Acid – base balance
Homeostasis

Homeostasis, compartments

- Blood plasma
- Interstitial fluid
- Intracellular fluid

Functions:

- transport of nutrients, oxygen, hormones, antibodies
- transport of catabolites, CO_2, hormones
- cell migration (cellular immunity)
- maintenance of pH, osmolality and ionic composition
- temperature stability

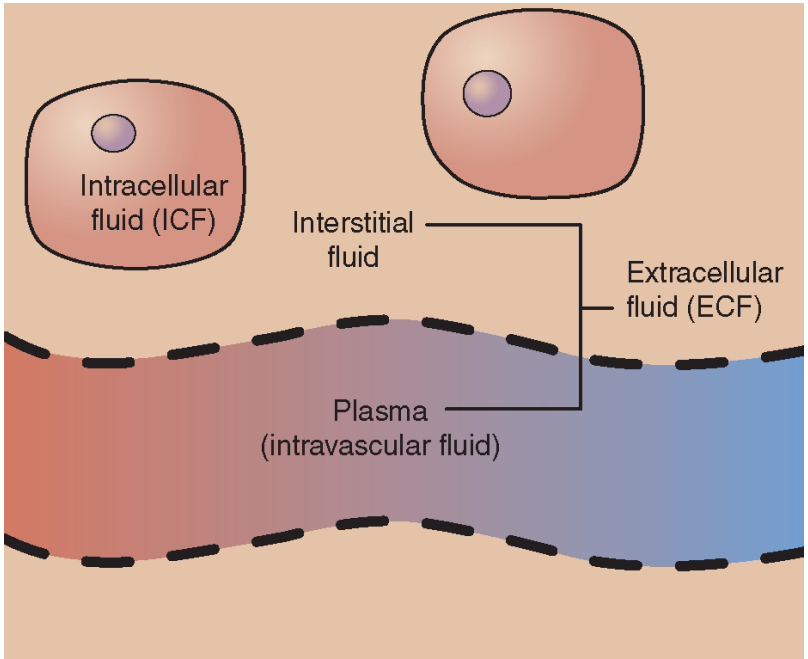
Body water

Human body contains approx. 50-80 % of water (depending on age)

- 80 % - newborns
- 60 % - adults
- 50 % - elderly people

Distribution of water in the body

- Intracellular (ICF) 40 % body weight
- Extracellular (ECF) 20 % body weight
  - Interstitial 15 %
  - Intravascular 5 %



Transcellular fluid

Physiologically

- GIT (after eating 2-3 liters)
- Cerebrospinal fluid (CSF) 120 -180 ml in adults

Pathologically

- Abdominal cavity (ascites)
- Thoracic cavity (hydrothorax)
- Intestine (ileus)
- Hematomas

Water balance

Intake (ml)		Excretion (ml)	
Drinking	1500	Urine	1500
Food	700	Perspiration	400
Oxidation of nutrients	300	Breath	400
		Sweat	100
		Faeces	100
Total	2500		2500

Can be measured

Can be estimated

Osmolality

The ratio of water to all dissolved substances, regardless of their size.

Normal ranges: 280 - 300 mmol/kg H_2O

Influenced by concentration of Na^+, urea, glucose

Calculation of plasma osmolality (approximately)

