DISORDERS OF WATER, ELECTROLYTE & ACID-BASE HOMEOSTASIS

LECUTRE IN PATHOPHYSIOLOGY
GENERAL MEDICINE
2024/2025

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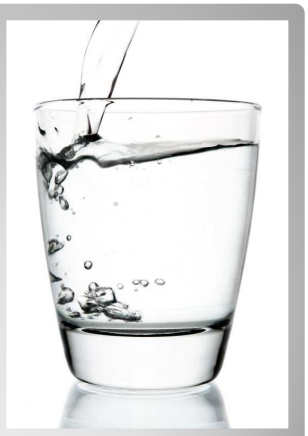

Water

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AGE DEPENDENCE OF FLUID HOMEOSTASIS

Age	Total water %	Daily exchange %
newborn	79	
3-6 mo.	70	14-16
7-12 mo.	60	12-15
adult man	60	2-4
adult woman	51	2-4

Newborns -ECS > ICS, danger of dehydratation
In old age - impaired adaptation, danger of dehydratation + less muscles, much adipose tissue – less water
Women – much addipose tissue, less water than men
Obese people – much fat, less water

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DISTRIBUTION OF WATER IN HUMAN BODY

Compartment	Volume litres	% of body mass	% of total water
ICS	28	40	67
ECS	14	20	33
ISF	11	15,7	26
IVF	3	4,3	7
SUMMA	42	60	100

Amount of water in body of young adult man, weight 70 kg

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Water intake

- beverages 1,0 - 1,5 l/d
- water in food cca 1 l/d
- water from metabolism cca 0.3 l/d

oxidation of	100 g proteins	35 ml water
	100 g sugar	60 ml water
	100 g fat	107 ml water

- Total intake of water cca 2.0 – 2.5 l/d

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Water output

- Urine 1.0 – 1.5 l/d
- Perspiratio 0.3 – 0.6 l/d
  - Skin (sweating) 0.2 – 0.4 l/d
    (more – hot environment, physical activity, fever)
  - Lungs – respiration cca 0.2 l/d
- feces 0.1 – 0.2 l/d (more in diarhea)

- Increased output
  - vomiting
  - bleeding
  - redistribution of water - edema

- Total output of water cca 2.0 – 2.5 l/d

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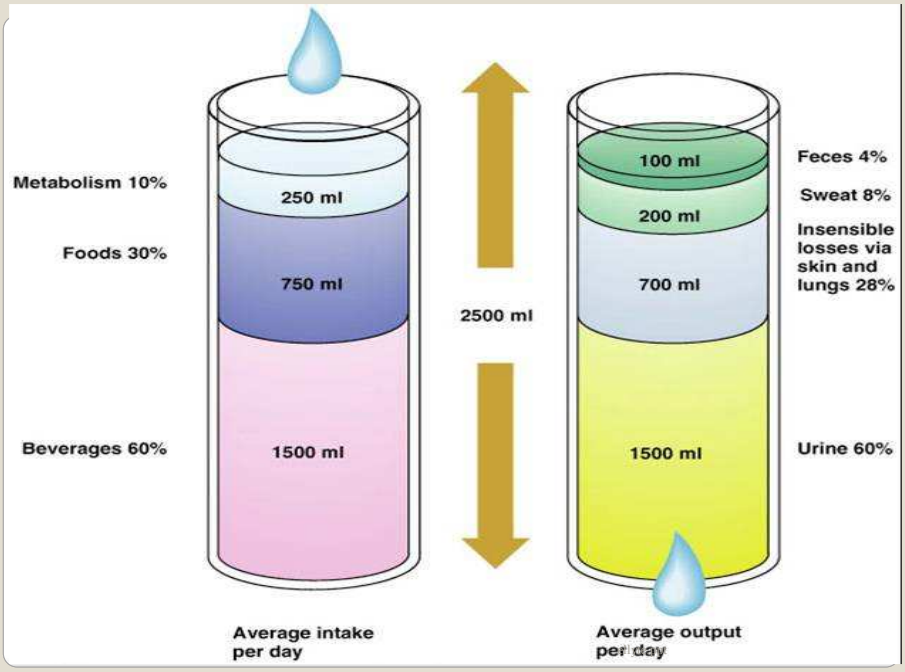

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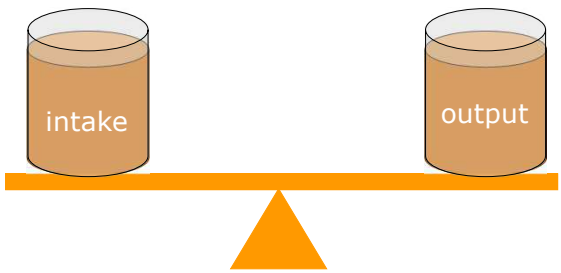
Water homeostasis

Water balance – equilibrium between intake and output of water



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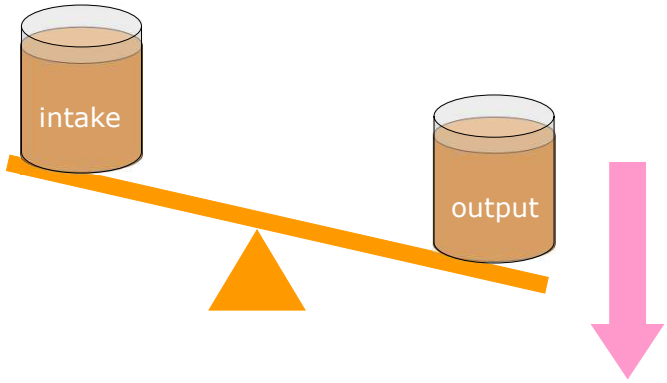
Water deficiency - ↑ thirst



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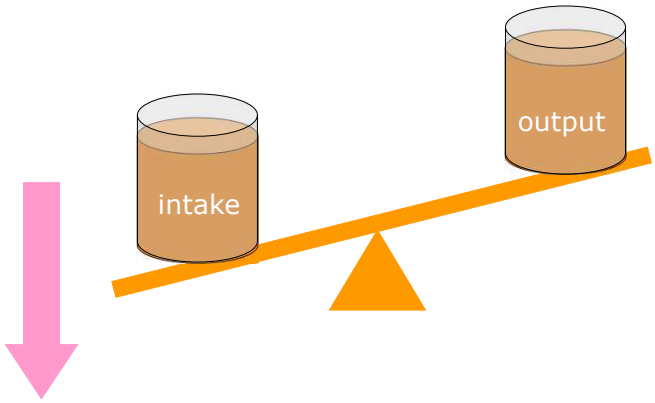
Water excess - ↑ urination



