**ABG interpretation**

Remember: \( \text{H}_2\text{O} + \text{CO}_2 \leftrightarrow \text{H}_2\text{CO}_3 \leftrightarrow \text{HCO}_3^- + \text{H}^+ \)

**STEP 1 – Oxygenation**

1. Assess oxygenation
   - Hypoxic?
   - Significant alveolar-arterial gradient?
     - Alveolar-arterial gradient = partial pressure of oxygen in airways (PAO\(_2\)) – partial pressure of oxygen in artery (PaO\(_2\))
     - Where \( \text{PAO}_2 = \text{FiO}_2 – (\text{PaCO}_2 / \text{kQ}) \)
     - Where kQ (constant) = 0.8

**STEPS 2-5**

2. Determine pH status
   - Acidosis (pH↓)
   - Alkalosis (pH↑)

3. Determine respiratory component (PaCO\(_2\))
   - Respiratory acidosis (pH↓, PaCO\(_2\)↑)
   - Respiratory alkalosis (pH↑, PaCO\(_2\)↓)
   - If the PaCO\(_2\) doesn’t agree with the pH, ignore it until step 5

4. Determine the metabolic component (HCO\(_3^-\) or BE)
   - Metabolic acidosis (pH↓, HCO\(_3^-\)↓)
   - Metabolic alkalosis (pH↑, HCO\(_3^-\)↑)
   - If the HCO\(_3^-\) doesn’t agree with the pH, ignore it until step 5

5. Combine
   - Primary disturbance: you know this from the above steps
     - NB. both the respiratory and metabolic component may have agreed with the pH ie. a ‘mixed respiratory and metabolic acidosis/alkalosis’
   - Compensation: If either the respiratory/metabolic component was not consistent with the pH, there is compensation...
     - Acidosis may be compensated by
       - Respiratory compensation: increasing respiratory rate to blow off CO\(_2\) (will result in ↓CO\(_2\))
       - Metabolic compensation: increased bicarbonate production by kidney (will result in ↑HCO\(_3^-\))
     - Alkalosis may be compensated by
       - Respiratory compensation: decreasing respiratory rate to retain CO\(_2\) (will result in ↑CO\(_2\))
       - Metabolic compensation: decreased bicarbonate production by kidney (will result in ↓HCO\(_3^-\))
   - Partial compensation = pH not quite back to normal yet; full compensation = pH normal (you cannot over-compensate)
     - NB. You can’t fully compensate metabolic alkalosis (you can only hypoventilate a bit)
     - NB. metabolic compensation by the kidneys takes 3 days, respiratory compensation is fast.
   - ± Respiratory failure (type 1 = 1 gas abnormal (↓O\(_2\)); type 2 = 2 gasses abnormal (↓O\(_2\) + ↑CO\(_2\)))

**Normal values:**
- pH: 7.35-7.45
- pCO\(_2\): 4.5-6.0 kPa
- pO\(_2\): 11-13 kPa
- HCO\(_3^-\): 22-26 mmol/l
- BE: -2 to +2
- SaO\(_2\): >95%
- PaO\(_2\) should ~ FiO\(_2\) % - 10
- Alveolar arterial gradient: <10 (increased in emphysema, fibrosing alveolitis, age)
- Lactate <2
Causes of Acid-Base Mismatch

<table>
<thead>
<tr>
<th>Respiratory</th>
<th>Acidoses</th>
<th>Alkaloses</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypoventilation in:</td>
<td>- Lung disease (COPD, severe asthma attack, pulmonary oedema)</td>
<td>Hyperventilation in:</td>
</tr>
<tr>
<td>- CNS depression</td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Mechanical lung dysfunction (e.g. obesity, guillain barre, myasthenia gravis)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

| Metabolic | | |
| Check anion gap = (Na⁺ + K⁺) – (Cl⁻ + HCO₃⁻) | Acidosis in: | |
| Normal = 3-12 | - Chloride responsive: vomiting, diuretics, corticosteroids | |
| INCREASED ANION GAP = new acid added to body | - Chloride resistant: any hyperaldosteronism state (e.g. Cushing’s, hypokalaemia) | |
| (MUDPILES: Methanol, Uraemia, DKA, Propylene glycol, Iron/isoniazid, Lactate, Ethylene glycol, Salicylates) | |
| NORMAL ANION GAP = retaining H⁺ (renal tubular acidosis, Addison’s) or losing HCO₃⁻ (diarrhoea) | |