$$2[Na+] + [Glucose] + [Urea]$$

$$2 * 140 + 5 + 5 = \underline{290\ mmol\ .\ kg^{-1}}$$

Osmolal gap

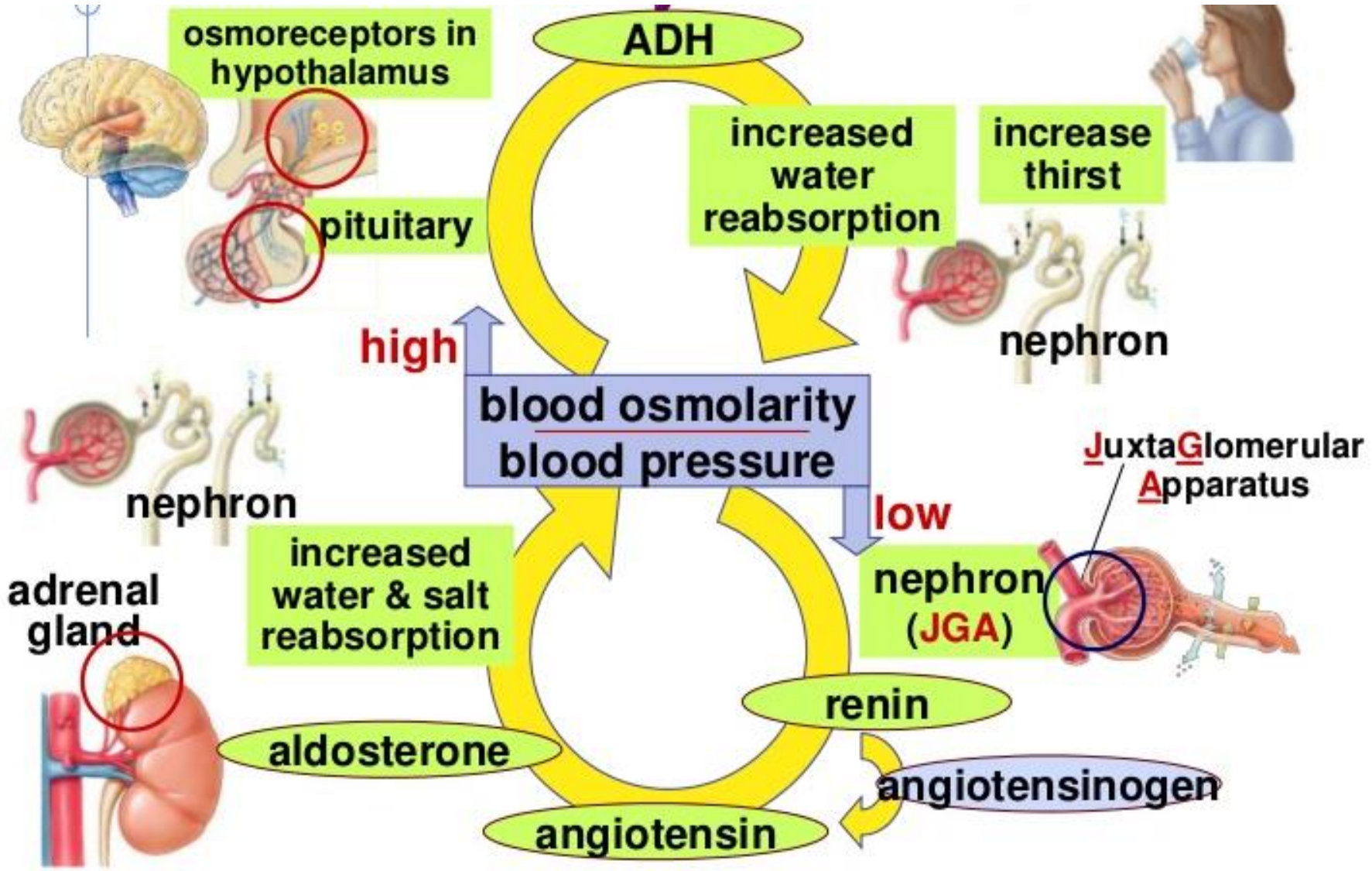
- Difference between measured and calculated osmolality

$$OsmGap = POsm_{measured} - POsm_{calculated}$$

- Detection of the presence of volatile substances (alcohol, ethylene glycol)
- If OsmGap > 10 mmol/kg, the presence of volatile substances is very likely
- 1 g of ethanol per litre of plasma (1 per mille of alcohol) increases osmolality by about 23 mmol/kg.

Regulation of osmolality

- Osmoreceptors
- Antidiuretic hormone (ADH) – regulation of clean water excretion in the kidneys


osmoreceptors in hypothalamus
ADH
increased water reabsorption
increase thirst
pituitary
nephron
high
blood osmolarity
blood pressure
low
nephron
JuxtaGlomerular Apparatus
increased water & salt reabsorption
adrenal gland
nephron (JGA)
renin
aldosterone
angiotensinogen
angiotensin

Hyperosmolality

Lack of water, many solutes

- Dehydration
- Temperatures, burns (loss of clean water), inability to drink (reduced intake of clean water)

or

- ↑ concentration of substances in the blood (glucose, urea, alcohol) but without dehydration

