Acid – Base Balance ( Respiratory Component )

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Objectives:
● Define Respiratory Acidosis & Alkalosis
● Describe the control of CO₂ by the lungs & the bicarbonate /carbonic acid buffer system
● State the blood biochemical findings in both conditions
● List some of the causes of such acid-base disorders
Gas Exchanges

Lungs

Veins

Arteries

Capillaries

(TCH2O) + O2 → CO2 + HOH

Tissue Cells
- In the lungs, the deoxygenated hemoglobin (HHb) of the venous blood coming into it releases its H+ (acting as an acid) and O2 enters to form HbO2 during respiration. This inhaled (inspired) O2 is carried to tissues as HbO2 by arterial blood.

- The released H+ combining with the bicarbonate HCO3 coming from tissues to form the carbonic acid (H2CO3) by the enzyme carbonic anhydrase or dehydratase (CA or CD).
H$_2$CO$_3$ will dissociate & CO$_2$ is released because of its low tension in the lung. Then, CO$_2$ is released in expired air.

- In tissues, HbO$_2$ carried by arterial blood (acting as a base) accept H$^+$ & release its O$_2$ into the tissues to become deoxygenated or protonated Hb i.e. HHb to be carried by venous blood to reenter the lung & release its H$^+$ & the CO$_2$ formed (recycle).

Where does CO$_2$ come from?
CO₂ is resulted from the aerobic metabolism that utilize O₂ & some carbon in organic compounds is oxidized to CO₂. 

The CO₂ of high tension in tissues is eliminated by diffusion into the blood & into the lung or it combines with H₂O forming H₂CO₃ (a weak acid that dissociates into H⁺ & HCO₃⁻) by CA enzyme.

- The rate of respiration (rate of CO₂ elimination) is controlled by Chemoreceptors in the Respiratory Center in the Medulla of the brain (also in the Carotid & aortic bodies). The Receptors respond to changes in the [CO₂] or
[H+] of blood plasma or Cerebrospinal fluid. If pCO₂ is ↑ due to retention of CO₂ (>5.3 kPa or 40 mmHg) then [H+] is ↑ & pH is ↓ (i.e. pH < 7.35) a case known as Acidosis.

If pCO₂ is ↓, then [H+] is ↓ & pH is ↑ (i.e. pH < 7.45) a case known as Alkalosis. Because this acid-base imbalance is a result of Respiration disorders, then the terms Respiratory Acidosis & Respiratory Alkalosis come true, usually due to impaired alveolar ventilation (Hypoventilation) or abnormally rapid or deep respiration (Hyperventilation).
• The CO₂ & H⁺ are potentially toxic products of aerobic & anaerobic metabolism, respectively. Most CO₂ is lost through the lungs, but some is converted to HCO₃⁻, thus contributing important extracellular buffering capacity.

The H₂CO₃ : HCO₃⁻ is one of the buffering systems of the blood. It is a good physiological buffer & act as a front line defence because:
a- it accounts for more than 60% of the blood buffering
b- H+ secretion by the kidney depends on it.
c- it is necessary for efficient buffering by hemoglobin (Hb), which provides most of the blood buffering capacity.

\[ \text{CO}_2 + \text{H}_2\text{O} \leftrightarrow \text{H}_2\text{CO}_3 \leftrightarrow \text{H}^+ + \text{HCO}_3^- \]

by the enzyme CA
The buffer pair HCO_3^- & H_2CO_3 can be expressed in the Henderson – Hasselbalch equation (i.e. weak acid & its conjugate base)
\[
\text{pH} = \text{pKa} + \log \left[ \text{Base} \right] / \left[ \text{Acid} \right]
\]

\[
\text{pH} = \text{pKa} + \log \left[ \text{HCO}_3^- \right] / \left[ \text{H}_2\text{CO}_3 \right]
\]

Normally, \( \text{pKa} = 6.1 \) (constant) & the Ratio of \( \text{HCO}_3^- \) to \( \text{CO}_2 \) or \( \text{H}_2\text{CO}_3 \) (changeable) is considered as 20:1 therefore pH is around 7.0. This ratio is < 20 in Respiratory Acidosis & > 20 in Respiratory Alkalosis.
In Acute Resp. failure (e.g. bronchopneumonia or status asthmaticus), pCO₂ i.e. H₂CO₃ is ↑ and pH is ↓ (Acidosis) the renal tubular mechanism (Kidney) ↑ the rate of HCO₃⁻ production (although limited in short time) but in chronic Resp. failure (e.g. chronic obstructive pulmonary disease), this kidney mechanism is of great importance & tubular cells generate HCO₃⁻ until the ratio is normal 20:1.
When $pCO_2$ & $[H_2CO_3]$ is ↓ & pH is ↑ (Alkalosis) the renal tubules tend to reabsorb less HCO$_3$ (i.e. its excretion is ↑) and the ratio returns to normal & pH is maintained within normal.

This is known as the Primary Compensation mechanism by the kidney & the secondary one is by inducing Hyperventilation for Resp.Acidosis & inducing Hypoventilation for Resp.Alkalosis.
The Biochemical findings:

<table>
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<tr>
<th>Resp. Acidosis</th>
<th>Resp. Alkalosis</th>
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<tbody>
<tr>
<td>pCO₂ ↑</td>
<td>Always ↓</td>
</tr>
<tr>
<td>[HCO₃⁻] ↑ (- Normal (acute) ↑ (chronic))</td>
<td>↓ (- Normal)</td>
</tr>
<tr>
<td>pH ↓</td>
<td>↑</td>
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The Hb in the RBCs act as a buffering agent as an acid through its acidic – COOH (carboxylic) group and basic through its imidazole group of the amino acid Histidine (His) which varies with the pH of the medium (blood plasma).
On oxygenation, the imidazole N group acts as an acid & donates H+ in the medium and vice versa. Acidity of the medium favors delivery of O$_2$ (as in tissues) & alkalinity of the medium favors oxygenation of Hb (as in lung).

**Causes of Resp. Acidosis:**
1- Suppression / depression of respiration as in damage of central nervous system (CNS) e.g. brain damage (trauma, inflammation ... etc), drug poisoning (morphine).
2- Pulmonary Tumors & Emphysema.  
3- Obstruction of Resp. tract & Asthma.  
4- Insufficient pulmonary blood flow ( certain Congenital heart failure ).

• **Causes of Resp. Alkalosis:**  
  1- CNS diseases e.g. meningitis  
  2- Salicylate poisoning ( large doses )  
  3- Hysteria  
  4- High altitude effect.
Arterial Blood
Normal Range (Reference value):
\[ \text{PaCO}_2 = 4.6 - 6 \text{ kPa} \]
\[ \text{PaO}_2 = 9.3 - 13.3 \text{ kPa} \]
Conversion factor to mmHg is multiply by 7.5
\[ \text{HCO}_3 = 24 - 32 \text{ mmol/L} \]
\[ \text{pH} = 7.35 - 7.45 \]