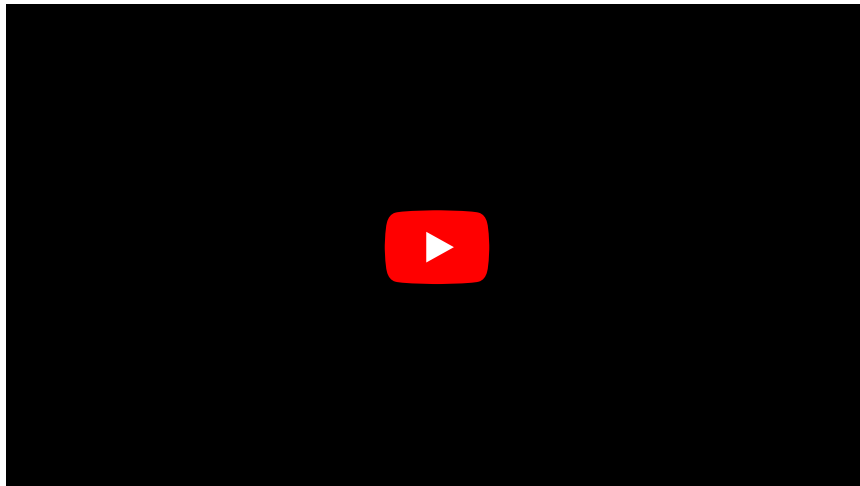


# Tests for the Acid-base Balance Status

## 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
  - $\text{HCO}_3^-$
  - $\text{PCO}_2$
  - 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  $\text{HCO}_3^-$  and  $\text{PCO}_2$ .
  - If  $\text{HCO}_3^-$  is elevated and pH is elevated, there is metabolic alkalosis.
  - If both are decreased, there is metabolic acidosis.
- **Next**, one must look at the  $\text{PCO}_2$  in the context of the  $\text{HCO}_3^-$ .
  - If  $\text{HCO}_3^-$  is within the normal reference range and  $\text{PCO}_2$  is elevated but the patient is acidotic,
    - the condition is respiratory acidosis.
  - If bicarbonate is within the normal reference range and  $\text{PCO}_2$  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)
<b>Increased Endogenous production:</b>  Ketoacidosis (Alcohol, Starvation, DKA)  Lactic Acidosis  Uremia	<b>Loss of Bicarbonate:</b> Diarrhea Carbonic anhydrase inhibitors Type 2 RTA (proximal) Pancreatic ileostomy Pancreatic, biliary, intestinal fistula  <b>Exogenous Administration:</b> ammonium chloride or HCL  <b>Decreased Renal Acid Excretion:</b> Type 1(distal) ,4 RTA Renal Failure
<b>Intoxications:</b> Methanol, Ethylene Glycol, Paraldehyde, Salicylates, INH	<b>Miscellaneous:</b> Hyperkalemia Recovery from DKA

- **Finally the pH**,  $\text{HCO}_3^-$ , and  $\text{PCO}_2$  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  $\text{HCO}_3^-$  and  $\text{PCO}_2$  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,  $\text{PCO}_2$  is converted to  $\text{H}_2\text{CO}_3$  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  $\text{CO}_2$ , 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  $\text{PCO}_2$  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