Reaction: ↑ ADH secretion → increase in resorption of clean water in the kidneys (a decrease in urine production that will be more concentrated) + feeling thirsty

Hypoosmolality

Too much of water and lack of solutes

- „Poisoning with the water“
- Inappropriate infusion treatment (glucose)
- Brain injury, ADH oversecretion

Reducing the concentration of substances in the blood (Na^+, albumin, proteins) → risk of water leakage into interstitium and the development of edema

Reaction: ↓ ADH secretion and increase in production of urine that will be less concentrated

Osmolality in urine

50 - 1400 mmol/kg H_2O

- in old age: max. 800 (decreased renal concentration capacity)

It depends on:

- renal concentration capability
- diuresis (water intake)

Hydration disorders

Natrium and water are regulated together. However, the organism reacts differently to the loss or excess of clean water and water with solutes.

Like a pond…

- Clean water
- Solutes (Na^+) are fish

Hydration disorders

Hydration dysbalance appears as a result of an excess or lack of:

- Clean water
- Water with solutes (water + Na^+)

Basic rules

- The resulting disorder depends on the type of missing / excess fluid
- Accordingly, the body reacts by activating the appropriate regulation system
- Natrium is osmotically active. The water follows Na^+.

Basic rules

The body regains what it has lost and gets rid of what it has excess

- Loses clean water, resorbs clean water... (ADH)
- Loses water with solutes, resorbs water with solutes... (aldosterone)

And vice versa... has an excess of water with solutes, excrets water with solutes (natriuretic peptide)

Basic rules

Hydration disorders (dehydration and hyperhydration) are divided according to what the resulting disorder is, i.e. whether it leads to:

- Isoosmolality – isotonic hyper/hypohydration
- Hypoosmolality – hypotonic hyper/hypohydration
- Hyperosmolality – hypertonic hyper/hypohydration

... not depending on which fluid is lost or dwells (isotonic, hypotonic or hypertonic).

Basic rules

The organism has 3 basic control systems that affect the metabolism of clean water or water with solutes:

- ADH - clean water resorption
- Aldosterone – resorption of Na^+ which is followed by water
- BNP (natriuretic peptide) – inhibits Na^+ resorption leading to natriuresis. Na^+ is followed by water.

Changes in the volume of clean water

Loss of clean water

→ hypertonic dehydration

Hypertonic dehydration

Loss of clean water → increase in osmolality

- Causes: insufficient water intake (elderly people), unconsciousness, polyuric phase of renal failure – loss of low concentrated urine, diabetes insipidus.
- Consequences: ↑ concentration of Na^+ in ECT and osmolality
- Reaction: clean water is missing → clean water must be resorbed (ADH system used). Activation of osmoreceptors in the hypothalamus, ↑ ADH and increase the resorption of clean water.

Excess clean water

→ hypotonic hyperhydration

Hypotonic hyperhydration

Excess clean water → decrease of osmolality

- Causes: inability to excrete clean water (cardiac patients, oliguria / anuria). Rarely "water poisoning", SIADH (syndrome of inadequate ADH secretion), tumors, brain damage. Excessive water resorption occurs, urine is hyperosmolal.
- Consequences: hypoNa and hypoosmolality,
- Reaction: ↓ ADH, production of unconcentrated urine
- Treatment: restrictions on water intake.

Changes in water volume with solutes

Loss of isoosmolar fluid

→ isotonic dehydration

Isotonic dehydration

Loss of isotonic fluid (water + Na^+)

- Causes: vomiting, bleeding, burns, shock
- Consequences: osmolality does not change, haemoconcentration is present, rise of haemoglobin and protein concentrations.
- Reaction: osmoreceptors do not react. Organism reacts when BP decreases and renal perfusion is reduced. Activation of the juxtaglomerular apparatus of the kidneys and secretion of renin (centralization of circulation).
- Renin → angiotensinogen → angiotensin I, which, using ACE is converted into angiotensin II → angiotensin III (peripheral vasoconstration and aldosterone production) – ↑resorption of Na^+ which is followed by the water