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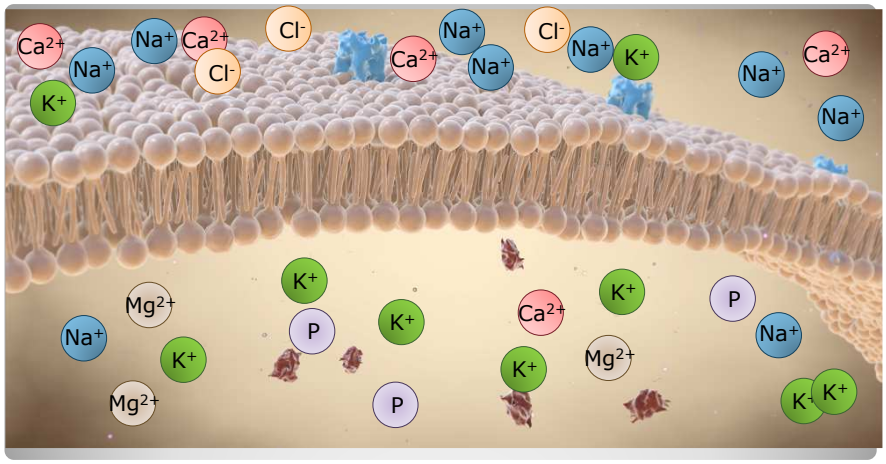

Electrolytes

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Ion	Amount in body	Plasma mmol/l	Cells mmol/l
Sodium, Na^+	92 g 4 mol	141	10
Potassium, K^+	100-140 g 2,5-3,5 mol	4	155
Calcium, Ca^{2+}	1200 g 30 mol	2,5	< 0,001 (uneven in organelles)
Magnesium, Mg^{2+}	26,5 g 1,1 mol	1	15
Chloride, Cl^-	50 g 1,4 mol	103	8
Phosphate (as phosphorus)	775 g 25 mol	1	65

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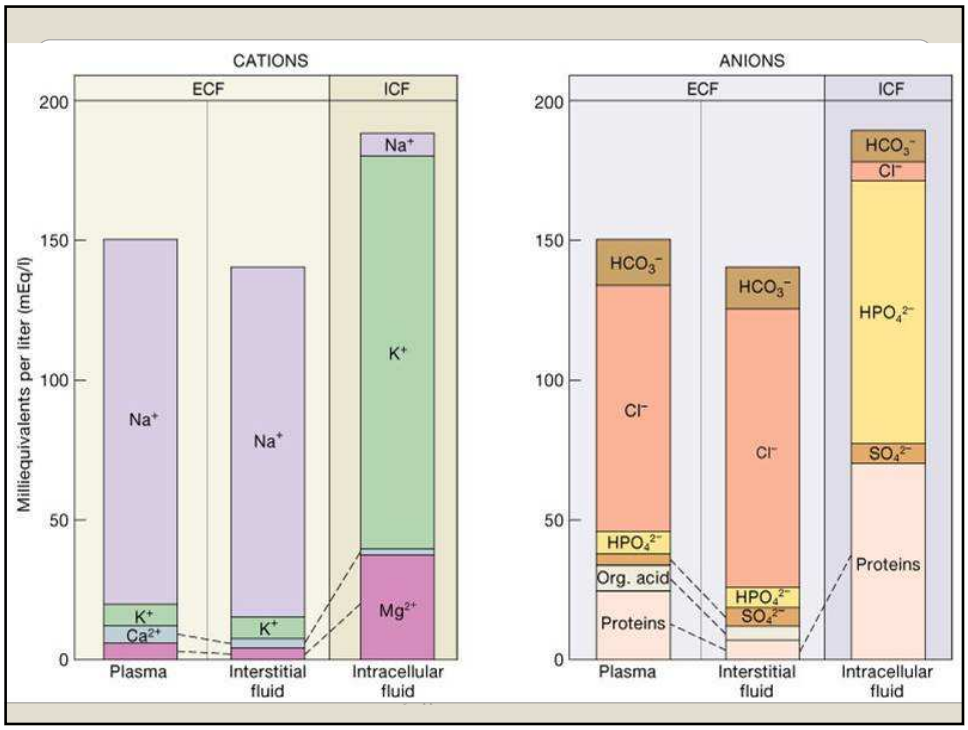

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Osmolality of plasma

Osmolality - mmol/kg of solvent
Osmolarity - mmol/l of solvent

Osmolarity of plasma = 2*[Na] + [glucose] + [urea]

(kations 140 mmol/l + anions 140 mmol/l + glucose 5 mmol/l + urea 5 mmol/l)

cca 290 ± 5 mmol/l

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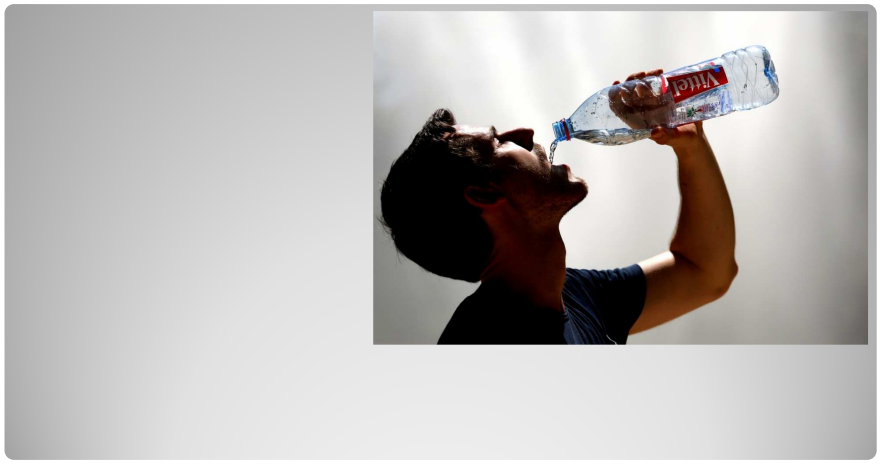

Regulation of water and sodium homeostasis

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Antidiuretic hormone

↓ plasma volume

↓

Baroreceptors

Heart atria, aa. pulomonales, carotic sinus, arcus aortae

↑ Osmolality
dry mouth

↓

Hypothalamic osmoreceptors

↘ ↙

↑ antidiuretic hormone
(arginine-vasopressin)

↓

↑ water reabsorption
(aquaporins) and ↓ urination

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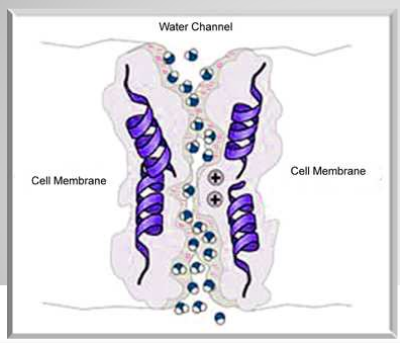
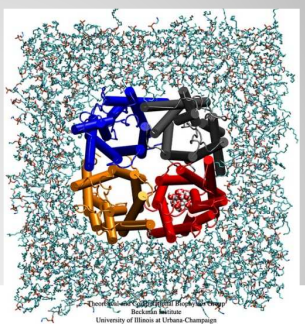
Aquaporins

