Tests for the Acid-base Balance Status

Clinical assesment

- The most clinical useful information comes from the clinical description of the patient by the history and physical examination.
  - The H&P usually gives an idea of what acid base disorder might be present even before collecting the ABG sample.

Agap:

- The major causes of simple and mixed disturbances shoud be kept in mind so that such disorders may be predicted from the patient's clinical picture. For example,
  - patients with severe vomiting and those on long-term thiazide therapy can be expected to have **metabolic alkalosis**,
  - those with renal failure may have **metabolic acidosis**
  - and patients with pneumonia or congestive cardiac failure may have **respiratory alkalosis…**
- The possibility of an acid-base balance disturbance suggested by clinical presentation should then be evaluated by appropriate laboratory tests.

Blood gases

- In order to interpret acid-base disturbances, the following five factors are considered:
  - pH
  - HCO_3-
  - PCO2
  - Anion gap
  - and assessment for compensation
- The **first step** is to determine if the patient is acidemic or alkalemic, based on pH.
- **Second**, the primary disorder is determined by evaluating HCO_3– and PCO_2.
  - If HCO_3– is elevated and pH is elevated, there is metabolic alkalosis.
  - If both are decreased, there is metabolic acidosis.
- **Next**, one must look at the PCO_2 in the context of the HCO_3–.
  - If HCO_3– is within the normal reference range and PCO_2 is elevated but the patient is acidotic,
    - the condition is respiratory acidosis.
  - If bicarbonate is within the normal reference range and PCO_2 is decreased but the patient is alkalotic,
    - the condition is respiratory alkalosis.
- **Next** determine the anion gap, using standard formulas below, to determine the etiology of metabolic acidosis.
  - Anion gap = [Na] – ([Cl-] + [HCO_3-])

Elevated Anion Gap (>16 meq)	Normal Anion Gap (8-16 meq)
Increased Endogenous production: Ketoacidosis (Alcohol, Starvation, DKA) Lactic Acidosis Uremia	**Loss of Bicarbonate:** Diarrhea Carbonic anhydrase inhibitors Type 2 RTA (proximal) Pancreatic ileostomy Pancreatic, biliary, intestinal fistula **Exogenous Administration:** ammonium chloride or HCL **Decreased Renal Acid Excretion:** Type 1(distal) ,4 RTA Renal Failure
Intoxications: Methanol, Ethylene Glycol, Paraldehyde, Salicylates, INH	**Miscellaneous:** Hyperkalemia Recovery from DKA

- **Finally the pH**, HCO_3–, and PCO_2 are considered to determine if compensation is as expected based on the typical ratio of 20:1 for bicarbonate to carbonic acid.
  - For example, both decreased HCO_3– and PCO_2 should produce a slightly decreased or nearly normal pH if they are in metabolic acidosis compensation.
  - To determine the actual ratio of bicarbonate to carbonic acid, PCO_2 is converted to H_2CO_3 using the relationship
    - $PCO_2 * 0.03 = H_2CO_3$
- Compensation for metabolic acidosis or alkalosis is achieved initially by the respiratory system.
  - Respiratory compensation for **acidosis** means that the lungs increase the level of alveolar ventilation, which raises the pH toward normal.
    - The increased ventilation eliminates or blos off CO_2, which eliminates carbonic acid.
    - Also, the presence of acidosis normally increases respiratory drive.
    - The respiratory system compensates for a metabolic defect.
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  - In metabolic alkalosis, some decrease in ventilation occurs but the PCO_2 generally remains normal since respiratory compensation doesn't occur until alkalosis has been severe and prolonged.
    - Compensation for metabolic alkalosis is less complete since hypoventilation is not a naturally sustainable condition.

References

Acid-Base Disorders by Walmsley Koay and Watkinson

Clinical chemistry (A laboratory perspective) by Wendy Arneson and Jean Brickell