Excess isoosmolar fluid

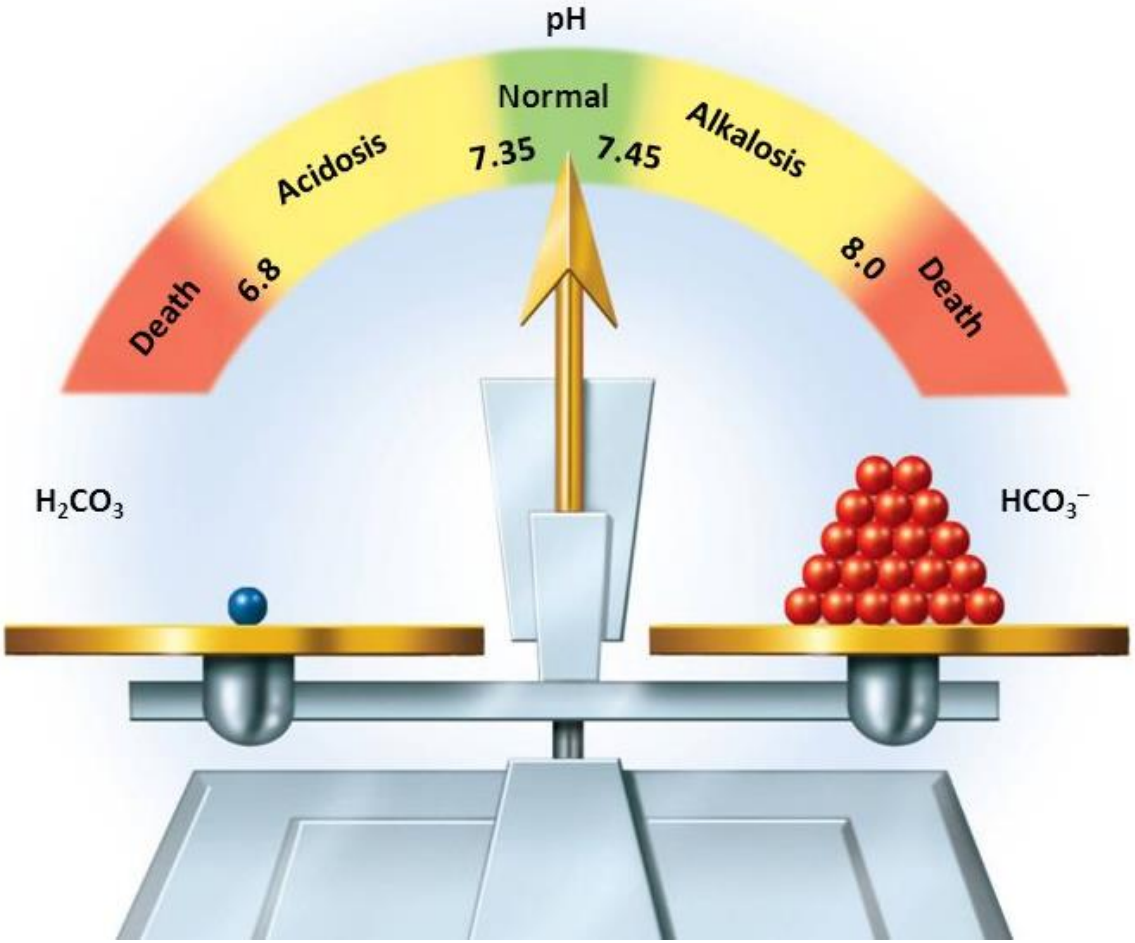
→ isotonic hyperhydration

Isotonic hyperhydration

Excess isotonic fluid (water + Na^+)

- Causes: cardiac failure, hypoproteinemia (nephrotic syndrome).
- Consequences: osmolality does not change (↑ water and Na^+), ECF volume is increasing. The development of oedema in hypoproteinemic patients (fluid moves to extravasal compartment) → reduced volume of circulating fluid in blood vessels activates RAAS → secondary hyperaldosteronism.
- Reaction: secretion of natriuretic peptides (BNP) from LA and LV in response to increased preload of the heart. Osmoreceptors don‘t react. Inhibition of Na^+ resorption in the distal tubule → natriuresis with the water excretion.

Acid-base balance



pH definition

Def.: pH is a negative decimal logarithm of activity (concentration) of hydrogen cations.

