

### ACIDOSIS – production of H<sup>+</sup>

- TISSUE METABOLISM...12 500meq/d CO<sub>2</sub>
- LIVER utilization of AMA for gluconeogenesis H<sub>2</sub>SO<sub>4</sub>, H<sub>3</sub>PO<sub>4</sub> a major H<sup>+</sup> load ..... 50 meq/day
- STRENUOUS EXERCISE .....lactic acid
- DIABETES MELLITUS ..... ketoacidosis
- INGESTION OF ACIDS, DIARRHOEA
- FAUILURE OF KIDNEY

### ALKALOSIS

- Vomit
- Fruits, vegetables

### Consequence of pH disturbances

- Change of tertiary and quaternary structure of proteins
- ↓ of activity of enzymes
- changes in permeability of membranes
- changes in distribution of electrolytes

### Regulation of pH of body fluids

- Buffer systems... immediately, small capacity
- Lungs... CO<sub>2</sub> ...quickly, limited capacity
- Kidney...H<sup>+</sup>, HCO<sub>3</sub><sup>-</sup> .slowly, high capacity

### BUFFERS – BUFFER CAPACITY OF BLOOD

- BUFFER- weak acid
- The Henderson-Hasselbalch equation



$\leftarrow + H^+$  added strong acid

$\Rightarrow + OH^-$  added strong base (H<sup>+</sup> bound to water)

Relative acidity of weak acids is expressed by dissoc. constant

- Negative logarithm of d.const. is pK ...it is pH, at which concentration of H<sup>+</sup> and undissociated molecules equals

$$pH = pK + \log \frac{[A^-]}{[AH]}$$

The highest buffering capacity if pH = pK

buffer capacity of blood - 48mmol/l

### BUFFERS IN BLOOD

- Plasma proteins dissociate

Free carboxyl: R-COOH  $\rightleftharpoons$  R-COO<sup>-</sup> + H<sup>+</sup>

Free aminogroups: R-NH<sub>3</sub><sup>+</sup>  $\rightleftharpoons$  R-NH<sub>2</sub> + H<sup>+</sup>

- Hemoglobin (histidin residua)

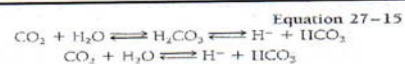
Buffering capacity 6x 1 than that of proteins

Hb is weaker acid than HbO<sub>2</sub> =>

Hb is better buffer than HbO<sub>2</sub> (↓ buffering capacity in lungs – easier release of CO<sub>2</sub>...opposite effect in tissue helps binding CO<sub>2</sub>)

- Phosphates H<sub>2</sub>PO<sub>4</sub><sup>-</sup>  $\rightleftharpoons$  HPO<sub>4</sub><sup>2-</sup> + H<sup>+</sup>

- Bicarbonate system HCO<sub>3</sub><sup>-</sup> + H<sup>+</sup>



Moreover, we can define a dissociation constant for this pseudo-equilibrium:

$$K = \frac{[H^+][HCO_3^-]}{[CO_2]} \quad \text{Equation 27-16}$$

☛ In logarithmic form, this equation becomes:

$$pH = pK + \log \frac{[HCO_3^-]}{[CO_2]} \quad \text{Equation 27-17}$$

Finally, we may express [CO<sub>2</sub>] in terms of PCO<sub>2</sub>, recalling from Henry's Law that [CO<sub>2</sub>] = s · PCO<sub>2</sub>:

$$pH = pK + \log \frac{[HCO_3^-]}{s \cdot PCO_2} \quad \text{Equation 27-18}$$

This is the Henderson-Hasselbalch equation, a loga-

### DISTURBANCES OF ACID/BASE BALANCE

Physiologic value	metabolic	respiratory
	acidosis    alkalosis	acidosis    alkalosis
$\text{HCO}_3^-$ 20	$\text{HCO}_3^-$ $\text{HCO}_3^-$	$\text{HCO}_3^-$ $\text{HCO}_3^-$
$\text{CO}_2$ 1	$\text{CO}_2$ $\text{CO}_2$	$\text{CO}_2$ $\text{CO}_2$
<b>Compensation</b>		
	hyper/hypo ventilation	kidney excretion
	$\text{HCO}_3^-$ $\text{HCO}_3^-$ $\text{HCO}_3^-$	$\text{HCO}_3^-$ $\text{HCO}_3^-$ $\text{HCO}_3^-$
	$\text{CO}_2$ $\text{CO}_2$ $\text{CO}_2$	$\text{CO}_2$ $\text{CO}_2$ $\text{CO}_2$

### COMPENSATION OF ACIDOSIS

- NEAD OF  $\text{NaHCO}_3$  mmol/l:  
 $\text{BE} \times 0,3 \times \text{WEIGHT}$
- Isotonic solution 1,39 % (0,167mol/l)

½ of amount needed, new measurement, (examination of  $\text{K}^+$ )

### ACIDOBASIC BALANCE AND MOVEMENT OF $\text{K}^+$ BETWEEN ECF AND ICF

PLASMATIC CONCENTRATION 3.5 – 5.3 mmol/l

ECF                      ICF

Acidosis                      alkalosis                      hypokalemia

↑Kalemia                      ↓ kalemia                      alkalosis

kaliuria

### Dependence of kaliemie on pH - at a constant total content of $\text{K}^+$

pH	7.2	7.4	7.6	
$\text{K}^+$	4.9	4.0	3.2	mmol/l
	6.2	5.0	3.8	
	7.3	6.0	4.5	

Physiologic PLASMATIC CONCENTRATION 3.5 – 5.3 mmol/l

### METABOLIC ACIDOSIS

- CAUSES
  - ↑Production of acids (diabetes, loss of weight, anaerobic glycolysis)
  - Loss of bicarbonate (diarrhea, inhibitors of carbonic anhydrase)
  - Intake of acids
  - Renal failure of acid secretion
- RESPIRATORY COMPENSATION
  - $\text{H}^+$  is bound ( $\text{H}^+ + \text{HCO}_3^- \rightarrow \text{H}_2\text{O} + \text{CO}_2$ )
  - EXPIRATION (Kussmaul respiration)-
  - ALKALIC RESERVE DECREASES

### METABOLIC ALKALOSIS

- CAUSES
  - Loss of  $\text{H}^+$  (vomit, deficiency of  $\text{K}^+$ )
  - Intake of bases
  - Production of bases (oxidation of lactate)
- RESPIRATORY COMPENSATION is limited – hypoventilation - hypoxia +  $\text{CO}_2$  – stim of respiration
- EXCRETION of BICARBONATE by KIDNEY
- $\uparrow\text{pH} \rightarrow \downarrow\text{Ca}^{++} \rightarrow \text{tetany}$

## RESPIRATORY ACIDOSIS

- CAUSES

↓ Ventilation (barbiturates)

Reduction of lung's tissue

- H<sup>+</sup> BUFFERED BY NONBICARBONATE BUFFERS
- EXCRETION of H<sup>+</sup> (NH<sub>4</sub><sup>+</sup>) by KIDNEY  
lag by 1-2 days

## RESPIRATORY ALKALOSIS

- CAUSES

hyperventilation (hypoxia, psychiatric)

- H<sup>+</sup> RELEASED BY NONBICARBONATE BUFFERS
- EXCRETION of BIKARBONATE by  
KIDNEY
- First aid - quieting, rebreathing