

## Acid Base Introduction

- K pp. 113-115
- C pp. 46-47
- B pp. 290-292
- Paramedic Care vol. 1 p. 191

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## Objectives

Upon completion of this lecture the learner should be able to:

- Discuss the role of hydrogen ions in normal metabolism.
- Discuss how the arterial blood pH is a reflection of the body's total number of hydrogen ions .
- Discuss the normal buffering and compensatory mechanisms for handling an excess of either a metabolic or a respiratory acid.

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## Objectives (cont.)

- Define Respiratory Acidosis, Respiratory Alkalosis, Metabolic Acidosis, Metabolic Alkalosis .
- Give clinical examples of uncompensated: Respiratory Acidosis, Respiratory Alkalosis, Metabolic Acidosis, Metabolic Alkalosis .
- Give clinical examples of compensatory buffering mechanisms for handling excess of a metabolic acid and of a respiratory acid.

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## Treatment Priorities - review

When fluids and electrolytes are altered, these should be corrected in the following order:

- volume
- **pH**
- potassium, calcium, and magnesium
- sodium and chloride

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## Physiology of Hydrogen Ions

- H<sup>+</sup> origin: a product of normal metabolism
- H<sup>+</sup> excretion:
  - lungs
  - kidney
    - kidneys maintain H<sup>+</sup> concentration by :
      - remove or retain HCO<sub>3</sub><sup>-</sup>
      - remove or retain organic acids (e.g., lactic acid)

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## Buffering of H<sup>+</sup> ions

- Chemical
  - H<sup>+</sup> + HCO<sub>3</sub><sup>-</sup>  $\rightleftharpoons$  H<sub>2</sub>CO<sub>3</sub>  $\rightleftharpoons$  H<sub>2</sub>O + CO<sub>2</sub>
- Cellular
  - “soaking up” H<sup>+</sup> ions
- Lungs
  - blow off CO<sub>2</sub>
- Kidneys
  - retain HCO<sub>3</sub><sup>-</sup>

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## Buffering of H<sup>+</sup> ions

- Chemical
  - $H^+ + HCO_3^- \rightleftharpoons H_2CO_3 \rightleftharpoons H_2O + CO_2$
  - instantaneous
- Cellular
  - seconds
- Lungs
  - minutes
- Kidneys
  - days

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## Definitions

- Normal pH
  - 7.35 - 7.45
- Acidosis
  - pH < 7.35
- Alkalosis
  - pH > 7.45

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## Definitions - Respiratory



- Respiratory (volatile) acid
  - reflected by : pCO<sub>2</sub> level
- Normal
  - pCO<sub>2</sub> : 35 - 45 [ 40 ]
- Acidosis
  - pCO<sub>2</sub> > 45
- Alkalosis
  - pCO<sub>2</sub> < 35

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## Definitions - Metabolic



Metabolic (nonvolatile) acid

- reflected by :  $HCO_3^-$  level

Normal Metabolism

- $HCO_3^-$  : 24 - 28 [ 25 ]

Metabolic Acidosis

- $HCO_3^- < 24$

Metabolic Alkalosis

- $HCO_3^- > 28$

## Respiratory Acidosis

- Pathophysiology
  - hypoventilation
    - poor  $CO_2$  exchange
    - $CO_2$  retention ;  $O_2$  may or may not be adequate
- Blood gas abnormalities
  - hypoxia (usually, but not always)
  - hypercarbia ( $pCO_2 > 40$ )
  - if uncompensated :  $HCO_3^-$  "nl" : 25

## Respiratory Acidosis

Clinical Presentation

- Sx.:
  - dyspnea
  - hx of WOB +/- ALOC
- Signs:
  - distress (RR, retractions, ...cyanosis)

Clinical Example of Uncompensated Respiratory Acidosis :

- Acute asthma attack
  - $pCO_2 : 60$  ( $pCO_2 > 40$ )
  - pH : 7.3
  - $HCO_3^- : 25$



## Respiratory Alkalosis

- Pathophysiology
  - hyperventilation
    - excessive CO<sub>2</sub> release
    - poor O<sub>2</sub> exchange may or may not occur
- Blood gas abnormalities
  - pO<sub>2</sub> < 80 [ sat. < 93 % ] ( suggests possible PE, ... )
  - hypocarbia (pCO<sub>2</sub> < 40)
  - pH : 7.6
  - if uncompensated : HCO<sub>3</sub> : 25

