Kidney

المحاضرة السابعة

- الجزء الأول -

Acid Base balance

بتاريخ ٣٠ مارس ٢٠١٨

المحاضرة الأخيرة

Kidney

المحاضرة السابعة

- الجزء الثاني -

Renal Function Tests
Chronic Renal Failure

بتاريخ ٣٠ مارس ٢٠١٨

It is an early test, specific test & quantitative test.

[4] Imaging techniques

ⓐ Plain x ray : shows opaque calculi & calcifications

ⓑ Intravenous urography IV iodine e.g diodrast, then series of radiographs. to show nonopaque calculi, stricture, dilated pelvis and nonfunctioning kidney.

ⓒ Ultrasonography : shows renal size, position, tumor, cysts or dilatation of collecting system in obstruction.

ⓓ Computed tomography CT shows tumor or cysts

Treatment of chronic renal failure

[1] Dialysis treatment : Diffusion of molecules via a semipermeable mem

ⓐ Kidney machine (hemodialysis) course 6 hours 2-3times/W remove urea 50-250g/6H & toxins and add HCO_3^- in acidosis

ⓑ Continuous ambulatory peritoneal dialysis CAPD

Fluid is collected from peritoneal cavity 4 to 8 hours later. Dialysis fluid should contain less K^+ than plasma, be buffered, free of urea. and has higher OP than normal plasma → -- ABP

[2] Kidney transplants: should correct :

GFR to 120-150 ml, abnormal Ca homeostasis & anemia.

Acid-base balance

- Acid base state = Acid base state of ECF

Acid	Base
Molecule produces H^+ i.e proton donor	Molecule combines with H^+ i.e H^+ acceptor
Strong e.g 100% dissociation	Strong e.g NaOH 100% dissociat.
Weak e.g H_2CO_3 1% "	Weak e.g NH_4OH 1% "

Free H^+ conc. in ECF is very low 0.00004 mmol/L 40 nmol/L

- Determined by measuring in ARTERIAL blood 2 out of 3
  1. $[H^+]$ 35 - 45 nanomol / L (pH 7.35 - 7.45)
  2. PCO_2 35 - 45 mmHg.
  3. $[HCO_3^-]$ 22 - 26 mEq/L.

  $H^+ \propto \frac{PCO_2}{HCO_3^-}$

- Protein mediated processes depend on pH
  i.e enzymes, transport molecules & contractile molecules
- Sources of H^+:
  1. Ingested : Food
  2. Metabolism
     - a CHO generates 12.000 - 20.000 mmol/day H_2CO_3 (volatile acids)
     - b Ptn & lipid generate 60 mmol / day H_2SO_4 H_3PO_4 (Fixed acids)
     - c Lactic acids in severe exercise due to inadequate O_2
     - d Ketoacids in DM

  Amount of H^+ in ECF is very small compared to amount of H^+ produced/D
  PH - log to base 10 of H^+ conc = - $\log_{10} 0.00004$ = 7.4 alkaline
  Death occurs if pH falls below 6.8 or rises above 8.0.
- Regulation of acid base balance :
  1. Chemical buffers minimize change in pH (fraction of a sec.)
  2. Respiratory system removes H^+ of volatile acids (minutes)
  3. Kidney (most important) removes H^+ of fixed acids (hours to days) restores ECF buffers

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Chemical buffers 7 points

- Substances which minimise changes of pH.
- Act within fraction of a second
- Molecules that combine with or release H^+
  Buffer system = weak acid + salt of its conjugate base
- Henderson Hasselbach equation :

$$pH = pK + \log_{10} \frac{[Salts]}{[Acids]}$$

Chronic renal failure

Azotemia

Anemia ↓ erythropoietin

Acidosis ↑ Acid waste products ↓ Buffers (Hb)

