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Arterial blood gases - indications and interpretation

What do arterial blood gases check for?

Arterial blood gases (ABGs) are an important routine investigation to monitor the acid-base balance of patients.[1] They may help make a diagnosis, indicate the severity of a condition and help to assess treatment. ABGs provide the following information:

- Oxygenation.
- Adequacy of ventilation.
- Acid-base levels.

Also see the full separate Acid-base balance article.

Blood pH

Blood pH has to be maintained within a tight normal range to avoid cellular death. This can be achieved by buffer mechanisms which can be either renal or respiratory in nature.[1]

Metabolic problems will require respiratory compensation and this occurs rapidly - eg, by increasing ventilation to blow off CO_2. On the other hand respiratory problems leading to acid-base abnormalities require renal compensation. This is slow and may need secretion of H^+ ions or reabsorption/new production of HCO_3^- ions.[2]

Indications

Obtaining a venous sample for analysis is less painful and often easier than arterial blood gas sampling.
Compared to arterial sampling, venous blood gases provide clinically interchangable measurements of pH and bicarbonate. Venous pCO2 measurements do not provide a reliable indicator of arterial pCO2, although a venous pCO2 ≤ 45 mm Hg (6 kPa) excludes clinically significant hypercarbia.[3]
Venous blood gas sampling is therefore a suitable alternative for many of the traditional indications for arterial blood gases, although it remains the best way to accurately determine arterial pO2 and pCO2.

- Respiratory failure - in acute and chronic states.
- Any severe illness which may lead to a metabolic acidosis - for example:
    - Cardiac failure.
    - Liver failure.
    - Renal failure.
    - Hyperglycaemic states associated with diabetes mellitus.
    - Multiorgan failure.
    - Sepsis.
    - Burns.
    - Poisons/toxins.
- Ventilated patients.
- Sleep studies.
- Severely unwell patients from any cause - affects prognosis.

Procedure

- Arterial blood can be obtained by direct arterial puncture most usually at the wrist (radial artery). Alternatives to the radial artery include the femoral and brachial artery - both of which are usually used in emergency settings. The dorsalis pedis artery and ulnar artery may also be used. It is important to ensure good collateral circulation (see below), as there is a theoretical risk of thrombus occlusion.
- If multiple samples are required then an indwelling arterial cannula can be placed.
- If the patient is on oxygen, allow them to titrate with the oxygen for 5-10 minutes (30 minutes if they have chronic obstructive pulmonary disease (COPD)) before taking a sample.
- If the radial artery is to be used, perform Allen's test to confirm collateral blood flow to the hand.

Allen's test

- Elevate the hand and make a fist for approximately 30 seconds.
- Apply pressure over the ulnar and the radial arteries occluding both (keep the hand elevated).
- Open the hand which will be blanched.
- Release pressure on the ulnar artery and look for perfusion of the hand (this takes under eight seconds).
- If there is any delay then it may not be safe to perform radial artery puncture.

- Explain the procedure to the patient - it is painful.
- Local anaesthetic makes the procedure less painful.[4] It should be used routinely, unless the short delay from doing so is genuinely clinically important (eg, in a true emergency).[5]
- ABG syringes usually come prepacked and are heparinised. Some contain a vacuum and thus the plunger does not always need to be pulled. (Check with your department as to which they use).

- The wrist is extended - a pillow under the hand may improve comfort.
- Palpate the artery and hold fingers firmly over the pulsation.
- Then introduce the needle at a 45° angle slowly with the bevel facing upwards, aiming for the point of maximum pulsation.
- Once you hit the artery, try to obtain at least a 1 ml sample.
- Once you have taken your sample and withdrawn the needle, apply firm pressure for a minimum of two minutes (longer if the patient is on any antiplatelet medication or anticoagulants).

How to interpret arterial blood gases

The following indices should be looked at in the following order (see local laboratory for reference ranges):

- Blood pH - high indicates alkalosis, low indicates acidosis and normal indicates either normal, mixed defect or a compensated defect.
- $PaCO_2$ level - is it a respiratory problem? If not, look at the bicarbonate level. High $PaCO_2$ with an acidosis indicates a respiratory problem. If the $PaCO_2$ is normal or low it indicates compensation.
- Bicarbonate - if the bicarbonate fits with the pH it suggests a primary metabolic problem. If not, it indicates compensatory changes.
- Look for any compensation - eg, low $PaCO_2$ in severe metabolic acidosis.
- Anion gap in metabolic acidosis - see below under 'Other useful information from arterial blood gases'.
- O_2 level - is hypoxaemia present?

Other useful information from arterial blood gases

Alveolar-arterial oxygen gradient - (A-a)pO_2; difference in oxygen partial pressures between the alveolar and arterial side.[6] It provides a measure of oxygen diffusion across the alveoli into the blood. Thus, will be impaired in lung disease such as COPD.[7] Raised (A-a)pO_2 may also represent the presence of an intrapulmonary shunt, ie a lung that is perfused but not ventilated - for example, pneumonia. The following table provides a list of some of the causes in which (A-a)pO_2 change:

(A-a) pO_2	
Normal (A-a)pO_2 in type 2 respiratory failure	Raised (A-a)pO_2
Central nervous system (CNS) depression. Neuromuscular disorders.	Intrinsic lung disease - eg, COPD.

Anion gap - this is useful in any cause of metabolic acidosis. In plasma, the sum of the cations (sodium plus potassium) is normally greater than that of the anions (chloride plus bicarbonate) by approximately 14 mmol/L (normal range 10-18 mmol/L). This is known as the anion gap. In some disorders, either the positive or negative ions may increase, leading to a change in the anion gap. The following table lists the causes of an abnormal anion gap:

Causes of changes in anion gap	
Raised anion gap metabolic acidosis	Normal anion gap (hyperchloraemia) metabolic acidosis
Accumulation of acids - for example: Ketoacids in diabetic ketoacidosis (DKA). Lactic acid - eg, shock, infection. Drugs/toxins - eg, salicylates, ethylene glycol, methanol.	Loss of bicarbonate or ingestion of acid - for example: Gastrointestinal tract causes - eg, diarrhoea, pancreatic fistula. Renal tubular acidosis. Addison's disease. Drugs - eg, carbonic anhydrase inhibitors.

Causes of a raised anion gap metabolic acidosis can be recalled using the 'MUDPILES' mnemonic (methanol, uraemia, DKA, paraldehyde, infection/ischaemia/isoniazid, lactic acidosis, ethylene glycol/ethanol, salicylates/starvation).

Primary acid-base disturbances

- **Respiratory acidosis**: low pH, high $PaCO_2$, normal or high normal bicarbonate.
  Causes: neuromuscular weakness, intrinsic lung disease - eg, COPD.
- **Respiratory alkalosis**: high pH, low $PaCO_2$, normal or high normal bicarbonate.
  Causes: any cause of hyperventilation - eg, anxiety, pain.
- **Metabolic acidosis**: low pH, normal or low normal $PaCO_2$, low bicarbonate.
  Causes: see anion gap table, above.
- **Metabolic alkalosis**: high pH, normal $PaCO_2$, high bicarbonate.
  Causes: vomiting, burns, ingestion of base.

Mixed disorders

Mixed acid-base disorders occur when there is a combination of primary acid-base disturbances (but not combined respiratory acidosis and alkalosis). Usually the ABG result does not fit into one of the above four clinical pictures easily. The therapy is directed towards correction of each primary acid-base disturbance.[8]

Further reading

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