- Water chanells
- Conduct water through cell membrane

- **2003 – Nobel price for chemistry**





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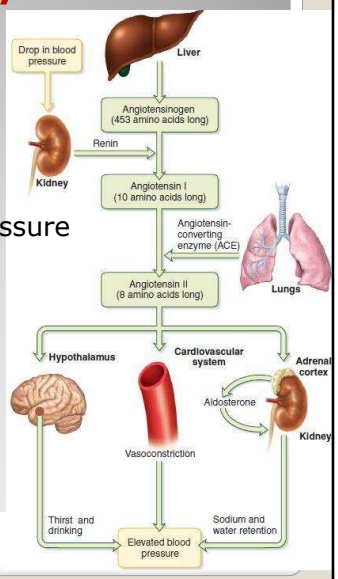
Renin-angiotensin-aldosteron system

- Angiotenzin II
  - Vasonconstriction – ↑ blood pressure
  - Antiinflamatory effect

- Aldosteron
  - Reabsorption of sodium
  - Secretion of potassium



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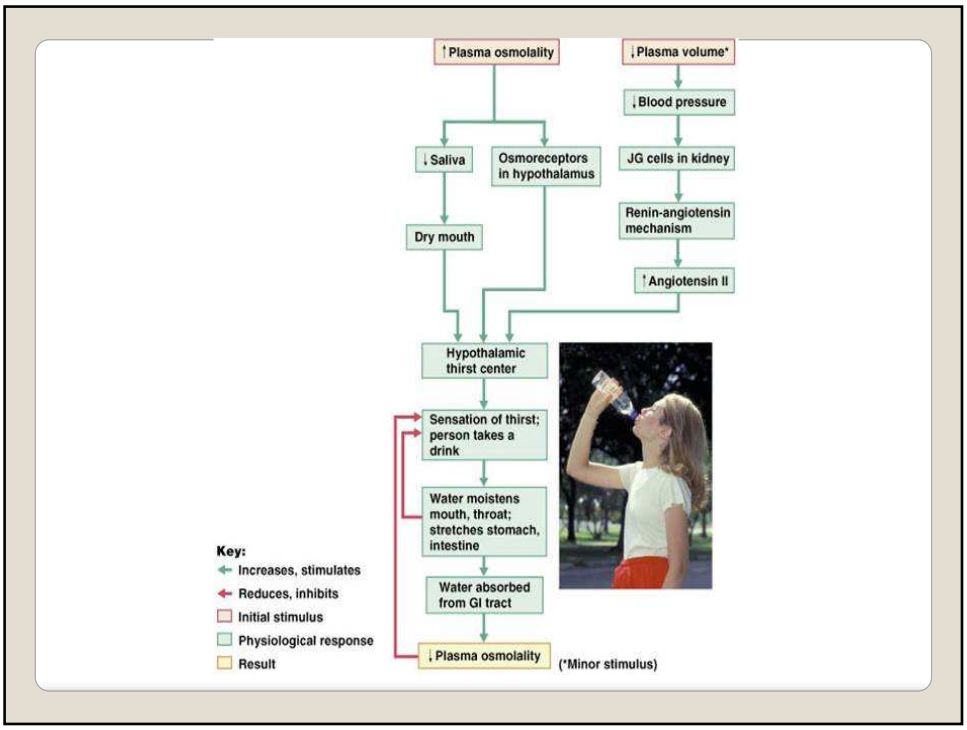

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Natriuretic peptides

- peptides which induce natriuresis

Types:
- Atrial natriuretic peptide (ANP) – produced in atria
- Brain natriuretic peptide (BNP) - ventricles in humans, brains in pigs
- C-type natriuretic peptide (CNP)
- Dendroaspis natriuretic peptide
- Urodilatin - kidneys

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Disorders of water and sodium homeostasis

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DISTURBANCES OF THE SYSTEM

- No pure forms – loss of water, salt...
- Immediate reaction of compensatory systems
- ECS is in contact both with external environment and with ICS
- ICS is in contact only with ECS
- Plasmatic concentrations are not amounts and does not inform on dynamics of compounds

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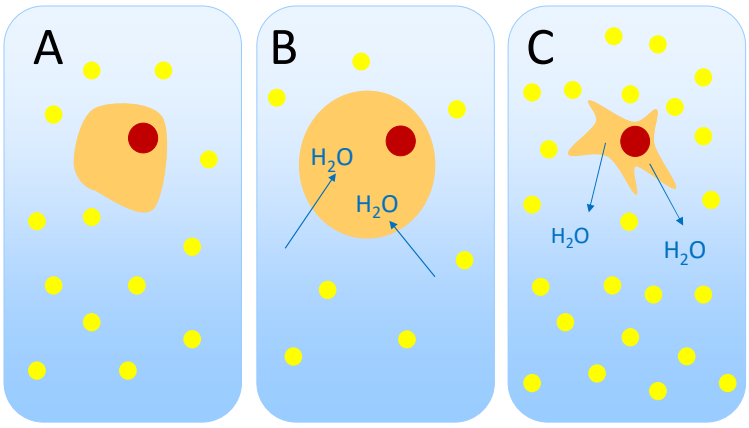

A. Isotonic fluid – No change of ICS, normal plasma sodium,
B. Hypotonic fluid – Hyponatremia compensated through water shift from ECS into ICS, reduction of ECS, swelling (oedema) of cells
C. Hypertonic fluid – Hypernatremia compensated through water shift from ICS, shrinkage of cells

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POSSIBLE CAUSES AND MECHANISMS

- Extreme deviations of external environment
  - *Dehydratation from insufficient water intake*
- Disturbances caused by damaged function of effector systems (kidneys, GIT, etc.)
  - *Diarrhoea, vomitus, kidney diseases*
- Disturbances caused by erroneous regulation (CNS, ADH, aldosterone)
  - *Diabetes insipidus, Conn sy., SIADH*
  - *Heart failure & RAA activation*

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WATER DEFICIENCY

Causes

- Insufficient fluid intake
- Inability to drink *(loss of consciousness)*
- Losses through GIT *(diarrhoe, vomitus)*
- Losses through kidneys
  - *(diuretics, osmotic diuresis, kideny diseases,*
  - *m. Addison)*
- Losses through skin *(increased sweating, burns)*
- Displacement into third place *(ileus, ascites)*
- Blood loss (?)

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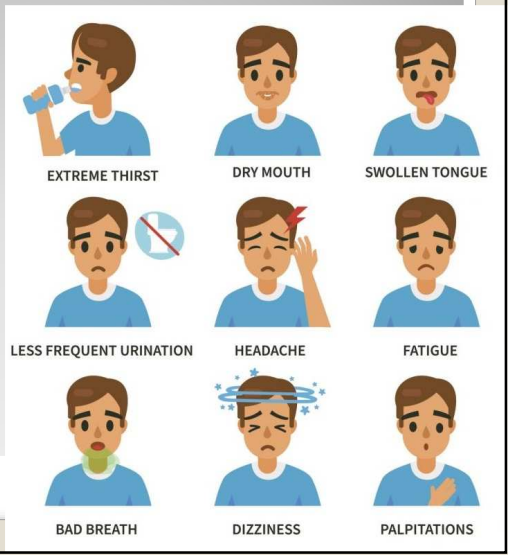
WATER DEFICIENCY
Signs

