Arterial Blood Gas
(ABG)

Dr. Anan Esmail
obtaining the arterial blood

- percutaneous needle puncture
- indwelling arterial catheter
Common sites include the radial, femoral, brachial Artery.

radial artery is used most often.
the amount of **heparin** solution should be **minimized**

at least **2 to 3 ml** of blood should be obtained
air bubbles should be removed

the specimen immediately placed on ice and analyzed as quickly as possible (15 min)
pressure should be applied to the puncture site for **five to ten minutes**
<table>
<thead>
<tr>
<th>Parameter</th>
<th>Arterial sample</th>
<th>peripheral venous sample</th>
<th>central venous sample</th>
</tr>
</thead>
<tbody>
<tr>
<td>PH</td>
<td>7.36 to 7.44</td>
<td>0.02 to 0.04 lower</td>
<td>0.03 to 0.05 lower</td>
</tr>
<tr>
<td>PCO₂</td>
<td>36 to 44 mmHg</td>
<td>3 to 8 mmHg higher</td>
<td>4 to 5 mmHg higher</td>
</tr>
<tr>
<td>HCO₃</td>
<td>21 to 27 mEq/L</td>
<td>1 to 2 meq/L higher</td>
<td>little or no increase</td>
</tr>
<tr>
<td>PaO₂</td>
<td>&gt;80 mmHg</td>
<td></td>
<td></td>
</tr>
<tr>
<td>SaO₂</td>
<td>&gt;95 %</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
HENDERSON EQUATION

\[
[H^+] = K \times \frac{P_a \text{CO}_2}{[\text{HCO}_3^-]}, \text{ where } K = 24
\]

<table>
<thead>
<tr>
<th>pH</th>
<th>[H⁺]</th>
<th>pH</th>
<th>[H⁺]</th>
</tr>
</thead>
<tbody>
<tr>
<td>7.00</td>
<td>100</td>
<td>7.35</td>
<td>45</td>
</tr>
<tr>
<td>7.05</td>
<td>89</td>
<td>7.40</td>
<td>40</td>
</tr>
<tr>
<td>7.10</td>
<td>79</td>
<td>7.45</td>
<td>35</td>
</tr>
<tr>
<td>7.15</td>
<td>71</td>
<td>7.50</td>
<td>32</td>
</tr>
<tr>
<td>7.20</td>
<td>63</td>
<td>7.55</td>
<td>28</td>
</tr>
<tr>
<td>7.25</td>
<td>56</td>
<td>7.60</td>
<td>25</td>
</tr>
<tr>
<td>7.30</td>
<td>50</td>
<td>7.65</td>
<td>22</td>
</tr>
</tbody>
</table>
Simple and mixed acid-base disorders
Establish the primary diagnosis

<table>
<thead>
<tr>
<th></th>
<th>PH (7.36-7.44)</th>
<th>PaCO2 (36-44)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Respiratory <strong>acidosis</strong></td>
<td>↓</td>
<td>↑</td>
</tr>
<tr>
<td>Metabolic <strong>acidosis</strong></td>
<td>↓</td>
<td>↓</td>
</tr>
<tr>
<td>Respiratory <strong>alkalosis</strong></td>
<td>↑</td>
<td>↓</td>
</tr>
<tr>
<td>Metabolic <strong>alkalosis</strong></td>
<td>↑</td>
<td>↑</td>
</tr>
</tbody>
</table>
Assess the degree of compensation

<table>
<thead>
<tr>
<th></th>
<th>PH</th>
<th>PaCO2</th>
<th>HCO3 (21-27)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Respiratory acidosis</strong></td>
<td>↓</td>
<td>↑</td>
<td>↑</td>
</tr>
<tr>
<td><strong>Metabolic acidosis</strong></td>
<td>↓</td>
<td>↓</td>
<td>↓</td>
</tr>
<tr>
<td><strong>Respiratory alkalosis</strong></td>
<td>↑</td>
<td>↓</td>
<td>↓</td>
</tr>
<tr>
<td><strong>Metabolic alkalosis</strong></td>
<td>↑</td>
<td>↑</td>
<td>↑</td>
</tr>
</tbody>
</table>
Metabolic acidosis
1.2 mmHg fall in arterial PCO2 for every 1 meq/L reduction in the serum HCO3 concentration

