

Acid-base chemistry

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Key Concepts

Arrhenius Acids and Bases

$\text{HCl} \rightarrow \text{H}^+ + \text{Cl}^-$
Acid - forms H^+ in water

$\text{NaOH} \rightarrow \text{Na}^+ + \text{OH}^-$
Base - forms OH^- in water

Acid and Base Definitions:

- Acid:** Donates a proton (H^+)
 - Example: $\text{HCl} \rightarrow \text{H}^+ + \text{Cl}^-$
- Base:** Accepts a proton
 - Example: $\text{H}^+ + \text{NH}_3 \rightarrow \text{NH}_4^+$

Acid-Base Pairs:

- Carbonic Acid/Bicarbonate ($\text{H}_2\text{CO}_3/\text{HCO}_3^-$)
- Ammonium/Ammonia ($\text{NH}_4^+/\text{NH}_3$)
- Monobasic/Dibasic Phosphate ($\text{H}_2\text{PO}_4^-/\text{HPO}_4^{2-}$)
- Lactic Acid/Lactate ($\text{C}_3\text{H}_6\text{O}_3/\text{C}_3\text{H}_5\text{O}_3^-$)

2. Acidemia vs. Alkalemia:

- Acidemia:** Low pH (<7.35)
- Alkalemia:** High pH (>7.45)

Acidosis and Alkalosis: Processes of acid or alkali accumulation

- Note: Acidosis can occur without acidemia if balanced by a corresponding alkalosis.

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pH: Key Points

Søren Sørensen first introduced the concept in 1909

Normal Range: 7.35 – 7.45

Consequences of Abnormal pH:

- Acidemia (pH < 7.35):**
 - Arterial vasodilation & venous vasoconstriction
 - Decreased myocardial contractility
 - Reduced hepatic & renal perfusion
 - Decreased oxygen-hemoglobin binding
 - Potential coma risk
- Alkalemia (pH > 7.45):**
 - Cerebral vasoconstriction
 - Reduced myocardial contractility
 - Increased oxygen-hemoglobin binding (impaired O_2 delivery)
 - Potential coma risk

$\text{pH} = -\log_{10}[\text{H}^+]$

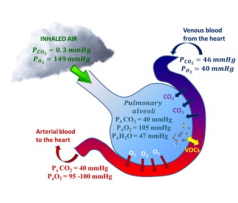
pH is a logarithmic measure of hydrogen ion concentration $[\text{H}^+]$ in a solution

each unit change in pH corresponds to a tenfold change in hydrogen ion concentration

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Arterial Partial Pressure of Carbon Dioxide (PaCO_2)

- Normal PaCO_2 Range:** 35–45 mm Hg (4.655–5.985 kPa)
- Role of PaCO_2 in Assessing Lung Function:**
 - Indicator of Ventilatory Adequacy:**
 - Assesses the lungs' effectiveness in excreting CO_2 .
- PaCO_2 and Carbonic Acid (H_2CO_3) Relationship:**
 - Directly Proportional:** $\text{PaCO}_2 \times 0.03 = \text{H}_2\text{CO}_3$ concentration
 - Impact on Acid-Base Balance:**
 - Elevated PaCO_2 contributes to acidosis due to increased carbonic acid.



The diagram illustrates the partial pressures of gases in different compartments of the lung and blood. Inhaled air has $\text{P}_{\text{CO}_2} = 0.3 \text{ mmHg}$ and $\text{P}_{\text{O}_2} = 149 \text{ mmHg}$. Pulmonary air has $\text{P}_{\text{CO}_2} = 40 \text{ mmHg}$, $\text{P}_{\text{O}_2} = 100 \text{ mmHg}$, and $\text{P}_{\text{H}_2\text{O}} = 47 \text{ mmHg}$. Arterial blood to the lung has $\text{P}_{\text{CO}_2} = 40 \text{ mmHg}$ and $\text{P}_{\text{O}_2} = 95-100 \text{ mmHg}$. Venous blood from the heart has $\text{P}_{\text{CO}_2} = 46 \text{ mmHg}$ and $\text{P}_{\text{O}_2} = 40 \text{ mmHg}$.

