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Acidosis and Alkalosis(2)

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SKUMS

Mixed Acid-Base Disorders

- Mixed acid-base disorders—defined as
  - independently coexisting disorders,
  - not merely compensatory responses—
  - are often seen in patients in critical care units and
  - can lead to dangerous extremes of pH.

Mixed Acid-Base Disorders

- A patient with diabetic ketoacidosis (metabolic acidosis) may develop an independent respiratory problem (e.g., pneumonia) leading to respiratory acidosis or alkalosis.
- Patients with underlying pulmonary disease (e.g., COPD) may not respond to metabolic acidosis with an appropriate ventilatory response because of insufficient respiratory reserve.
- Such imposition of respiratory acidosis on metabolic acidosis can lead to severe acidemia.

Mixed Acid-Base Disorders

- When metabolic acidosis and metabolic alkalosis coexist in the same patient, the pH may be normal or near normal.
- When the pH is normal, an elevated anion gap (AG) reliably denotes the presence of an AG metabolic acidosis.
- A discrepancy in the ΔAG (prevailing minus normal AG) and theΔ $HCO3^-$ (normal minus prevailing $HCO3^-$) indicates the presence of a mixed high-gap acidosis—metabolic alkalosis.

Mixed Acid-Base Disorders

- A diabetic patient with ketoacidosis may have renal dysfunction resulting in simultaneous metabolic acidosis.
- Patients who have ingested an overdose of drug combinations such as sedatives and salicylates may have mixed disturbances as a result of the acid-base response to the individual drugs (metabolic acidosis mixed with respiratory acidosis or respiratory alkalosis, respectively).
- Triple acid-base disturbances are more complex.
- For example,
  - patients with **metabolic acidosis** due to alcoholic ketoacidosis
  - may develop **metabolic alkalosis** due to vomiting
  - and superimposed **respiratory alkalosis** due to the hyperventilation of hepatic dysfunction or alcohol withdrawal.

Approach to the Patient: Acid-Base Disorders

- A stepwise approach to the diagnosis of acid-base disorders follows (Table 47-3).
- Care should be taken when measuring blood gases to obtain the arterial blood sample without using excessive heparin.
- Blood for electrolytes and arterial blood gases should be drawn simultaneously prior to therapy, because an increase in [HCO_3^-] occurs with metabolic alkalosis and respiratory acidosis.
- Conversely, a decrease in [HCO_3^-] occurs in metabolic acidosis and respiratory alkalosis

Calculated Vs Measured Value

- In the determination of arterial blood gases by the clinical laboratory, both pH and Paco2 are measured, and the [HCO_3^-] is calculated from the Henderson-Hasselbalch equation.
- This calculated value should be compared with the measured [HCO_3^-] (total CO_2) on the electrolyte panel.
- These two values should agree within 2 mmol/L.
- If they do not, the values may not have been drawn simultaneously, a laboratory error may be present, or an error could have been made in calculating the [HCO_3^-].
- After verifying the blood acid-base values, the precise acid-base disorder can then be identified.

Steps in Acid-Base Diagnosis

- 1. Obtain arterial blood gas (ABG) and electrolytes simultaneously.
- 2. Compare [HCO3–] on ABG and electrolytes to verify accuracy.
- 3. Calculate anion gap (AG).
- 4. Know four causes of high-AG acidosis (ketoacidosis, lactic acid acidosis, renal failure, and toxins).

Steps in Acid-Base Diagnosis

- 5. Know two causes of hyperchloremic or nongap acidosis (bicarbonate loss from GI tract, renal tubular acidosis).
- 6. Estimate compensatory response.
- 7.Compare ΔAG and ΔHCO3–.
- 8. Compare change in [Cl– with change in [Na+].

