Pathophysiology

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Lecture 7

Learning Objectives

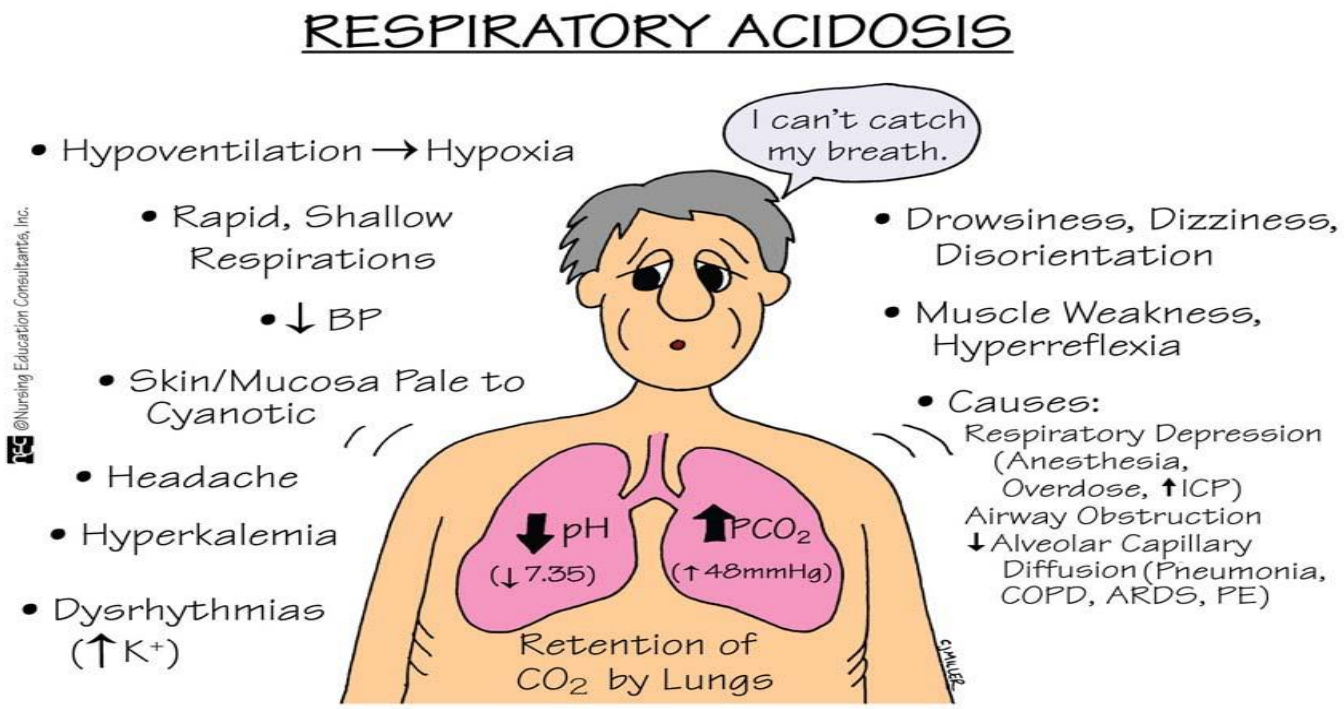
Upon completing this Lecture the students should be able to:

1. Comprehend the Pathophysiology of Respiratory Acidosis.
2. Comprehend the Pathophysiology of Respiratory Alkalosis.

IX. Respiratory Acidosis

A fall in the blood pH occurring due to raised ***Pco_2 > 40mmHg*** *consequent to hypoventilation of lungs (CO2 retention) causes respiratory acidosis.*

Respiratory acidosis is the acid-base disturbance initiated by **an increase in $PaCO_2$**. Primary hypercapnia is a synonymous term.



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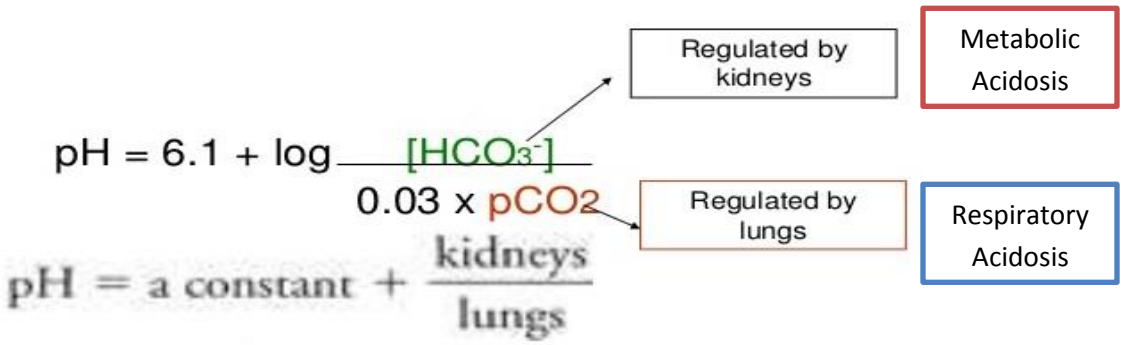
The level of $PaCO_2$ is determined by the interaction of two factors, the rate of carbon dioxide production (VCO_2) and the rate of alveolar ventilation (V_A), as follows:

$$\uparrow PaCO_2 = K \times V_{CO2} / V_A \downarrow$$ ***where K is a constant.***



Most cases of respiratory acidosis reflect a **decrease in alveolar ventilation** leading to hypercapnia.