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## Respiratory Alkalosis

- Clinical Presentation
  - Sx.: dyspnea : hx of WOB +/- ALOC
  - chest pain
  - Signs: increased RR ( no cyanosis,... )
- Clinical example of uncompensated Respiratory Alkalosis :
  - Acute hyperventilation attack
  - pCO<sub>2</sub> : 20 (pCO<sub>2</sub> < 40)
  - pH : 7.6
  - HCO<sub>3</sub> : 25

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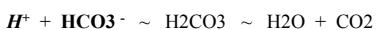
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## Metabolic Acidosis



- Pathophysiology
  - excess H<sup>+</sup> ions consume the HCO<sub>3</sub><sup>-</sup>
  - this causes a HCO<sub>3</sub><sup>-</sup> deficit
  - if uncompensated, then the pCO<sub>2</sub> will be 40
- Blood gas abnormalities
  - HCO<sub>3</sub><sup>-</sup> : ~ 8 (depletion due to excessive acid)
  - pH : ~ 7.2
  - if uncompensated, then the pCO<sub>2</sub> will be 40

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## Metabolic Acidosis

- Clinical presentation of *acute* metabolic acidosis
  - Sx.: no dyspnea
  - Signs: If uncompensated : RR unchanged  
(If compensated : RR rapid, deep)
- Clinical example of uncompensated **metabolic** acidosis:
  - acute TCA OD
    - HCO<sub>3</sub> : 8
    - p H : 7.2
    - pCO<sub>2</sub> : 40 (uncompensated )



## Metabolic Alkalosis

- $$\text{H}^+ + \text{HCO}_3^- \simeq \text{H}_2\text{CO}_3 \simeq \text{H}_2\text{O} + \text{CO}_2$$
- Pathophysiology
    - excessive HCO<sub>3</sub> ions will consume H<sup>+</sup>
    - this causes a “ H<sup>+</sup> deficit ”
    - if uncompensated, then the pCO<sub>2</sub> remains 40
  - Blood gas abnormalities of acute metabolic alkalosis
    - pCO<sub>2</sub> : 40 (no respiratory component)
    - p H : 7.6
    - HCO<sub>3</sub> : 45

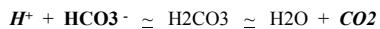


## Metabolic Alkalosis

- Clinical Presentation
  - acute metabolic alkalosis is rare.
  - Sx. + “ full arrest, 2 amps of bicarb ;
  - Signs: with ‘adequate ventilations’ ”
- Clinical example of uncompensated or straight metabolic alkalosis :
  - “2 amps of bicarb”
    - HCO<sub>3</sub> : 45
    - p H : 7.6
    - pCO<sub>2</sub> : 40 (no respiratory component)



## Mixed Acidosis and Alkalosis



- Pathophysiology
  - excess  $H^+$  ions can also be compensated for by blowing off  $CO_2$  the and moving the equation to the respiratory side
  - in an uncompensated patient,
    - the  $pCO_2$  is 40
  - but in the **compensated** patient,
    - the  $pCO_2$  will be  $< 40$

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## Mixed Acidosis and Alkalosis



- Example of blood gas abnormalities of **compensated** DKA
  - $pCO_2 : 18$
  - $pH : 7.33$
  - $HCO_3 : 12$  (partial depletion due to excessive acid)

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## Treatment

- $CO_2M_3E BI_{NS}G$
- Treatment for acidosis
- always begins with hyperventilation
- $H^+ + HCO_3^- \simeq H_2CO_3 \simeq H_2O + CO_2$
- or
- $H^+ + HCO_3^- \simeq H_2CO_3 \simeq H_2O + CO_2$

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## Summary

We have discussed :

- Homeostasis of CO<sub>2</sub> and H<sup>+</sup>
- Normal buffering and compensatory mechanisms for handling an excess of either a metabolic or a respiratory acid.
- Definitions and clinical examples of some uncompensated Respiratory Acidosis and Metabolic Acidosis and one clinical example of compensation.

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