- PCO2 = 1.5 x serum HCO3 + 8 ± 2
- PCO2 = Serum HCO3 + 15
- PCO2 should be similar to the decimal digits of the arterial pH
Metabolic acidosis

<table>
<thead>
<tr>
<th>PH</th>
<th>PaCO2</th>
<th>HCO3</th>
</tr>
</thead>
<tbody>
<tr>
<td>↓</td>
<td>↓ red</td>
<td>↓</td>
</tr>
</tbody>
</table>

- Calculate $PCO2 = PaCO2$

- Calculate $PCO2 > PaCO2$
- حماض تنفسي مرافق

- Calculate $PCO2 < PaCO2$
- قلء تنفسي مرافق
Metabolic alkalosis
### Metabolic Alkalosis

<table>
<thead>
<tr>
<th>Metabolic alkalosis</th>
<th>PH</th>
<th>PaCO2</th>
<th>HCO3</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>↑</td>
<td>↑</td>
<td>↑</td>
</tr>
</tbody>
</table>

- Raise the PaCO2 by **0.7 mmHg** for every **1 meq/L** elevation in the serum HCO3 concentration.

- PCO2 = 0.9 x HCO3 + (9 to 16)
- PCO2 should be similar to the decimal digits of the arterial pH.
<table>
<thead>
<tr>
<th>Metabolic alkalosis</th>
<th>PH</th>
<th>PaCO2</th>
<th>HCO3</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>↑</td>
<td>↑</td>
<td>↑</td>
</tr>
</tbody>
</table>

calculat PCO2 = PaCO2

- calculat PCO2 > PaCO2
- calculat PCO2 < PaCO2
Respiratory disorder
# Respiratory disorder (acute or chronic)

<table>
<thead>
<tr>
<th>Chronic (3-5DAY)</th>
<th>Acute</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>PH</td>
<td>Pa CO2</td>
<td>PH</td>
</tr>
<tr>
<td>↓ 0.03</td>
<td>↑ 10 mmHg</td>
<td>↓ 0.07</td>
</tr>
<tr>
<td>↑ 0.03</td>
<td>↓ 10 mmHg</td>
<td>↑ 0.08</td>
</tr>
</tbody>
</table>
Respiratory acid-base disorders

<table>
<thead>
<tr>
<th></th>
<th>chronic</th>
<th>acute</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>3 to 5 days</td>
<td></td>
</tr>
<tr>
<td>HCO₃</td>
<td>↑3.5 to 5 meq/L</td>
<td>↑1 meq/L</td>
</tr>
<tr>
<td>PaCO₂</td>
<td>↑10 mmHg</td>
<td>↑10 mmHg</td>
</tr>
<tr>
<td></td>
<td>↓4 to 5 meq/L</td>
<td>↓2 meq/L</td>
</tr>
<tr>
<td></td>
<td>↓10 mmHg</td>
<td>↓10 mmHg</td>
</tr>
</tbody>
</table>

- **Respiratory acidosis**
- **Respiratory alkalosis**
Assess the degree of compensation

- $\text{HCO}_3^-$ predicted in arterial gases < $\text{HCO}_3^-$ predicted in venous gases
  
  Decreased compensatory reserve

- $\text{HCO}_3^-$ predicted in arterial gases > $\text{HCO}_3^-$ predicted in venous gases
  
  Increased compensatory reserve
Calculat anion gap

Calculat $\text{AG} = \text{Na} - (\text{CL} + \text{HCO}_3)$

Normal AG ($=12$)

Adjusted AG = Calculated AG + [2.5 × (4.5 – alb in g/dl)]
AG = 

( total cations – total anions ) = 0

(measured cations + unmeasured cations) – (measured anions + unmeasured anion) = 0

measured cations – measured anions = unmeasured anions - unmeasured cations

measured cations – measured anions = 23 - 11 = 12
AG = 12

unmeasured anions - unmeasured cations
measured cations – measured anions

• unmeasured anions (albumin, phosphate, sulfate, and organic anions such as lactate)