- hypotension,
- tachycardia,
- dry skin,
- thirst,
- oliguria & decreased sodium excretion,
- increase of hematocrit
- xerostomia
- dysphagia – problem with swallowing
- dysfonia – loss of voice
- dysgeusia – loss of taste



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WATER RETENTION

Causes

~~Increased fluid intake~~

- Increased intake & disturbed regulation – SIADH
- kidney failure
- nephrotic sy.
- heart failure
- liver cirrhosis

Symptoms: Oedema

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Hyponatremia
Na <135 mmol/l

Causes

- Loss of sodium
  - Addison disease
  - Diuretics
  - GIT diseases – vomiting, diarrhea
- Decresed intake of sodium (rare)
  - Combination of low sodium diet and treatment by diuretics
- Dilute hyponatremia
  - Driking too much water – water intoxication
  - SIADH – antidiuretic hormone hyperpoduction
  - Kidney failure
  - Heart failure
  - Liver cirhosis
  - Shift of water from ICF to ECF (hyperlycemia, hyperlipidemia, hyperproteinemia)

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Hyponatremia

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Hyponatremia

Clinical signs

- nausea and vomiting,
- headache,
- confusion,
- fatigue,
- irritability,
- muscle weakness,
- spasms, cramps, seizures,
- edema
- hypotension
- unconsciousness, coma

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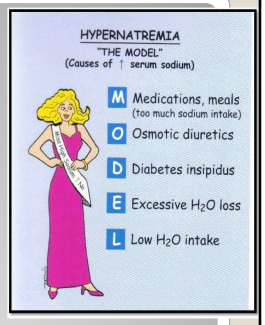
Hypernatraemia >145 mmol/l



Causes

- Increased sodium intake
  - per os
  - parenteral
- Decreased eliminatin of sodium
  - Renal insufficiency
  - Endocrine diseases – hyperaldosteronism (Conn syndrome), Cushing syndrome
- Loss of water
  - Diabetes insipidus
  - Decreased water intake - unconsciousness , brain injury or tumor
  - Loss of water from GIT (diarrhea)
  - Sweating – fever

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Hypernatraemia >145 mmol/l

Clnical signs

- Thirst
- Confusion
- Hyperreflexia, muscle spasms
- Hypotension
- Tachycardia
- Coma

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Disorders of chloride homeostasis

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Hypochloremia
< 97 mmol/l

The chloride concentration is directly depenedent on the sodium concentration and inversly dependent on the bicarbonate concentration.

Causes
- Hyponatremia (↓ Na^+ → ↓ Cl^-)
- Metabolic alkalosis (↑ HCO_3^- → ↓ Cl^-)
- Cystic fibrosis

Clinical signs
- No clinical signs
- Signs of hyponatremia
- Metabolic alkalosis
  (↓ Cl^- → ↑ HCO_3^-)

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Hyperchloremia >109 mmol/l

Causes
- Dehydration
- Diabetes insipidus
- Diuretics
- Hypernatremia (↑ Na^+ → ↑ Cl^-)
- Metabolic acidosis (↓ HCO_3^- → ↑ Cl^-)

Clinical signs
- No clinical signs
- Dehydration
- Metabolic acidosis (↑ Cl^- → ↓ HCO_3^-)

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Disorders of potassium homeostasis

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POTASSIUM HOMEOSTASIS

- Serum concentration: 3,8 – 5,5 mmol/l
- Total amount depends on muscle mass
  (young > old; man > women)
  37 – 52 mmol/kg body mass
- Intake: 2-6 g/d = 50-150 mmol/d
- Excretion through kidneys 10 – 20 mmol/d
  (0,4 – 0,8 g/d).
- Inverse association with Na excretion
- GIT excretion is important in kidney failure and in pathological conditions (diarrhoea)

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FUNCTIONS OF POTASSIUM & INTERPRETATION OF RESULTS

Functions

- intracellular osmotic pressure
- resting & action potential
- enzyme activity, proteosynthesis

Problems:

- assesment of cell homeostasis from extracellular concentration
- pH changes: exchange H/K between ECF/ICF

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INTERNAL & EXTERNAL BALANCE

Internal = K^+ shift between ICF and ECF

- acidosis: H^+ enters the cells, K^+ released into ECF
- alkalosis: H^+ released into ECF, K^+ enters the cells
- K^+ entry into cells: insulin (together with glucose), aldosterone, adrenaline
- K^+ entry into cells: rapid cellular proliferatiom (treatment of pernicious anaemia with B_{12} vitamin
- K^+ released into ECF: cell necrosis (crush sy, malignancies), hemolysis

External = K^+ shift between ECF and environment

- kidney or GIT retention/losses, parenteral intake
- dietary deficiency/excess as an additional factor

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HYPOKALAEMIA
< 3.5 mmol/l

Causes

Disorders of external balance

- GIT – diarrhoea, vomitus, tumors of colon, rectum, pancreas
- Kidneys - diuretics, polyuric stage of renal failure, hereditary tubulopathies,
- Primary & secondary hyperaldosteronism, abuse of liquorice, Cushing, ectopic ACTH production

Disorders of internal balance

- Treatment of diabetic hyperglycaemia with insulin (K^+ entry into cells together with glucose)
- Alkalosis
- Rapid cellular proliferation
- Familiar hypokalaemic periodic paralysis (hereditary)

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HYPOKALAEMIA

Symptoms

- Membrane hyperpolarisation
- Weakness, constipation, ileus, hypotonia
- Depression, confusion
- Arrhytmia, potentiation of digitalis toxicity
- ADH resistance, polyuria, polydipsia
- ECG flat/inversed T, prolonged PR,
  ST depression, prominent U

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HYPERKALAEMIA >5.5 mmol/l

Causes

Disorders of external balance

- Decreased excretion
- Increased intake (infusions, NaCl substitution) only in the case of impaired kidney function
- m. Addison, adrenogenital sy., inhibitors of angiotensin converting enzyme

Disorders of internal balance

- Acidosis
- Cell necrosis - rhabodmyolysis, burns, cytostatic treatment of malignanacies
- Digitalis overdosis
- Hyperkalaemic periodic paralysis (hereditary)
- Malignant hypertermia (hereditary)

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HYPERKALAEMIA - SYMPTOMS

Symptoms

- Low resting potential, short cardiac action potential, increased speed of repolarization ➔
- Can kill without warning
- Ventricular fibrillation and cardiac arrest may be the first signs! (if you do not check K & ECG)
- ECG: abnormal/absent P; broad QRS, peaked T, ST depression

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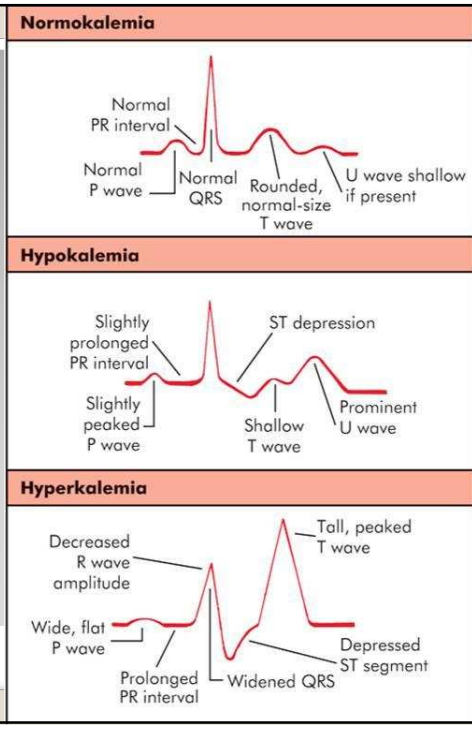