The **renal response to chronic hypercapnia** includes a transient increase in chloride excretion and generation of hypochloremia. This reduction in plasma chloride concentration balances the increase in plasma bicarbonate concentration, **plasma anion gap remaining unchanged**.

The kidneys compensatory mechanism is by increasing HCO_3^- which will tack a matter of hours to days ….

In Acute respiratory acidosis the kidneys compensation will be one mmol of HCO_3^- to each ten mmHg Pco_2.

While in Chronic respiratory acidosis the kidneys compensation will be four mmol of HCO_3^- to each ten mmHg Pco_2.

Causes:

1. **CNS depression:**
   i) Sedatives.
   ii) CNS disease.
   iii) Obesity-hypoventilation syndrome.
2. **Pleural disease:**
   i) Large pneumothoraxes.
   ii) Pleural effusions).
3. **Lung disease:**
   i. **Air obstruction:** Chronic obstructive pulmonary disease(COPD),acute respiratory distress syndrome (ARDS).
   ii. Musculoskeletal p.
   iii. **Restricted thoracic movement** e.g. in pleural effusion, ascites, pregnancy, kyphoscoliosis.
4. **Neuromuscular disorders** (kyphoscoliosis, Guillain-Barre syndrome, myasthenia gravis, polymyositis & polyneuritis).

Clinical Manifestations

1- **Neurological**: "**hypercapnic encephalopathy**" is mainly represented by irritability, inability to concentrate, headache, anorexia, apathy, confusion, hallucinations, delirium, transient psychosis, progressive narcosis and might lead **to coma**.
2- **Cardiovascular**: Respiratory acidosis causes inhibition of myocardial contractility, decreases in both cardiac output and blood pressure might be observed in severe cases.
3- **Renal**: Salt and water retention.
Clinically, there is peripheral vasodilatation and raised intracranial pressure. If there is severe CO2 retention, patients may develop confusion, drowsiness and coma. The arterial Pco2 level is raised.