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Arterial Partial Pressure of Carbon Dioxide (PaCO₂)

4. Effects of Ventilation on PaCO₂ Levels:

- Hypoventilation:**
 - Decreases CO₂ excretion → Increased PaCO₂ → Acidosis
- Hyperventilation:**
 - Increases CO₂ excretion → Decreased PaCO₂ → Alkalosis

5. Respiratory Compensation Mechanism:

- Primary Regulation:** Ventilatory adjustments help compensate for metabolic acid–base disturbances.
- Significance:** Key mechanism for maintaining acid–base homeostasis.

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Arterial Partial Pressure of Oxygen (PaO₂)

1. Normal PaO₂ Range: 80–100 mm Hg (10.64–13.3 kPa)

2. Importance of PaO₂:

- Indicator of Arterial Oxygenation:** Reflects lung function and efficiency of oxygen delivery to tissues.

3. Hypoxemia Threshold:

- Clinically Significant Hypoxemia:** PaO₂ < 80 mm Hg
- No absolute cutoff; typically assessed in relation to metabolic needs.

PaO₂:

- PaO₂ is the partial pressure of oxygen in the alveoli
- The pressure of oxygen dissolved in the arterial blood
- Measurement of how well oxygen is moving from the lungs to the blood
- Normal range is 80–100 mm Hg

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Arterial Partial Pressure of Oxygen (PaO₂)

4. Factors Influencing PaO₂:

- Ventilation Levels
- Fraction of Inspired Oxygen (FIO₂)
- Lung Functional Capacity
- Oxyhemoglobin Dissociation Curve

5. Oxyhemoglobin Dissociation Curve:

- Relationship:** PaO₂ vs. Oxygen Saturation
- Acidemia Shift:** Curve shifts right → Easier O₂ release from hemoglobin
- Alkalemia Shift:** Curve shifts left → More difficult O₂ release (impaired tissue delivery)
- Other Influencing Factors:** Temperature, 2,3-diphosphoglycerate levels

ALVEOLAR GAS EQUATION

$$P_{A,O_2} = F_{I,O_2} (P_{ATM} - P_{H_2O}) - \frac{P_{A,CO_2}}{RQ}$$

OXYGENATION

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Oxygen–hemoglobin dissociation curve

Shape and Meaning: The curve is sigmoid-shaped (S-shaped) and shows the relationship between the partial pressure of oxygen (PO₂) and hemoglobin's saturation with oxygen. As PO₂ increases, hemoglobin binds more oxygen, up to a point where it becomes saturated.

Affinity Changes: The curve's shape reflects hemoglobin's changing affinity for oxygen. At high PO₂ (like in the lungs), hemoglobin has a high affinity for oxygen, binding it easily. At lower PO₂ (like in tissues), it has a lower affinity, releasing oxygen more readily.

Shifts of the Curve: The curve can shift left or right, depending on factors like pH, temperature, CO₂, and 2,3-DPG levels. A right shift means oxygen is released more easily (helpful in active tissues), while a left shift means oxygen is held more tightly by hemoglobin.

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Arterial Partial Pressure of Oxygen (PaO₂)

6. PaO₂ vs. Oxygen Saturation:

- **PaO₂**: Measured in mm Hg; can exceed 100 with supplemental O₂
- **Oxygen Saturation (SaO₂)**: Percentage of hemoglobin-bound O₂; cannot exceed 100%
- **Example**: PaO₂ of 80 mm Hg typically corresponds to SaO₂ > 93%, while SaO₂ of 80% is critically low.

7. Clinical Relevance:

- **PaO₂**: Crucial for assessing pulmonary status; does not directly affect acid-base balance.
- **SaO₂**: Directly relates to O₂ delivery adequacy, critical in hypoxic states.

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Arterial Bicarbonate (HCO₃⁻)

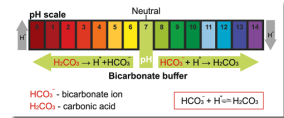
- Normal HCO₃⁻ Range:**
 - 22-26 mEq/L (22-26 mmol/L)

2. Measurement of Arterial Bicarbonate:

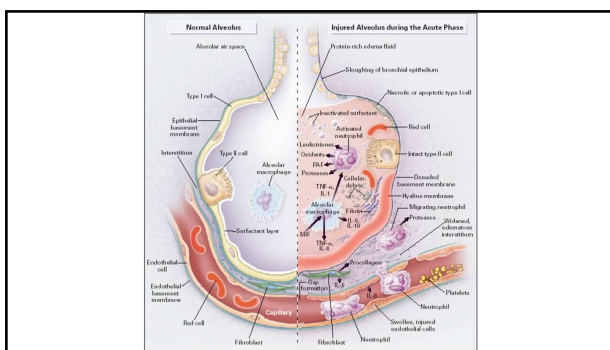
- **Indirect Calculation:**
 - Derived from pH and PaCO₂ via the Henderson-Hasselbalch equation in ABG (Arterial Blood Gas) analysis.

