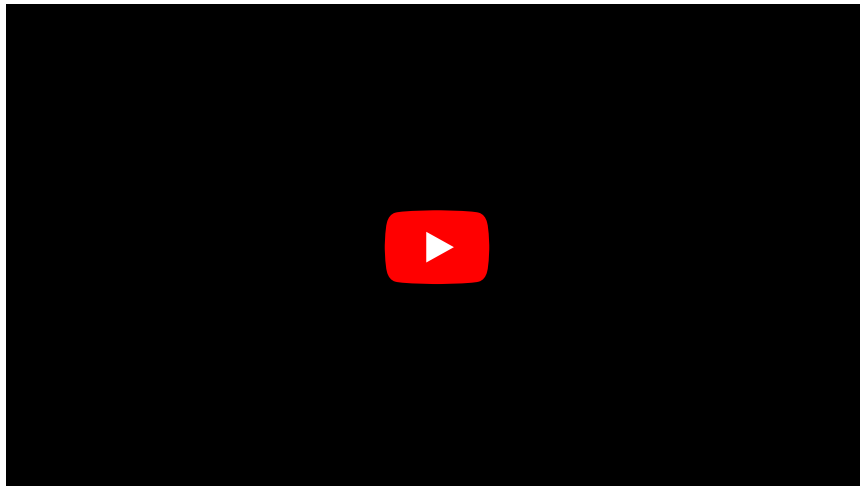


Tests for the Acid-base Balance Status

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 should 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^-
 - PCO_2
 - 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.
 - $\text{Anion gap} = [\text{Na}] - ([\text{Cl}] + [\text{HCO}_3^-])$

| 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
 - $\text{PCO}_2 * 0.03 = \text{H}_2\text{CO}_3$
- 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 blows off CO_2 , which eliminates carbonic acid.
 - Also, the presence of acidosis normally increases respiratory drive.
 - The respiratory system compensates for a metabolic defect.
- 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