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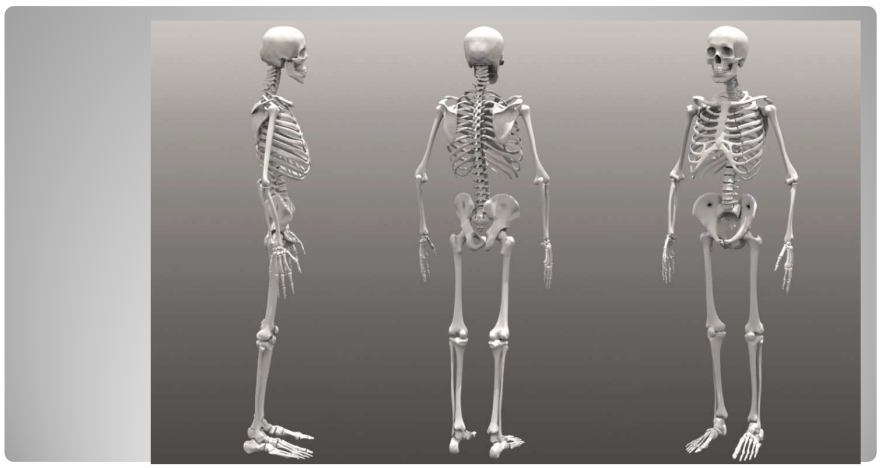

Disorders of calcium homeostasis

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CALCIUM

• Total body	1200 g	30 mol
• ECF	0,9 g	22,5 mmol
• Plasma	0,36 g	9,0 mmol
• Bone / ECF exchange		500 mmol/d
• Daily losses		25 mmol/d (1g)
◦ urine 6 mmol		
◦ faeces 19 mmol		
◦ skin 0,3 mmol		

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FUNCTIONS OF CALCIUM

- Structural
- Neuromuscular

- Bone, teeth
- Control of excitability; Neurotransmitter release Muscle contraction

- Blood
- Signal systems

- Coagulation
- Second messenger

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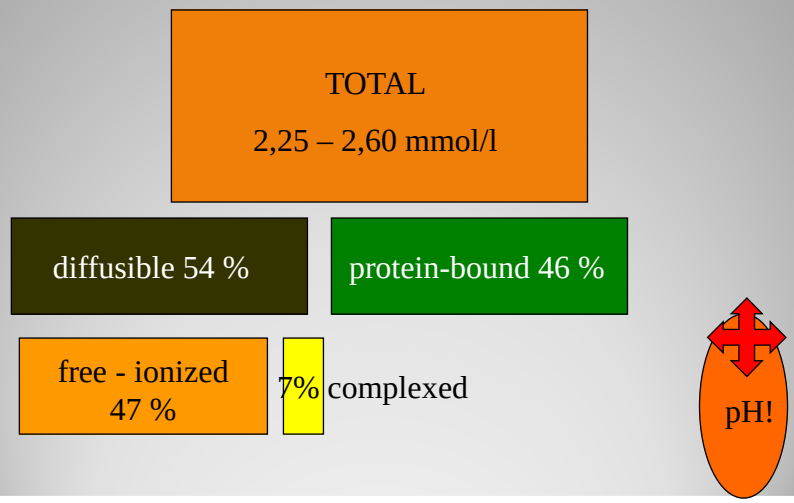
PLASMA CALCIUM



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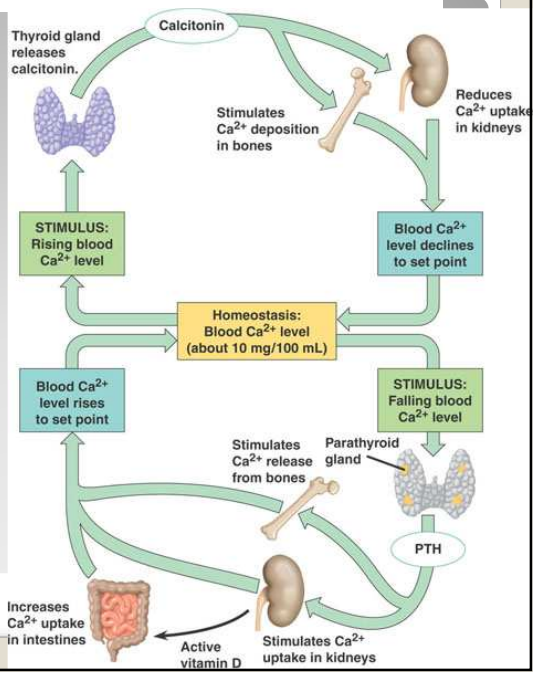
CALCIUM REGULATING HORMONES

- Parathormon
- Calcitriol
- Calcitonin



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FUNCTIONS OF PARATHORMON

BONES
- Release of calcium ↑ $[Ca^{2+}]$
- Osteoclastic resorption

KIDNEY
- Calcium reabsorbtion ↑ $[Ca^{2+}]$
- 2nd hydroxylation of vit.D ↑ Ca, P absorbtion
- Phosphaturia ↓ $[PO_4]$
- Decrease of HCO_3^- reabsorbtion ↓ pH

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HYPOCALCAEMIA
< 2.2 mmol/l

Causes

- Hypoparathyroidism
  - Congenital (with Di George sy.)
  - Acquired – autoimmune, surgery, hemochromatosis, tumors
- Pseudohypoparathyroidism
- Magnesium deficiency
- Deficiency of vitamin D
- Disorders of vitamin D metabolism – end stage renal disease
- Acute pancreatitis, transfusions with citrate, neonatal

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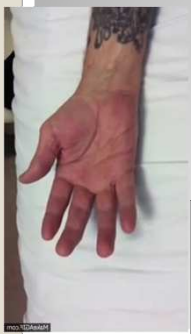
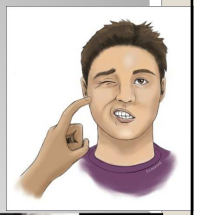
HYPOCALCAEMIA

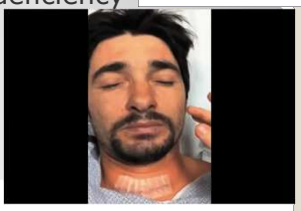
Symptoms

- Stupor, numbness, paraesthesia
- Muscle cramps and spasms „tetany"
- Laryngeal stridor
- Convulsions
- Positive Chvostek and Trousseau signs
- Long QT on ECG
- Cataract in chronic hypocalcaemia
- Rickets (rachitis) in vitamin D deficiency