Hypertension ↑ Renin

Abnormal Ca^{++} homeostasis ↓ 1-25 $(OH)_2$ CC

$$[H^+] \propto \frac{\text{Physical } CO_2}{\text{Chemical } CO_2} \propto \frac{PCO_2}{HCO_3^-}$$

$$pH \propto -\log_{10}[H^+]$$

$$\propto -\log 10 \frac{[\text{Acid}]\ (H_2CO_3)}{[\text{Salt}]\ (HCO_3^-)}$$

$$\propto +\log 10 \frac{[\text{Salt}]}{[\text{Acid}]}$$

$$= pK + \log 10 \frac{[\text{Salt}]}{[\text{Acid}]}$$

If $pH = pK$

$[\text{salt}] = [\text{Acid}]$

i.e 2 components of buffer system r equal

- Effectiveness of any buffer system depends on:
  1. Amount of buffer pair
  2. pK — Most effective when pH = pK
- Three major buffers:

[1] Bicarbonate

Amount 24 mmol/L — Disadvantage

pK 6.1

2 components can be regulated < lung (PCO_2), Kidney (HCO_3^-) — Advantage

[2] Phosphate

Amount 1 mmol/litre — Except in ICF & tubular fluid

PK 6.8 i.e close to pH

[3] Ptn — Most plentiful.

[a] Plasma ptns

[b] Hb — 6 times buffering power of plasma ptn.
Large amount 700 g in adult — 38 histidine/molecule
Deoxy Hb is better buffer than oxy Hb
It buffers CO_2 (Cl^- shift phenomenon)
In renal failure, ↓↓ Hb leads to more acidosis

[c] Intracellular ptns & organic phosphate.
Advantages: Backup extracellular buffer
Most plentiful.
Disadvantages: Slow (hours); cell mem has permeability to H^+
H^+ is exchanged with K^+ & Ca^{++} → depletes cells of K^+ and bones of Ca^{++}

- Isohydric principle: The buffer systems buffer each other by shifting of H^+ ions from one buffer to others

Respiratory control

- Controls blood PCO_2
- Mechanisms via peripheral chemoreceptors

++$[H^+]$ → stim. of periph. chem → -- PCO_2

- Effectiveness of respiratory control.

1 Returns $[H^+]$ 2/3 the way back to normal. within 1 - 12 minutes

2 1 - 2 times buffering power of chemical buffers.

3 Limited ability PCO_2 changes have ≠ effect on resp.

Example

$$10\ HCl + 26\ NaHCO_3 \longrightarrow 10\ NaCl + 10\ H_2CO_3 + 16\ NaHCO_3$$

i.e H_2CO_3 ++ 10 mmol/L $NaHCO_3$ -- 10 mmol/L

According to Henderson-Hasselbalch equation:

$$pH = 6.1 + \log \frac{26 - 10}{1.3 + 10} = 6.1 + \log \frac{16}{11.3} = 6.25$$

incompatible with life

However, hypervent. eliminates 10 mmol/L of carbonic acid.

$$So,\ pH = 6.1 + \log \frac{26 - 10}{1.3} = 6.1 + \log \frac{16}{1.3} = 7.3$$

compatibe with life

Renal control

- It is the most powerful.
- Returns pH back to normal within 12 - 14 Hours.
- Mechanism discussed .

Acid - Base disturbance table page 30

pH depends on $\frac{CO_2 \text{ in chemical}}{CO_2 \text{ in physical}}$ i.e $\frac{[HCO_3^-]}{PCO_2}$

So, disturbace is caused by changes either in $[HCO_3^-]$ or PCO_2

It is corrected by change in the second system

→ ratio return to normal but HCO_3^- & PCO_2 are (++) or (--)

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Fate of H^+ secreted:

Three important reactions remove Free H^+

① In PCT — pH of tubular fluid is very little changed.