3. Comparison with Serum Bicarbonate:

- **Total CO₂ Content:** Measured on an electrolyte panel, often referred to as **serum bicarbonate**.
- **Expected Difference:**
 - HCO₃⁻ from ABG typically 1.5-3 mEq/L lower than total CO₂ in a plasma panel (higher difference in venous samples).



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Anion Gap (AG)

- Normal Range:** 3-16 mEq/L (3-16 mmol/L)

2. Purpose of Anion Gap:

- **Estimate of Unmeasured Anions:** Useful for evaluating potential causes of **metabolic acidosis**.
- **Electrochemical Balance:** Total anions and cations should balance; the AG highlights excess unmeasured anions.

3. Anion Gap Calculation:

- **Formula:** $AG = Na^+ - (Cl^- + HCO_3^-)$ or $AG = Na^+ - (Cl^- + HCO_3^-)$
- **High AG (>16 mEq/L):** Indicates anion accumulation, often due to conditions like lactic acidosis, ketoacidosis, toxic ingestions, or renal failure.

ANION GAP ACIDOSIS

<p>Glycols • Ethylene • Propylene</p> <p>Oxoprolone • Fentanyl</p> <p>L-lactate • Type A: Ischemic • Type B: Non-Ischemic • Metformin • Renal • Starvation • Alcohol • Diabetes</p> <p>D-lactate • Short gut</p>	<p>Methanol • Ethylene • Propylene</p> <p>Aspirin • Other salicylates (e.g. bismuth)</p> <p>Renal • Uremia • Organic acids</p> <p>Ketones • Starvation • Alcohol • Diabetes</p>
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Anion Gap (AG)

4. Influence of Albumin on AG:

- Albumin Adjustment:** For hypoalbuminemia, adjust AG by adding 2.5 for each 1 g/dL decrement in albumin: $\text{Corrected AG} = \text{AG} + 2.5 \times (4.4 - \text{measured albumin})$
- Corrected AG:** $\text{Corrected AG} = \text{AG} + 2.5 \times (4.4 - \text{measured albumin})$

5. Additional Factors Affecting AG:

- Confounding Variables:** Serum phosphate, magnesium, calcium, and certain medications (e.g., β -lactam antibiotics) can impact AG interpretation.

Graph Credit: <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3388133/figure/>

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Serum Lactate

1. Normal Range: 0.6-2 mmol/L

2. Importance of Serum Lactate:

- Byproduct of Anaerobic Metabolism:**
 - Elevations indicate **inadequate tissue perfusion** or oxygenation.

3. Clinical Significance in Sepsis:

- Sepsis Diagnostic Criteria:**
 - Lactate >1 mmol/L as per the **Surviving Sepsis Campaign**.
- High-Risk Threshold:**
 - Lactate >4 mmol/L is associated with increased mortality risk.

BACKGROUND

- PRODUCT OF PYRUVATE METABOLISM
- PRODUCED BY MUSCLE & BRED
- IF LACTATE LEVELS >4 IT IS CAUSE LACTIC ACIDOSIS
- THIS IS ALSO INTERFERED WITH "TYPICAL" TISSUE PERFUSION
- THIS IS CAUSED BY INADEQUATE TISSUE PERFUSION (IMPERFECT TISSUE PERFUSION) (IMPERFECT TISSUE PERFUSION)

CAUSES

- IF LACTATE:
 - IF LACTIC ACID PRODUCTION
 - IF LACTIC ACID CLEARANCE
 - CONCENTRATION IN BLOOD

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Serum Lactate

4. Use of Serial Lactate Measurements:

- Monitoring Resuscitation:**
 - Serial lactate levels provide insight into the **effectiveness of resuscitation efforts** and tissue perfusion recovery.
- Indicator of Prognosis:**
 - Persistent elevation may indicate ongoing hypoperfusion and a need for further intervention.

5. Interpretation and Clinical Decisions:

- Rising Lactate Levels:** Suggest inadequate perfusion and potential for deteriorating patient status.
- Decreasing Lactate Levels:** Suggest improving perfusion, a positive sign in response to therapy.

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