Trousseau sign





Chvostek sign

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HYPERCALCAEMIA
> 2.7 mmol/l

Causes

- COMMON (90% of all)
  - Primary hyperparathyroidism
  - Malignancies – bone metastasis
- LESS COMMON
  - Thyreotoxicosis, sarcoidosis
- UNCOMMON
  - Lithium treatment, tbc, immobilisation, adrenal failure, renal failure, hereditary

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HYPERCALCAEMIA

Clinical signs

- Weakness, tiredness, weight loss
- Imparied concentration, drowsiness (coma)
- Anorexia, nausea, vomiting, constipation
- Polyuria, dehydration
- Renal calculi, nephrocalcinosis
- short QT, arrhytmias

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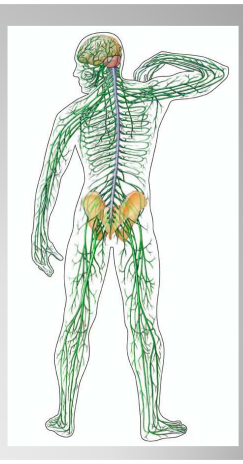

Disorders of magnesium and phosphates homeostasis

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MAGNESIUM

- 60 % in bones, higher in ICF than in ECF
- Only 0,3 % in blood, 30% protein bound
- Serum 0,7 – 1,0 mmol/l
- Regulator is not known! *adrenal medulla, insulin, parathormon ???*
- Regulated resorption from GIT ?
- Excretion through urine and stool

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MAGNESIUM

- Neuromuscular excitability (inhibition – mediated through decreased secretion of acetylcholine?)
- Bone structure
- Enzyme activity, energy production, transport mechanisms, ribosomes
- Regulation of haemocoagulation and membrane function
- Cardioprotective antiischemic, antihypoxic effects
- Sedative effect on NS
- Antihypertensive
- Antithrombotic

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Hypomagnesiemia < 0.7 mmol/l

Causes

- Deficiency associated with soil and plant deficit ⇒ grass tetany of cattle
- Some drugs and stress can increase excretion
- Unhealthy diet (alcohol)
- High doses of calcium

Signs

- Spasms
- Tiredness, irritability, tremor
- Dysmenorea, preeklampsia
- Arrythmias

Hypermagnesiemia > 1 mmol/l

- Rare

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PHOSPHATE

- 85 % in bones
- in ICF and in ECF
- In plasma – phospholipids, phosphate esters and ionized phosphate
- Regulation – PTH, vit. D and calcitonin (together but opposite with calcium)

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Hypophosphatemia
< 0.8 mmol/l

Causes

- Intestinal malabsorption
  - Vit. D deficiency
  - Use of Mg- and Al-containing acids that bind phosphates
  - Alcohol abuse
  - Malabsorption abuse
- Increased renal secretion
  - Hyperparathyroidism

Signs and symptoms

- Only in higher deficit
- Disturbed energy metabolism – nerves and musles dysfunction
- Erythrocyte, leukocyte and platelets dysfunction
- ↑ risk of infection
- Hemorhage

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Hyperphosphatemia
> 1.6 mmo/l

Causes

- Destruction of cells – tumors or anticancer therapy
- Long term using of phosphate-containing drugs (laxatives)
- Hypoparathyroidism

Signs and symptoms

- Symptoms of hypocalcemia – tetany
- Calcification of tissues

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Acid-Base Disorders

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Acid–base balance - homeostatic regulation of the pH.
Balance between acids and bases (pH) - is crucial
for the normal metabolism of the body

- H^+ concentration (activity) in arterial blood
  - 40 ± 4 nmol/l (or $4*10^{-8}$ mol/l, or 0,00000004 mol/l)
- Sörensen (1909)
  - pH = -log $[H^+]$ (pH – negative logarithm of $[H^+]$)

$[H^+] = 40$ nmol/l = 40.10^{-9} mol/l = 4.10^{-8} mol/l
pH = - log $[H^+]$
pH = - log 4.10^{-8}
pH = - (log 4 + log 10^{-8})
pH = - (0,6 + [-8])
pH = - (-7,4)
pH = 7,4

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pH in some cells and body fluids

	pH	aH^+ nmol/l
arterial blood	7,36 - 7,44	44 - 36
urine	5 – 6 (4,50 - 8,00)	32000 - 10
erythrocytes	7,28	53
muscle cells	6,90	126
bile	6,2 - 8,5	631 - 3
gastric juice	1,2 - 3,0	1000 - 63

Daily production of acids

- Daily H_2CO_3 production: 20 mol (300 – 360 l)
  - complete oxidation of fats, carbohydrates and proteins
- Non-volatile (fixed) acids: 60 – 70 milimol/day
  - oxidation of –SH groups (amino acids) - H_2SO_4
  - hydrolysis of phosphoproteins, phospholipids... – H_3PO_4
  - keto-acids
  - lactic acid

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Buffering and regulatory systems

- Buffer systems
  - bicarbonate system – HCO_3^-/H_2CO_3 (major buffer of ECF)
    - Henderson-Hasselbalch equation
      - $pH = pK + \log [HCO_3^-]/[H_2CO_3]$
    - simplyfied
      - $pH \sim [HCO_3^-]/pCO_2$
  - phosphate - $HPO_4^{2-}/H_2PO_4^-$ (in kidneys, ICF)
  - protein (in ICF, ECF)
  - haemoglobin (in RBC)
- Regulatory systems
  - respiratory
  - kidneys
  - liver

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- Role of the lungs in acid-base homeostasis
  - CO_2 excretion
  - Hyperventilation - $\downarrow pCO_2$
  - Hypoventilation - $\uparrow pCO_2$
- Role of the kidney in acid-base homeostasis
  - excretion of H^+ ions
    - phosphate buffer
    - ammonia
  - excretion of fixed acids
  - reabsorption of filtered HCO_3^-
- Other
  - Liver
    - Albumin synthesis, ammonia metabolism, formation of ketone bodies, lactate...
  - Exchange of ions between intracellular and extracellular space
    - Exchange of H^+ for K^+ or Na^+ ions
  - Bones
    - Exchange of H^+ ions for other cations (Ca^{2+}, K^+, Na^+)

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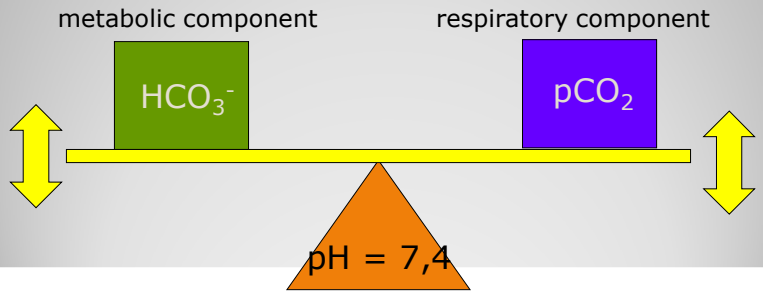
Classification of acid-base disorders

metabolic component respiratory component

$pH \sim [HCO_3^-]/pCO_2$



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Classification of acid-base disorders