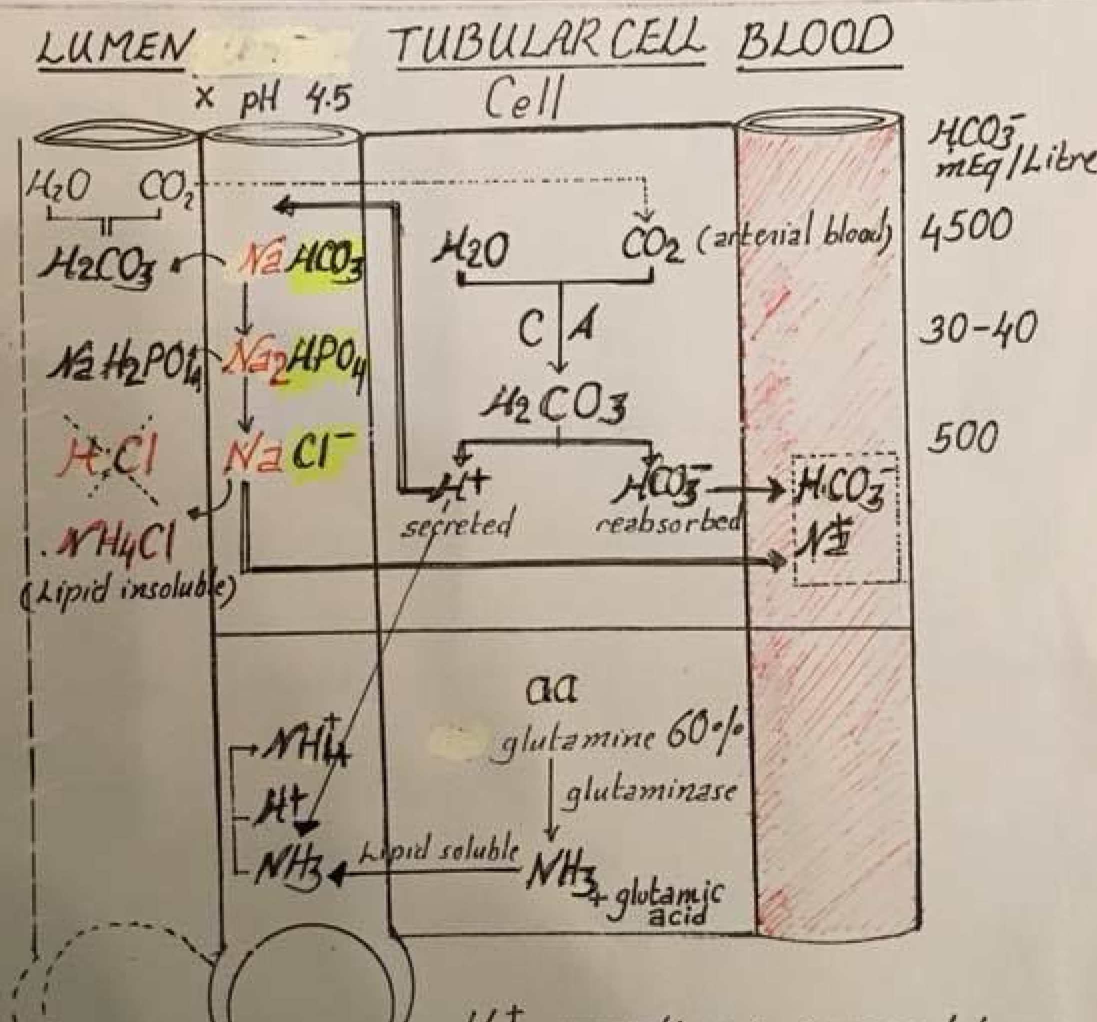
Buffering by $NaHCO_3 \rightarrow H_2CO_3 \xrightarrow{CA} H_2O + CO_2$ diffuses to cell.

② In DCT & collecting tubules — pH never falls below 4.5 (H^+ secretion stops)

Buffering by phosphate buffer $\rightarrow$ titratable acidity

Buffering by $NH_3 + H^+ \rightarrow NH_4 + Cl^- \rightarrow NH_4Cl$

In all 3 buffers Na is reab. with HCO_3^- → ++ $NaHCO_3$ in bl (alkali reserve)
formed in tubular cells



H^+ secretion is increased by:

1. Aldosterone
2. ++ intracellular CO_2
3. -- intracellular K^+

Acidosis

	Respiratory	Metabolic
↓ pH	$\frac{[HCO_3^-]}{\uparrow PCO_2}$ HYPOVENT	$\frac{\downarrow [HCO_3^-]}{PCO_2}$
Causes	1 Resp. centre -- 2 " nerves 3 Resp muscles paralysis 4 Chest cage 5 Lung diseases Obstr. or restrictive	1 DM 2 Shock 3 Loss of intest content 4 Renal failure
↑↓ pH compensation	1 Chemical buffers 2 ↑ $[HCO_3^-]$ Renal 3 ↑ PCO_2 Resp Limited	↓ $[HCO_3]$ ↑ ↓ PCO_2 HYPERVEN
Laboratory diagnosis 22-28 mEq/L 35-45 mmHg	pH meter Alkali reserve + Arterial PCO_2 +	− −
Clinically CNS Resp.	Drowzy & finally comatosed HYPO ventilation	HYPER

Alkalosis

	Respiratory	Metabolic
↑ pH	$\frac{[HCO_3^-]}{\downarrow PCO_2}$ HYPERV	$\frac{\uparrow [HCO_3^-]}{PCO_2}$
Causes	1 High altitude 2 Early in exercise 3 Fever 4 Meningitis 5 Psychological dyspnea or anxiety	Persistant vomiting Diuretics HYPO volemia, kalemia 5 Cushing & Conn's
↓↑ pH Compensation	1 Chemical buffers 2 ↓ $[HCO_3^-]$ Renal 3 ↓ PCO_2 Resp Limited	↑ $[HCO_3^-]$ ↓ ↑ PCO_2 HYPOVEN
Laboratory diagnosis Arterial blood	pH meter Alkali reserve − Arterial PCO_2 −	+ +
Clinically CNS Respiration	Tetany & convulsion HYPER ventilation	HYPO

N.B Resp. compensation is less powerful in metabolic alkalosis than in metabolic acidosis

Kidney function tests

All values ++ in renal insufficiency

1 Blood analysis:

ⓐ Blood urea: 20 - 40 mg / dl nonspecific rough test
++ liver diseas & ++ ptn intake & dehydration

ⓑ Plasma creatinine: 0.6 – 1.5 mg/dl more accurate.

ⓒ Potassium: 3.5 – 5 mEq/L

ⓓ Inorganic phosphate: 2.6 - 4.5 mg/dl

ⓔ Serum uric acid: ♂ 3.6 - 8.5 ♀ 2.3 - 6.6 mg/dl

ⓕ Blood pH: arterial 7.4 venous 7.35

2 Urine analysis:

ⓐ Volume 500 - 1500 ml / day
Polyuria: DM, DI & diuretics Oliguria: Dehydration acute renal failure
Anuria: Acute vascular thrombosis & total urinary obstruction.

ⓑ Sp. gr 1010 - 1020 Low: DI High: DM
Low fixed at 1010 = plasma in chronic renal failure

ⓒ H_2O diuresis test: evacuates bladder then drink 1.5 L & collect urine for 4 hours Normal results 800 ml sp.gr < 1003 OP below 80 mOsm.

ⓓ H_2O conc. test: evacuates bladder & stop drinking for 12 hours
Normal results -- urine vol sp. gr 1025-1030 OP of urine > 900 mOsm.

3 Tests depending on blood and urine analysis

Plasma clearance

ⓐ Creatinine clearance measures GFR

ⓑ PAH clearance measures ERPF

ⓒ Urea clearance measures Renal tubular function

Vol of urine 2 ml or more / min	Urine volume less than 2 ml / min
Maximal urea clearance $C_m = \frac{U \times V}{P}$ Normally 70 ml / min.	Standard urea clearance $C_s = \frac{U \times \sqrt{V}}{P}$ Normally 54 ml / min.

Urea clearance 5% below normal indicates renal impairement

تم غلق القناة الرئيسية من اليوتيوب
اشترك في القناة الجديدة
لتصلك المحاضرات ودياجرامات 2018

Ali Gamal

رابط المحاضرة على اليوتيوب
اضغط هنا

Part 1 >> https://youtu.be/B5O3B1dH7gg
Part 2 >> https://youtu.be/DUlJ0R8CzEs