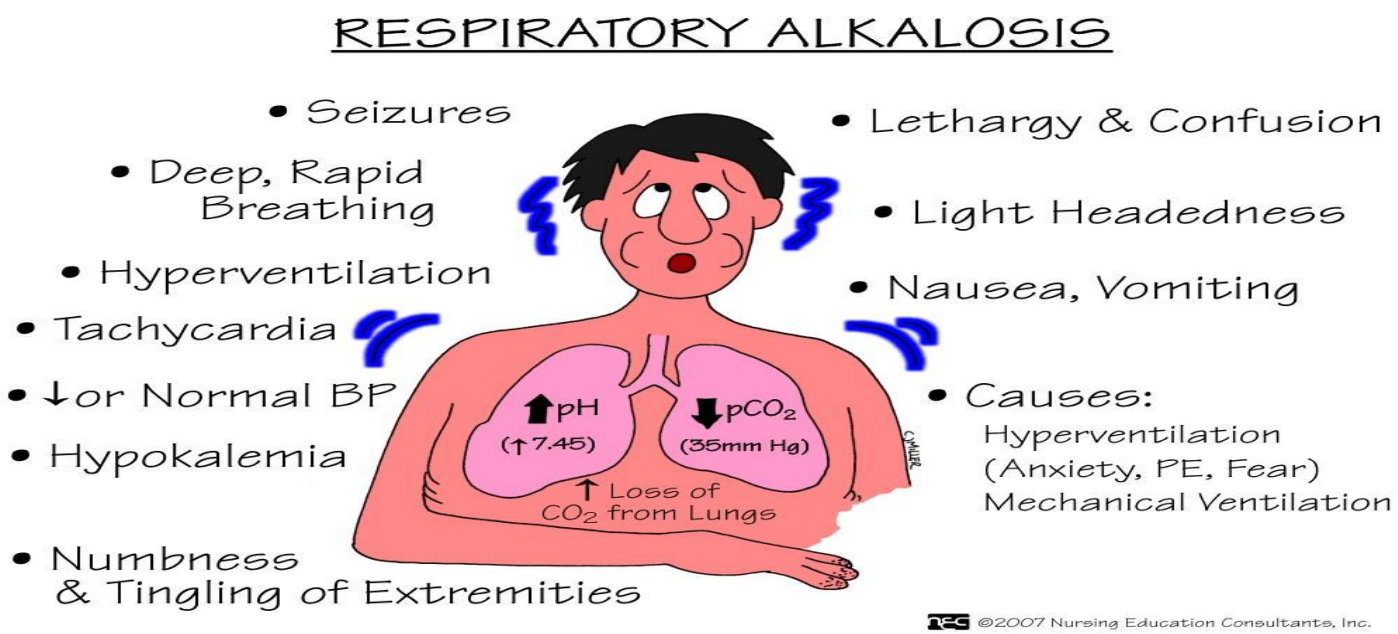
X. RESPIRATORY ALKALOSIS

A rise in the blood pH > 7.45 occurring due to lowered Pco2 < 40mmHg consequent to hyperventilation of the lungs (excess removal of CO2) is called respiratory alkalosis.
Respiratory alkalosis is the acid-base disturbance initiated by a reduction in $PaCO_2$. Primary hypocapnia < 40 mmHg is a synonymous term.

$$\uparrow PH = \frac{HCO3-}{PaCO2} \begin{matrix}\uparrow \\ \downarrow\end{matrix}$$

Metabolic Alkalosis

Respiratory Alkalosis



Pathophysiology

1. Most cases of respiratory alkalosis reflect **an increase in alveolar ventilation**.
2. Primary decreases in carbon dioxide production are generally attended by parallel decreases in alveolar ventilation, thus preventing expression of respiratory alkalosis.
However, in the presence of constant alveolar ventilation (i.e., mechanical ventilation), decreased carbon dioxide production (e.g., sedation, skeletal muscle paralysis, hypothermia, hypothyroidism) can cause respiratory alkalosis.

Adaptation

1. Acute adaptation

a. It is completed within 5-10 min from onset of hypocapnia.
b. It originates principally from alkaline titration of the body's non bicarbonate buffers (hemoglobin, intracellular proteins and phosphates, plasma proteins):

$HBuf \leftrightarrow H^+ + Buffer^-$

$HCO3^- + H^+ \leftrightarrow H2CO3 \leftrightarrow H_2O + CO_2$

where HBuf refers to the acid component and $Buffer^-$ to the base component of non-bicarbonate buffers.

In Acute Respiratory alkalosis the kidneys compensate by decreasing the Bicarbonate ($HCO3^-$) falls 2 mEq/l for each decrease 10 mmHg of $PaCO_2$ there for the PH will decrease by 0.08.

2. Chronic adaptation

a. It requires 2-3 days of sustained hypocapnia for completion.
b. It originates from down regulation of renal acidification mechanisms

i. A transient decrease in urinary net acid excretion (mostly a fall in ammonium excretion).
ii. A persistent decrease in the rate of renal bicarbonate reabsorption.

In Chronic Respiratory alkalosis the kidneys compensate by decreasing the Bicarbonate (HCO3-) falls 5 mEq/l for each decrease 10 mmHg of PaCO2 there for the PH will decrease by 0.02.

Causes:

Primary hypocapnia is the most frequent acid-base disturbance encountered, occurring in normal Anxiety, panic attaches, pregnancy & high-altitude residence.

1- Hypoxemia or tissue hypoxia:

a) High altitude.
b) Bacterial or viral pneumonia.
c) Aspiration of food.
d) Foreign body or vomitus.
e) Severe anemia.
f) Drowning.
g) Hypotension…etc.

2- Drugs or hormones: Nikethamide, ethamivan, Doxapram, Xanthines, Salicylates, Catecholamines, Angiotensin II, Vasopressor agents, Progesterone, Nicotine.

3- Miscellaneous:

a) Pregnancy.
b) Sepsis.
c) Hepatic failure.
d) Mechanical hyperventilation.
e) Heat exposure.

Clinical manifestations

1- **Central Nervous System**:

a) Reduction in intracranial pressure,
b) Light-headedness,
c) Confusion.
d) Generalized seizures.