• remaining cations (potassium, calcium, and magnesium)
  – normal concentrations of these cations are so low that a reduction in concentration has only a small effect on the AG
Anion gap

\[ AG = ([Na^+] + [K^+]) - ([Cl^-] + [HCO_3^-]) \]

Or as just discussed,

\[ AG = \text{[unmeasured anions]} - \text{[unmeasured cations]} \]

Increase in anion gap (>20 mEq/L)
Can be due to:
- Fall in unmeasured cations
  - Hypocalcemia
  - Hypomagnesemia
- Rise in unmeasured anions
  - Hyperalbuninemia (e.g., due to volume contraction)
  - Increase in organic anions

Decrease in anion gap (<7 mEq/L)
Can be due to:
- Rise in unmeasured cations
  - Hyperkalemia*
  - Hypermagnesemia
  - Lithium intoxication
  - Paraproteinemias
- Fall in unmeasured anions
  - Hypoalbuminemia (see next section)
Check the $\Delta / \Delta$

there is a superimposed process which is causing a further fall in the HCO₃⁻

$\Delta$HCO₃ / $\Delta$ AG

Normal HCO₃ – measured HCO₃ / measured AG - normal AG
\[ \Delta \text{AG}/\Delta \text{HCO}_3 = 1-2 \]

pure uncomplicated high AG metabolic acidosis

\[ \Delta \text{AG}/\Delta \text{HCO}_3 < 1 \]

AG metabolic acidosis

NAG metabolic acidosis

\[ \Delta \text{AG}/\Delta \text{HCO}_3 > 2 \]

AG metabolic acidosis

metabolic alkalosis
causes of Anion gap metabolic acidosis

- Ketoacidosis
- Lactic acidosis
- Alcohol-induced
- Methanol
- Uremia
- Salicylates
- Starvation
- Ethylene glycol
- Paraldehyde
causes of Nonanion gap metabolic acidosis

GI loss of HCO3
- Diarrhea
- Ileostomy - colostomy
- Pancreatic fistula

Renal loss of HCO3
- Renal tubular acidosis
- Hypoaldosteronism
- Hyperkalemia
Urine gap = (UNa + Uk) – UCl

- If positive → a **renal** cause of the metabolic acidosis (RTA).
- If negative → **extrarenal** cause of metabolic acidosis.
causes of metabolic alkalosis

- Urinary Cl $< 20$ mmol/l → Cl – responsive metabolic alkalosis
- Urinary Cl $> 20$ mmol/l → Cl – resistant metabolic alkalosis
causes of metabolic alkalosis

Cl responsive

GI loss of H +
- Vomiting
- nasogastric suction
- Cl – -rich diarrhea

Renal loss of H +
- Diuretics
- Hypovolemia
- Posthypercapnia
causes of metabolic alkalosis

Renal loss of H+

Primary hyperaldosteronism

Increased corticosteroid activity

Hypokalemia

Increased renin activity
causes of Respiratory acidosis

- Hypoventilation (CNS disorder, drug)
- Chest wall restriction - Neuromuscular disease
- Lung disease (ILD – COPD – ASTHMA – OSA - OHS)
causes of **Respiratory alkalosis**

- Anxiety, pain, psychogenic
- Pulmonary embolism, Pneumothorax, Asthma exacerbation, Pulmonary congestion
- Fever, Sepsis, infection, Pregnancy
- Liver failure with encephalopathy
MECHANISMS OF HYPOXEMIA
Arterial oxygen saturation (SaO2)

- Most of the oxygen that diffuses from the alveolus to the pulmonary capillary binds to hemoglobin in red blood cells.

the proportion of red blood cells whose hemoglobin is bound to oxygen
Arterial oxygen saturation (SaO2)

- Resting SaO2 ≤95 percent or exercise desaturation ≥5 percent abnormal
Arterial oxygen tension (PaO2)