Simple acid-base disorders

- Acidosis pH < 7,36
  - Metabolic HCO_3^- < 22 mmo/l
    - •acute
    - •chronic
  - Respiratory CO_2 > 5.8 kPa
- Alkalosis pH > 7,44
  - Metabolic HCO_3^- > 26 mmo/l
    - •uncompensated
    - •partly compensated
    - •compensated
  - Respiratory CO_2 < 4.8 kPa

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Metabolic acidosis (MAC)

1. Increased production of H^+
- infusion of NH_4Cl
- toxins (salicylate, ethanol – acetate, methanol)
- ketoacidosis - diabetes mellitus, starvation
- lactic acidosis – in hypoxia

} ↑ anion gap

2. Decreased renal excretion of H^+
- renal failure
- renal tubular acidosis (RTA) type I – distal
  (disorder of H^+ excretion)

3. Loss of HCO_3^-
- acute diarhoea
- RTA type II – proximal
  (disorder of HCO_3^- reabsorption)
- diuretics – acetazolamide, tiazide
  (inhibitors of carbonic anhydrase)

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Metabolic alkalosis (MAL)

1. Deficiency of Cl^-
Missing chloride in plasma is replaced by bicarbonate → MAL
- vomiting
- chloride diarhoea

2. Increased exogenous bicarbonte
- oral/intravenous bicarbonate
- antacid therapy
- organic acid salts – lactate, acetate, citrate

3. K^+ depletion
Exchange of H^+ and K^+ between plasma and intracellular space
- Bartter syndrome
- hyperaldosteronism

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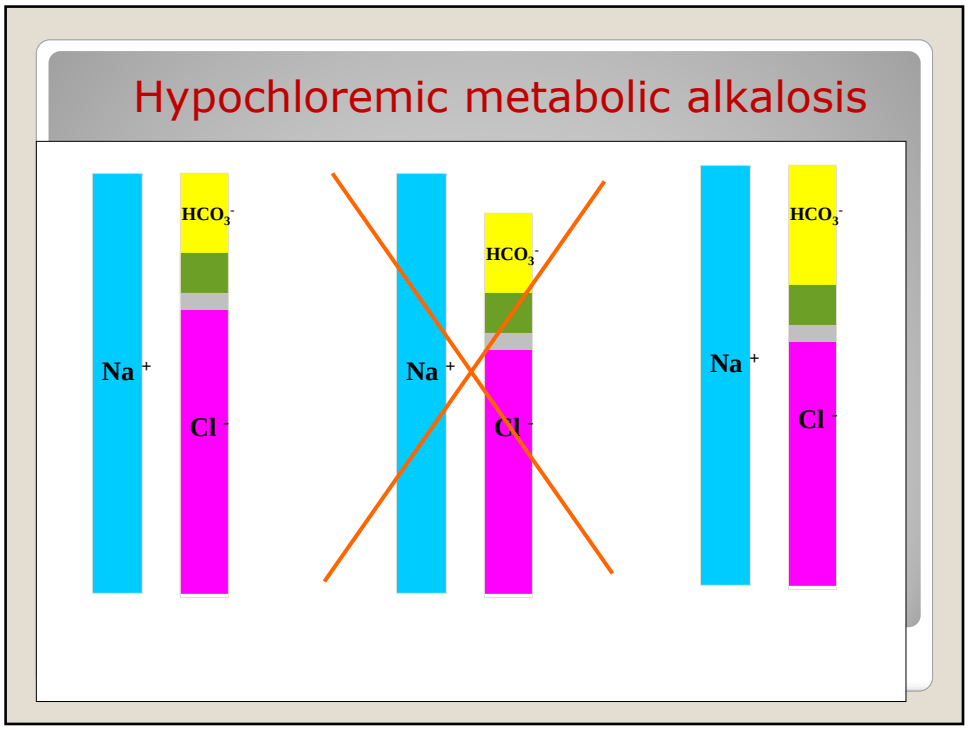
Hypochloremic metabolic alkalosis
Na +
HCO3-
Cl -
Na +
HCO3-
Cl -
Na +
HCO3-
Cl -

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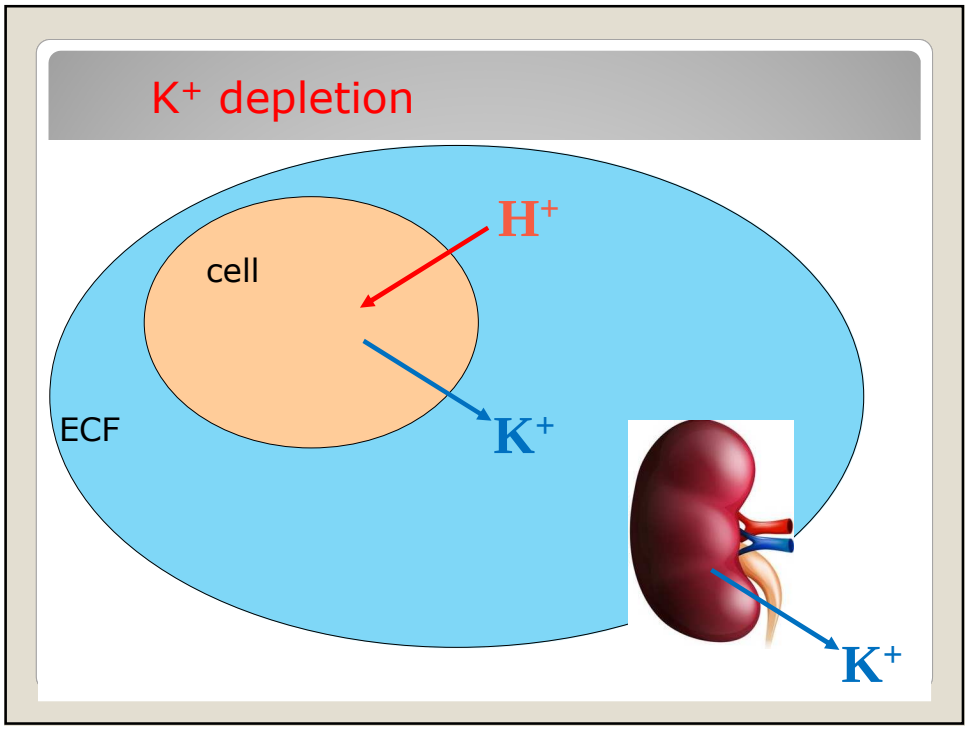
K+ depletion
H+
cell
ECF
K+
K+

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Respiratory acidosis (RAC)

1. Central depresion

- trauma, cerebrovacular accidents, CNS infection, tumors, ischaemia, Pickwick sy.
- drugs – sedative, narcotics

2. Ventilation disorders

A. Thoracic diseases
- trauma
- kyfoscoliosis

B. Neuromuscular diseases
- myopathies, multiple sclerosis, poliomyelitis

C. Lung diseases
- obstructive disease
- empysema
- pneumonia...

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Respiratory alkalosis (RAL)

1. Central stimulation

- anxiety/hysteria
- pregnancy (stimulation by progesterone)
- gram-negative septicaemia
- hepatic encephalophaty
- salicylate overdose
- infection, trauma
- tumour