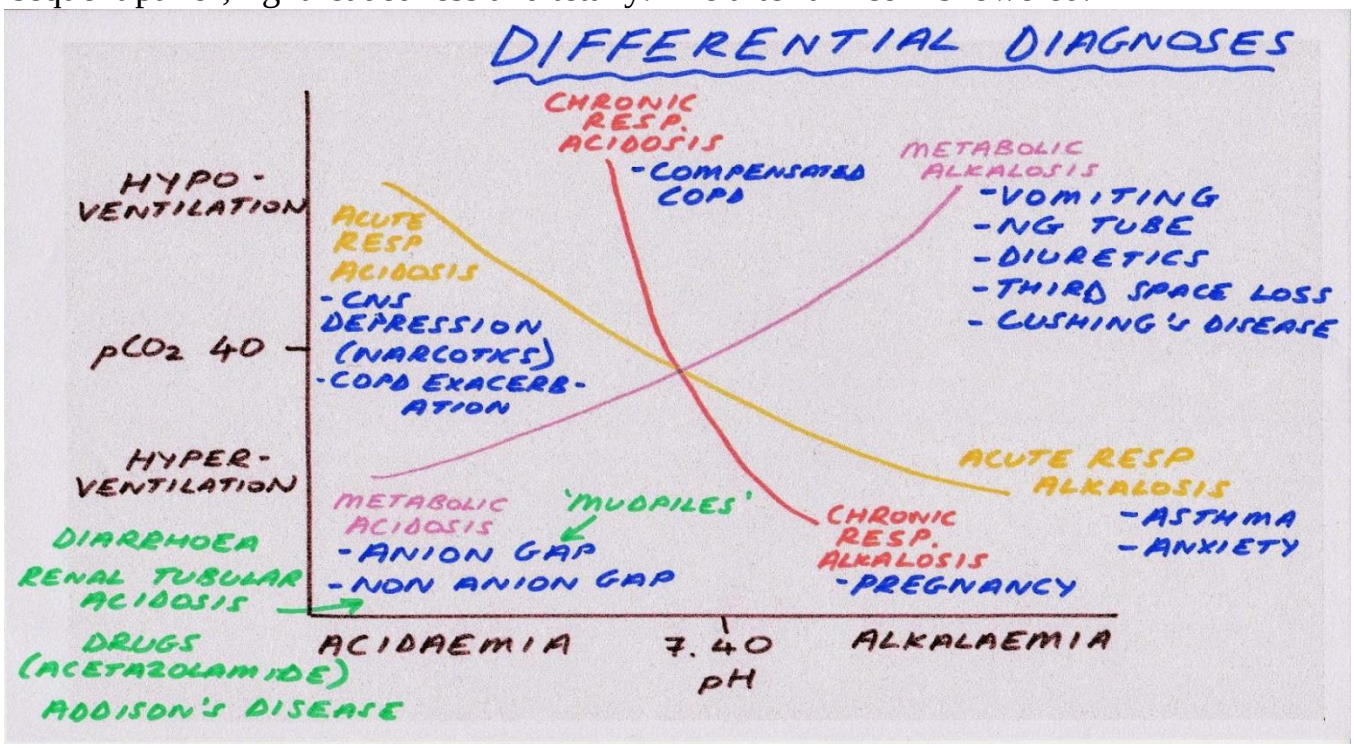
2- **Cardiovascular System**:

a) Normal or decreased blood pressure.
b) Cardiac arrhythmias.
c) Peripheral vasoconstriction.
d) Angina pectoris & even Ischemic changes.

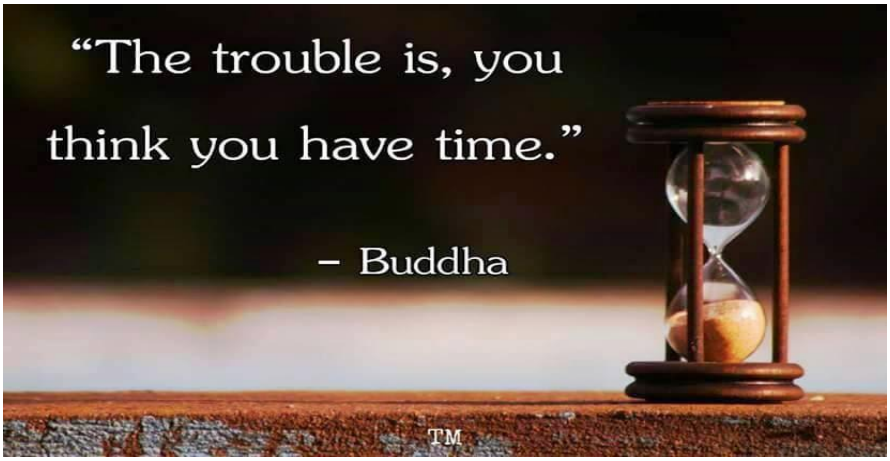
3- **Neuromuscular System**:

a) Numbness and paresthesias of the extremities.
b) Muscle cramps.
c) Circumoral numbness.
d) Laryngeal spasm.
e) Manifestations of tetany.

The patients with respiratory alkalosis are characterized by peripheral vasoconstriction and consequent pallor, lightheadedness and tetany. The arterial Pco2 is lowered.



- Whenever the PH and the $PaCO_2$ are moving in the same direction (i.e. up or down), **a metabolic process is occurring.**
- Whenever the PH and PCO_2 are moving in opposite directions, **a respiratory process is occurring.**



Thank you....