Anion Gap (1)

- Unmeasured Anions - Unmeasured Cations

- measured Cations - measured Anions

- $[Na+] - ([Cl^-] + [HCO3^-])$

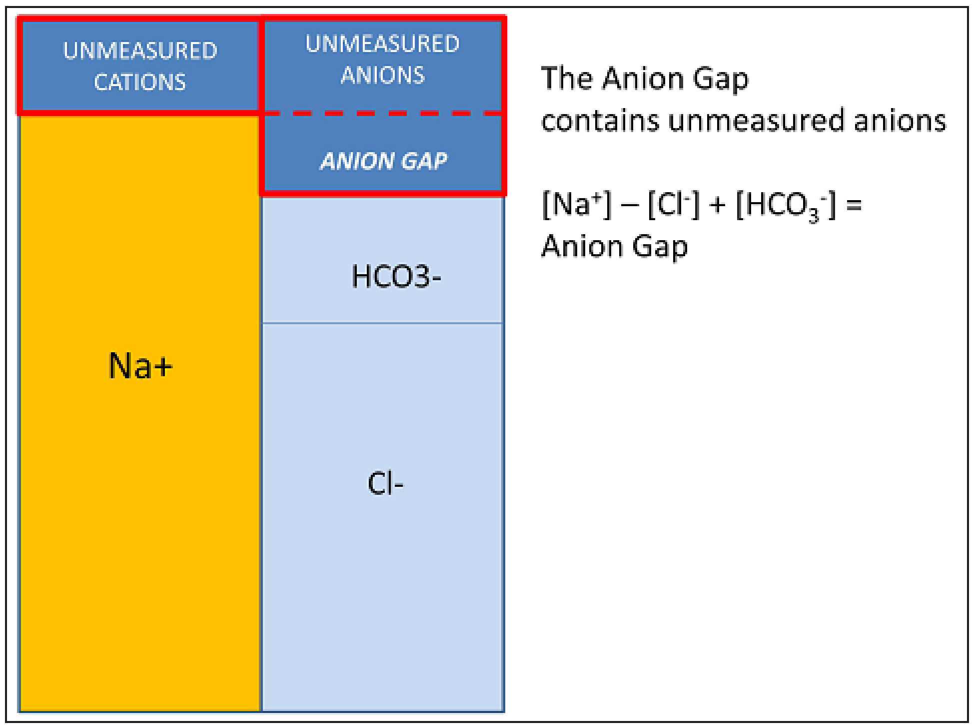


Fig 3
(a) Illustration of the "normal" anion gap

Calculate the Anion Gap

- All evaluations of acid-base disorders should include a simple calculation of the AG;
- it represents those unmeasured anions in plasma (normally 10 to 12 mmol/L) and is calculated as follows: $AG = Na^+ – (Cl^– + HCO_3^–)$.
- The unmeasured anions include anionic proteins, (e.g., albumin), phosphate, sulfate, and organic anions.
- When acid anions, such as acetoacetate and lactate, accumulate in extracellular fluid, the AG increases, causing a high-AG acidosis.

Calculate the Anion Gap

- An increase in the AG is most often due to an increase in unmeasured anions
- and, less commonly, is due to a decrease in unmeasured cations (calcium, magnesium, potassium).

