ACID BASE DISTURBANCE

An approach to interpretation of blood gas:

- Is the patient acidaemic or alkalaemic?
  - <7.35 acidaemic
  - >7.45 alkalaemic

- What is the primary acid base disorder?

primary disorder	primary change	compensation
metabolic acidosis	↓ HCO_3	↓ $PaCO_2$
metabolic alkalosis	↑ HCO_3	↑ $PaCO_2$
respiratory acidosis	↑ $PaCO_2$	↑ HCO_3 acute = 0.75 rise in HCO3 every 1kPa ↑ PaCO2 chronic = 3.0 rise in HCO3 every 1kPa ↑ PaCO2
respiratory alkalosis	↓ $PaCO_2$	↓ HCO_3 acute = 1.5 fall in HCO3 every 1kPa ↓ PaCO2 chronic = 3.5 fall in HCO3 every 1kPa ↓ PaCO2

- Determine expected compensatory responses.
  If the actual and the expected response are different, there may be a secondary acid-base disturbance.

- Anion gap

$$AG = (Na^+ + K^+) - (Cl^- + HCO^{3-})$$

Normal = 8 – 16
Increased AG ⇒ increased AG metabolic acidosis
AG made up of unmeasured anions, consist of proteins, mainly albumin. Hence, hypoalbuminaemia can reduce the baseline anion gap so that patient may not have a high anion gap even in the presence of a disorder which usually produces an increased in anion gap. AG reduced by approx 2.5 mmol/L for every 10g/L fall in albumin

$$\text{Corrected AG} = 0.2(\text{albumin}) \times 1.5(\text{phosphate})$$

The fall in HCO_3 should be of comparative magnitude with the increase in AG. Otherwise, there is a co-existing normal AG acidosis or a metabolic alkalosis.

Respiratory acidosis and alkalosis is mentioned elsewhere under respiratory problems.

METABOLIC ACIDOSIS

Causes:

Increase anion gap (list not exhaustive)

- ketoacidosis – diabetic, alcoholic, starvation
- lactic acidosis – Type I and II
- renal failure
- salicylic acid
- paraldehyde
- formaldehyde
- ethanol
- methanol
- ethylene glycol

Normal anion gap (hyperchloraemic acidosis)

- Hypokalaemic – GI Loss (below pylorus)
  Renal tubular acidosis
- Hyperkalaemic – Renal tubular acidosis (I and II)
  Diabetic ketoacidosis with insulin treatment
  Posthypocapnic acidosis

Adverse Effects:

CVS - decrease contractility, arrhythmia, increase sensitivity to catecholamine
arteriolar dilation

Resp - hyperventilation

CNS – obtundation, coma

Metabolic – effects on glycolysis, O_2 dissociation etc...

Management :

- treat underlying cause
- HCO3 treatment should be considered in hyperchloraemic metabolic acidosis when pH < 7.2
  Problems with $NaHCO_3$ administration
  1. Na load
  2. fluid load
  3. osmolar load
  4. intracellular acidosis
- Dialysis

METABOLIC ALKALOSIS

Causes:

HCO_3 gain
- Exogenous HCO_3 gain
- Conversion of citrate, acetate

Loss of H^+
- Urine Cl^- < 10mmol/L (saline responsive)
  - GI loss – vomiting, villous adenoma
  - Renal Loss – diuretics, nonreabsorbable anion
  - Rapid correction of chronic hypercapnia
- Urine Cl^- >10mmol/L (saline unresponsive)
  - mineralocorticoid excess
  - glucocorticoid excess
  - hypokalaemia
  - hypomagnesmia
  - refeeding alkalosis etc

Adverse Effects:

CVS - arrhythmia, arteriolar constriction
Resp - hypoventilation
CNS - decrease cerebral blood flow, tetany, delirium
Metabolic – effects on O_2 dissociation, glycolysis etc...

Management:
- treat underlying cause
- adequate volume
- replace K^+
- consider acetazolamide (250mg oral)