- small amount of the oxygen that diffuses from the alveolus to the pulmonary capillary dissolves into the plasma

the amount of oxygen dissolved in the plasma
Arterial oxygen tension (PaO2)

- it seems reasonable to consider a PaO2 <80 mmHg abnormal
MECHANISMS OF HYPOXEMIA

Hypoxemia defined as a decrease in the partial pressure of oxygen in the blood.
Gradient $P(A - a)O_2$

$FiO_2$ is the fraction of inspired oxygen (0.21 at room air)
Gradient $P(A - a)O_2$

$Patm$ is the atmospheric pressure (760 mmHg at sea level)

$Patm = 760 - \{760 \times (\text{elevation} / -7000)\}$
Gradient $P(A-a)O_2$

$PH_2O$ is the partial pressure of water (47 mmHg at 37ºC)

$PH_2O = 47 + \{47 \times (\text{patient temperature} - 37)/18.4\}$
Gradient $P(A - a)O2$

PaCO2 is the arterial carbon dioxide tension

R is the respiratory quotient is approximately 0.8 at steady state
Gradient

\[ P(A - a)O_2 \]

\[ = PAO_2 - PaO_2 \]

\[ PAO_2 = (FiO_2 \times [Patm - PH_2O]) - (PaCO_2 \div R) \]

- at room air
- at sea level
- at 37ºC

\[(FiO_2 \times [Patm - PH_2O]) = 150\]
Gradient

\[ P(A - a)O_2 \]

\[ = \text{PAO}_2 - \text{PaO}_2 \]

\[ \text{PAO}_2 = (\text{FiO}_2 \times [\text{Patm} - \text{PH}_2\text{O}]) - (\text{PaCO}_2 \div R) \]

- at room air
- damascus
- at 37\(^\circ\)C

\[(\text{FiO}_2 \times [\text{Patm} - \text{PH}_2\text{O}]) = 138\]
Gradient

$P(A - a)O_2$

Normal $= 5-15$

$= 2.5 + 0.21 \times \text{(age)}$
# MECHANISMS OF HYPOXEMIA

<table>
<thead>
<tr>
<th>Increase FIO2</th>
<th>P(A – a)O2</th>
<th>PaCO2</th>
<th>PaO2</th>
<th>CAUSE</th>
</tr>
</thead>
<tbody>
<tr>
<td>correct</td>
<td>⇐ createContext</td>
<td>↑</td>
<td>↓</td>
<td>Hypoventilation</td>
</tr>
<tr>
<td>correct</td>
<td>↑</td>
<td>⇐ createContext</td>
<td>⇐ createContext</td>
<td>V/Q mismatch</td>
</tr>
<tr>
<td>correct</td>
<td>⇐ createContext</td>
<td>⇐ createContext</td>
<td>⇐ createContext</td>
<td>decrease FIO2</td>
</tr>
<tr>
<td>correct</td>
<td>↑</td>
<td>⇐ createContext</td>
<td>⇐ createContext</td>
<td>Diffusion limitation</td>
</tr>
<tr>
<td>difficult to</td>
<td>↑↑</td>
<td>⇐ createContext</td>
<td>⇐ createContext</td>
<td>Right-to-left shunts</td>
</tr>
<tr>
<td>correct</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
HYPOXEMIA

A-a gradient

Normal

Increased

Response to 100% FiO₂

Hypoxemia corrects

Hypoxemia does not correct*

5 Causes of Hypoxemia

↓ FiO₂

Hypoventilation

Associated with ↑ PCO₂

CNS depression (e.g., brainstem stroke, narcotic overdose)
Obesity hypoventilation
Muscular weakness

High altitude

V/Q mismatch

Most common cause of hypoxemia

Interstitial lung disease
Emphysema
Pulmonary vascular disease
↑ cardiac output states (↑ transit time through alveolar-capillary membrane)

Obstructive lung disease
PE
Mild alveolar filling diseases ("blood, water, pus, protein")

Diffusion impairment

Shunt

*Only large shunts do not correct completely

"Blood, water, pus, protein"
Alveolar collapse (atelectasis, pleural processes)
Pulmonary AVMs, intracardiac shunts
Thank You