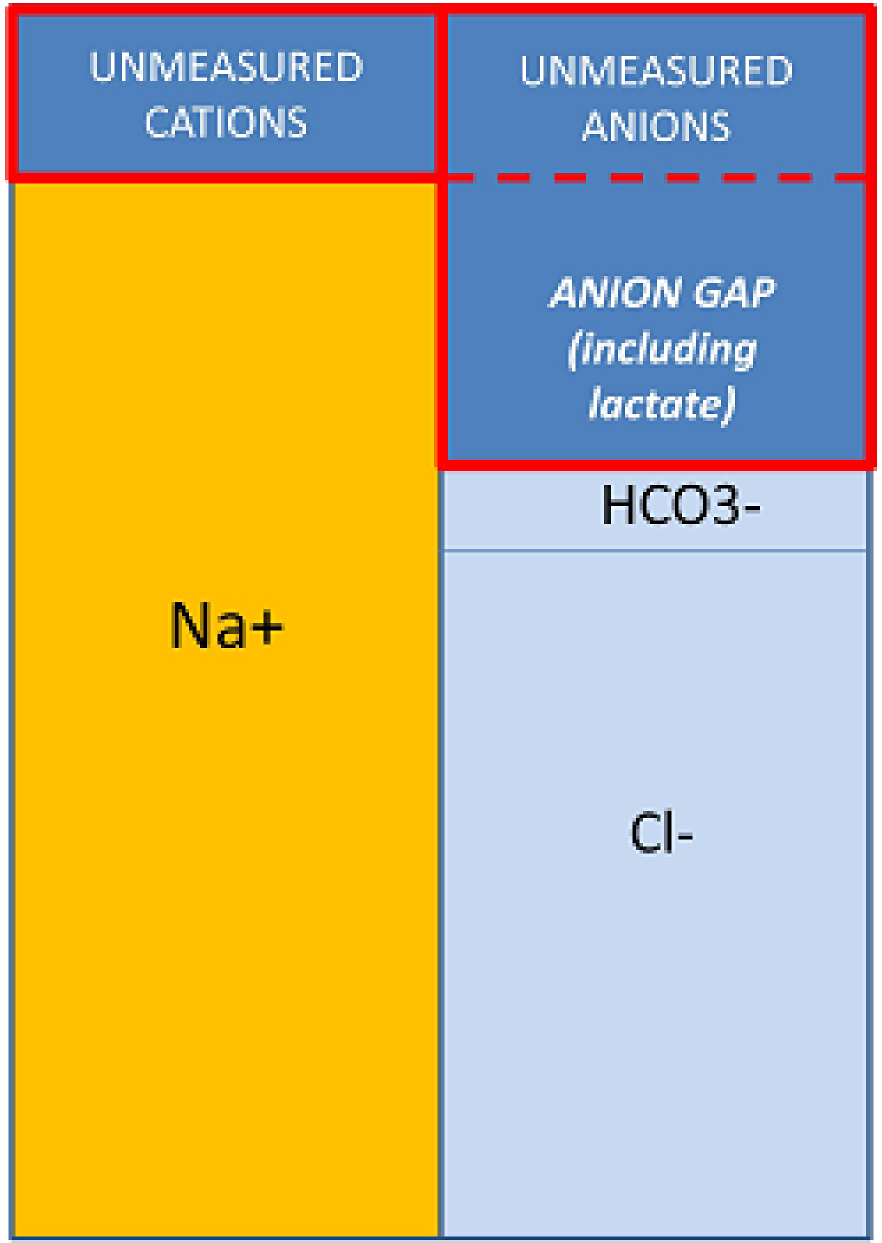
Calculate the Anion Gap

- A decrease in the AG can be due to
  - (1) an increase in unmeasured cations;
  - (2) the addition to the blood of abnormal cations, such as lithium (lithium intoxication) or cationic immunoglobulins (plasma cell dyscrasias);
  - (3) a reduction in the major plasma anion albumin concentration (nephrotic yndrome);

- A fall in serum albumin by 1 g/dL from the normal value (4.5 g/dL) decreases the AG by 2.5 meq/L.

Compare the change in [HCO3–] (ΔHCO3–) and the change in the AG (ΔAG).

- Similarly, normal values for [HCO3–], Paco2, and pH do not ensure the absence of an acid-base disturbance.
- For instance, an alcoholic who has been vomiting may develop a metabolic alkalosis with a pH of 7.55, Paco2 of 47 mmHg, [HCO3–] of 40 mmol/L, [Na+] of 135, [Cl–] of 80, and [K+] of 2.8.
- If such a patient were then to develop a superimposed alcoholic ketoacidosis with a beta –hydroxybutyrate concentration of 15 mM, arterial pH would fall to 7.40, [HCO3–] to 25 mmol/L, and the Paco2 to 40 mmHg.
- Although these blood gases are normal, the AG is elevated at 30 mmol/L, indicating a mixed metabolic alkalosis and metabolic acidosis.
- A mixture of high-gap acidosis and metabolic alkalosis is recognized easily by comparing the differences (delta values) in the normal to prevailing patient values.
- In this example, the ΔHCO3– is 0 (25 – 25 mmol/L) but the ΔAG is 20 (30 – 10 mmol/L).
- Therefore, 20 mmol/L is unaccounted for in the Δ / Δ value (ΔAG to ΔHCO3–).



In a High Anion Gap Metabolic Acidosis (HAGMA), e.g. lactic acidosis, the anion gap will increase following addition of "new" anions (lactate) with a corresponding fall in bicarbonate as it is used to buffer the additional acid (H^+)

(b) High anion gap present in a metabolic acidosis