Causes of Respiratory Failure

TYPE 1 = 1 gas abnormal = ↓O₂, normal CO₂
Caused by ventilation-perfusion (V/Q) mismatch ie. either:
- Low V/Q: areas of lung are perfused with deoxygenated blood but not ventilated with oxygen (ie. airway obstruction)
  - Causes: mucus plug in asthma/COPD, airway collapse in emphysema
- High V/Q: areas of lung are ventilated with oxygen but not perfused with deoxygenated blood (ie. block in blood flow)
  - Causes: PE

The reason CO₂ is normal is that the areas of the lung which are perfused and ventilated can blow off extra CO₂ by increasing ventilation rate (making CO₂ low in this area and high in the area with V/Q mismatch which makes it normal overall). Extra oxygen, however, cannot be absorbed (without giving a higher oxygen concentration) because the maximum amount of oxygen diffuses across the alveolar membrane in normal circumstances anyway.

TYPE 2 = 2 gasses abnormal = ↓O₂, ↑CO₂
Caused by alveolar hypoventilation. This means oxygen cannot get into alveoli and carbon dioxide cannot get out.
Causes: obstructive lung diseases (e.g. COPD), restrictive lung diseases, decreased respiratory drive, neuromuscular disease, thoracic wall disease

Lactic Acidosis

- Lactic acid = a product of anaerobic metabolism
- Types of lactic acidosis
  - TYPE 1 (hypoxic) = produce too much lactic acid (e.g. DKA, starvation, cardiovascular/respiratory depression)
  - TYPE 2 (non-hypoxic) = cannot break down lactic acid (e.g. secondary to metformin or poisoning)
- LDH = an enzyme involved in anaerobic metabolism pathway (increased in tissue breakdown/turover e.g. muscle trauma, MI, stroke, haemolysis, cancer, acute pancreatitis, HIV, meningitis/encephalitis)

Common ABG patterns

<table>
<thead>
<tr>
<th>Situation</th>
<th>pH</th>
<th>CO₂</th>
<th>HCO₃⁻</th>
<th>O₂</th>
<th>Details</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypoventilation</td>
<td>↑</td>
<td>↓</td>
<td>N</td>
<td>↑</td>
<td>Respiratory alkalosis (lungs overdrive)</td>
</tr>
<tr>
<td>Stable chronic COPD</td>
<td>N</td>
<td>↑</td>
<td>↑</td>
<td>↓</td>
<td>Fully compensated respiratory acidosis + T2 RF (hypoxic drive)</td>
</tr>
<tr>
<td>Acute COPD exacerbation</td>
<td>↓</td>
<td>↑↑</td>
<td>↑</td>
<td>↓↓</td>
<td>Partially compensated respiratory acidosis + T2 RF</td>
</tr>
<tr>
<td>Stable asthmatic</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>Respiratory alkalosis (wheeze → anxiety → hyperventilation)</td>
</tr>
<tr>
<td>Decreased respiratory drive (life-threatening asthma, opiate OD)</td>
<td>↓</td>
<td>↑</td>
<td>N</td>
<td>↓</td>
<td>Respiratory acidosis + T2 RF (lungs give up breathing, Hypoxia comes first)</td>
</tr>
<tr>
<td>Pulmonary fibrosis</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>↓</td>
<td>Isolated T1 RF (despite hypoxia, there is no increase in respiratory rate. Likley due to chronic hypoxia desensitisation)</td>
</tr>
<tr>
<td>P.E.</td>
<td>↑</td>
<td>↓</td>
<td>N</td>
<td>↓</td>
<td>Respiratory alkalosis + T1 RF (hypoxia drives respiratory rate up causing excess CO₂ excretion. Can blow off extra CO₂ in perfused lung to compensate but can’t absorb extra oxygen)</td>
</tr>
</tbody>
</table>

NB. Respiratory alkalosis ΔΔ = hyperventilation (↑O₂), asthma exacerbation (normal O₂), PE (↓O₂)