ABG interpretation

Remember: $H_2O + CO_2 \leftrightarrow H_2CO_3 \leftrightarrow HCO_3^- + H^+$

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

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 $PAO_2 = FiO_2 – (PaCO_2 / kQ)$
      Where kQ (constant) = 0.8

STEPS 2-5

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

3. Determine **respiratory component (PaCO2)**
    - 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 (HCO3- 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))

Causes of Acid-Base Mismatch

	Acidosis	Alkalosis
Respiratory	**Hypoventilation** in: -Lung disease (COPD, severe asthma attack, pulmonary Oedema) -CNS depression -Mechanical lung dysfunction (e.g. obesity, guillain barre, myasthenia gravis)	**Hyperventilation** in: -Anxiety -Hypoxia -Acute pulmonary insult (e.g. PE, pneumonia, asthma attack, pulmonary oedema)
Metabolic	**Check anion gap = ($Na^+ + K^+$) – ($Cl^- + HCO_3^-$)** *Normal = 3-12* **INCREASED ANION GAP** = new acid added to body (**MUDPILES**: **M**ethanol, **U**raemia, **D**KA, **P**ropylene glycol, **I**ron/isoniazid, **L**actate, **E**thylene glycol, **S**alicylates) **NORMAL ANION GAP** = retaining H^+ (renal tubular acidosis, Addison's) or loosing HCO_3^- (diarrhoea)	**Acid loss** in: -Chloride responsive: vomiting, diuretics, corticosteroids -Chloride resistant: any hyperaldosterone state (e.g. Cushing's, hypokalaemia)

Causes of Respiratory Failure

TYPE 1 = 1 gas abnormal = ↓O_2, normal CO_2
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 CO_2 is normal is that the areas of the lung which are perfused and ventilated can blow off extra CO_2 by increasing ventilation rate (making CO_2 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 = ↓O_2, ↑CO_2
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 poisening)
- LDH = an enzyme involved in anaerobic metabolism pathway (increased in tissue breakdown/turnover e.g. muscle trauma, MI, stroke, haemolysis, cancer, acute pancreatitis, HIV, meningitis/encephalitis)

Common ABG patterns

Situation	pH	CO_2	HCO_3^-	O_2	Details
Hyperventilation	↑	↓	N	↑	Respiratory alkalosis (lungs overdrive)
Stable chronic COPD	N	↑	↑	↓	Fully compensated respiratory acidosis + T2 RF (hypoxic drive)
Acute COPD exacerbation	↓	↑↑	↑	↓↓	Partially compensated respiratory acidosis + T2 RF
Stable asthmatic	N	N	N	N	
Asthma exacerbation	↑	↓	N	N/↑	Respiratory alkalosis (wheeze → anxiety → hyperventilation)
Decreased respiratory drive (life-threatening asthma, opiate OD)	↓	↑	N	↓	Respiratory acidosis + T2 RF (lungs give up breathing. Hypoxia comes first)
Pulmonary fibrosis	N	N	N	↓	Isolated T1 RF (despite hypoxia, there is no increase in respiratory rate. Likley due to chronic hypoxia desensitisation)
P.E.	↑	↓	N	↓	Respiratory alkalosis + T1 RF (hypoxia drives respiratory rate up causing excess CO_2 excretion. Can blow off extra CO2 in perfused lung to compensate but can't absorb extra oxygen!)

NB. Respiratory alkalosis ΔΔ = hyperventilation (↑O_2), asthma exacerbation (normal O_2), PE (↓O_2)