓ | (↓) |
| Metabolic acidosis | ↓ | (↓) | ↓ |
| Metabolic alkalosis | ↑ | (↑) | ↑ |

How do we use ABGs for assessment and monitoring?

- Monitoring oxygen for COPD patients – concern regarding CO₂ retention if giving oxygen and changing rate of oxygen flow
- Assessment for Long Term Oxygen Therapy (LTOT) of COPD patients – do they meet the criteria for LTOT?
- Assessment of how ill someone is and monitoring required – e.g. DKA and need to go on ICU or step down to HDU bed...
- Correction of acid-base of patient on ICU e.g. use of bicarbonate or increasing the respiratory rate on the ventilator

Practice Examples of ABGs

1a. What does the following ABG show?

pH 7.48 (7.35-7.45)

pCO₂ 2.9 kPa (4.9-6.1)

pO₂ 15.5 kPa (10-13.1)

HCO₃ 17 (22-28)

1b. Do you know any causes of this?

Causes of Metabolic Alkalosis

- Loss of gastric secretions - vomiting, nasogastric suction.
- Loss of colonic secretions – chloride losing diarrhoea, villous adenoma.
- Thiazides and loop diuretics (after discontinuation)
- Posthypercapnia
- Cystic fibrosis
- Urinary loss of chloride – high blood pressure, adrenal adenoma, renovascular disease, glucocorticoid remediable aldosteronism, renin secreting tumour, Cushing's syndrome, liquorice ingestion....

2a What does this ABG show?

| | | |
|------------------|--------|---------------|
| pH | 7.20 | (7.35-7.45) |
| pCO ₂ | 92mmHg | (35-45mmHg) |
| pO ₂ | 76mmHg | (75-100mmHg) |
| HCO ₃ | 21 | (22-28) |

2b Do you know any causes of this condition?

Causes of Respiratory Acidosis

- Hypoventilation
- COPD
- Airway obstruction
- Chest trauma
- Drug overdose
- Pulmonary oedema
- Neuromuscular disease

Clinical Scenario

72 year old Yusuf Abu Raif is a lifelong smoker and takes a Ventolin and Spiriva (Tiotropium) inhaler. Over the last 3 days he has become increasingly breathless and has a productive cough. On arrival in the ER department he is found to have an oxygen saturation of 75% and is put on 100% oxygen at 12L/min. His initial ABG is below and then it is repeated after 2 hours after he becomes increasingly confused.

| Initial ABG on air | | After 2 hours on 100% oxygen | |
|--------------------|---------------------|------------------------------|--------|
| pH | 7.44 | pH | 7.20 |
| paO ₂ | 52mmHg (75-100mmHg) | paO ₂ | 70mmHg |
| paCO ₂ | 63mmHg (35-45mmHg) | paCO ₂ | 80mmHg |
| HCO ₃ | 42 (22-28) | HCO ₃ | 48 |

What do these results show has happened?

Metabolic Acidosis

Diabetic Ketoacidosis
Diarrhoea
Renal failure
Shock
Aspirin overdose
Sepsis

Metabolic Alkalosis

Loss of gastric secretions
Overuse of antacids
K⁺ wasting diuretics

Respiratory Acidosis

Hypoventilation
COPD
Airway obstruction
Drug overdose
Chest trauma
Pulmonary oedema
Neuromuscular disease

Respiratory Alkalosis

Hyperventilation
Hypoxia
Anxiety
High altitude
Pregnancy
Fever