2. Pulmonary pathology

- embolism
- congestive heart failure (lung oedema)
- asthma, pneumonia

} *only mild forms*

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Clinical signs

Metabolic acidosis
hyperventilation (Kussmaul breathing)

Metabolic alkalosis
$\downarrow Ca^{2+}$ (binding on proteins) $\rightarrow \uparrow$neuromuscular activity
tetanic cramps, dysrhytmias

Respiratory acidosis
cerebral vasodilatation – headache, stupor, coma

Respiratory alkalosis
cerebral vasoconstriction – headache
$\downarrow Ca^{2+} \rightarrow$ tetany

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Mixed acid-base disturbances

MAC and MAC
- Diabetes mellitus
  lactate acidosis and ketoacidosis

MAC and RAC
- Chronic obstructive pulmonary disease (COPD)
  RAC (hypoventilation) + lactate MAC (hypoxia)

MAC and MAL
- Diarhea and vomiting
- Alcohol intoxication and vomiting

MAC and RAL
- Lung oedema in early stage
  lactate MAC + hyperventiltion (RAL)

MAL and RAL
- MAC compensation + alcalisation

~~RAC and RAL~~
NEVER

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Compensation of acid-base disorders

- Body's homeostatic response to an acid-base imbalance in attempt to bring the pH of the body fluids as near normal as possible
- if the pH change is caused by metabolic component (HCO_3^-)
  - compensation by respiratory component (lungs)
    - MAC - Hyperventilation, Kussmaul breathing
    - MAL - Hypoventilation
    - Delays 12 – 24 hours
- if the pH change is caused by respiratory component (pCO_2)
  - compensation by metabolic component (kidneys)
    - RAC - Increased kidney HCO_3^- reabsorption
    - RAL - Decreased kidney HCO_3^- reabsorption (increased HCO_3^- secretion)
    - Delays 2 – 3 days

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Compensation

metabolic acidosis

$$pH \sim \frac{HCO_3^-}{pCO_2}$$

compensation

$$pH \sim \frac{HCO_3^-}{pCO_2}$$

lungs
(hyperventilation)

respiratory acidosis

$$pH \sim \frac{HCO_3^-}{pCO_2}$$

compensation

$$pH \sim \frac{HCO_3^-}{pCO_2}$$

kidneys
(higher HCO_3^- reabsorption)

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Compensation

metabolic alkalosis

$$pH \sim \frac{HCO_3^-}{pCO_2}$$

compensation

$$pH \sim \frac{HCO_3^-}{pCO_2}$$

lungs
(hypoventilation)

respiratory alkalosis

$$pH \sim \frac{HCO_3^-}{pCO_2}$$

compensation

$$pH \sim \frac{HCO_3^-}{pCO_2}$$

kidneys
(lower HCO_3^- reabsorption)

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Compensation

$$pH \sim \frac{HCO_3^-}{pCO_2}$$

	primary change	pH change	compensation
MAC	↓ HCO_3^-	↓	↓ pCO_2
MAL	↑ HCO_3^-	↑	↑ pCO_2
RAC	↑ pCO_2	↓	↑ HCO_3^-
RAL	↓ pCO_2	↑	↓ HCO_3^-

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Correction of acid-base disorders

- Correction is bodie´s response to an acid-base imbalance in attempt to bring the pH to normal by the same component, that caused acid-base disorder
- possible only in metabolic disorders – correction by kidneys
  - Metabolic acidosis
    e.g. in diabetic ketoacidosis
    - corrected by increased kidney HCO_3^- reabsorption
  - Metabolic alkalosis
    e.g. in liver disease
    - corrected by increased kidney HCO_3^- secretion
- Correction also refers to the treatment (both metabolic and respiratory acid-base disorders) aimed at resolving the underlying cause of the acid-base disorder, thereby returning the pH and other parameters (e.g., CO_2, HCO_3^-) back to normal.

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Acid-base parameters

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pH = 7,4 ± 0,04
- pH < 7,36 acidaemia
- pH > 7,44 alkalaemia

pCO_2 =5,3 - 0,5 kPa (40 mmHg)
- pCO_2 < 4,8 kPa hypocapnia
- pCO_2 > 5,9 kPa hypercapnia

HCO_3^- = 24 ± 2 mmol/l
- HCO_3^- < 22 mmol/l hypobasemia
- HCO_3^- > 26 mmol/l hyperbasemia

BE = 0 ± 2 mmol/l
- Base excess/deficit - an excess or deficit, respectively, in the amount of base present in the blood

pO_2 = 10,0 - 13,3 kPa

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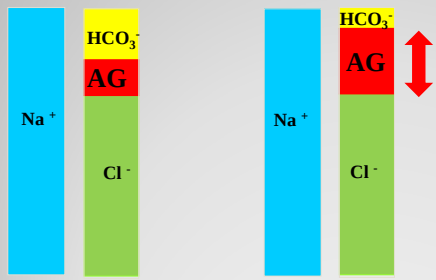
BB = 48,0 ± 2,0 mmol/l
- Buffer base – sum of all bases in 1 l of blood
  Calculation: BB = HCO_3^- + proteins or BB = $(Na^+ + K^+) - Cl^-$

AG = 15,2 ± 1,6 mmol/l
- evaluation of metabolic acidosis to determine the presence of unmeasured anions (ketone bodies, lactate...)
  Calculation: AG = $(Na^+ + K^+) - (Cl^- + HCO_3^-)$



MAC with ↑ anion gap
- Ketoacidosis
- Lactate acidosis

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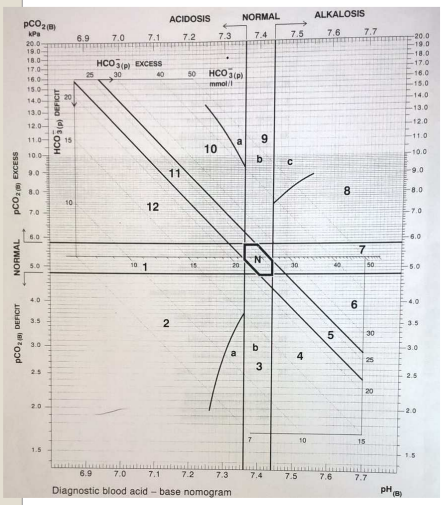
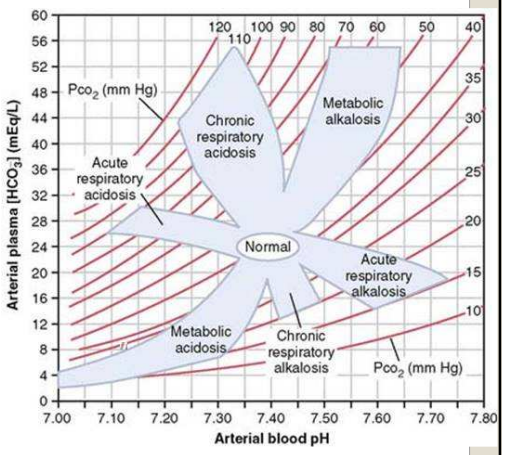
Acid-base normograms





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