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

pH stability in the organism

- pH is strictly regulated (pH = 7,35 – 7,45)
- pH < 6,80 or > 7,80 is dangerous!
- pH stability is necessary to maintain the stability of the homeostasis
- Distribution of substances in the organism, ion and water balance, pH optimum of enzymes, changes in protein structure when pH changes, etc.
- pH stability is a priority needed to survive, therefore effective compensatory mechanisms are available: buffers, kidneys, lungs (+ liver activity – urea synthesis)

Maintaining physiological pH

3 systems:

- Extracellular buffers[1)]
- Lungs[2)]
- Kidneys[2)]

---

1) primary quick compensation

2) secondary slow compensation

AB dysbalance

Acidosis

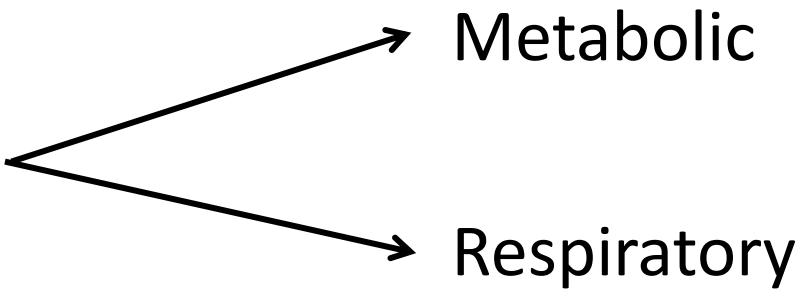
- pH < 7,35
- Severe… pH < 6,80

Alkalosis:

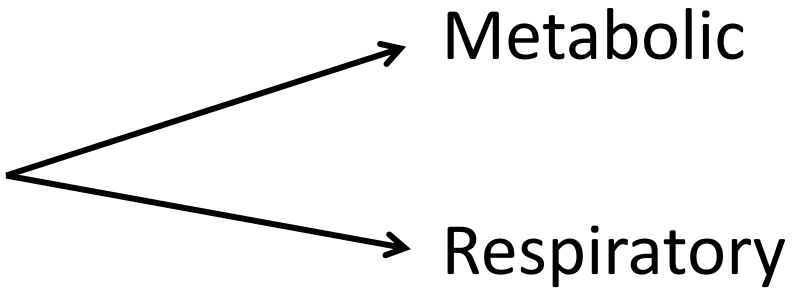
- pH > 7,45
- Severe… pH > 7,70

Basic AB disorders

- Acidosis



- Alkalosis



+ combined AB disorders

Metabolic acidosis - causes

Acid accumulation:

- Ketosubstances - starvation, diabetes
- Acid metabolites - renal failure
- Poisoning (methanol, strong acids)

Loss of bicarbonate (diarrhea) or ↑chloridemia

Lactate acidosis

- overproduction of lactate
- ↓ lactate utilization (liver failure, sepsis, biguanide poisoning)

Metabolic alkalosis - causes

Chloride loss:

- Vomiting HCl (hypoCl MAlk)
- Nasogastric tube – suction of gastric juices
- Diuretics

Excess bicarbonate

- Overdose in the treatment of acidosis

Respiratory acidosis - causes

Accumulation of carbonic acid in insufficient breathing (CO_2 accumulation)

- diseases of the lungs, diaphragm, respiratory nerves, respiratory center (drug poisoning!)

Respiratory alkalosis - causes

Lack of carbonic acid due to excessive breathing (decrease in CO_2)

- Hyperventilation syndrome (anxieta, hysteria, stress)
- Cerebral lesions (encephalitis, meningitis, tumors, trauma)
- Pulmonary embolization

Maintaining a normal pH

Limiting the influence of acids and bases using the buffers

- Reaction with acids, bases
- Maintaining physiological pH
- Binding excess H^+ ions (temporary solution)

Permanent solution = excretion of H^+ ions by the lungs and/or kidneys

Main buffers

Blood

- Sodium hydrogen carbonate: $NaHCO_3$
- Hemoglobin
- Proteins

Intracellular fluid

- Phosphates

Hydrogencarbonate buffer

$$H^+ + HCO_3^- \leftrightarrow \ H_2CO_3 \ \leftrightarrow \ CO_2 + H_2O$$

- Synthesis in the kidneys
- Blood concentration: 24 ± 2 mmol/l

Dissolution CO_2 in the blood

$$CO_2 + H_2O \leftrightarrow H_2CO_3 \leftrightarrow H^+ + HCO_3^-$$

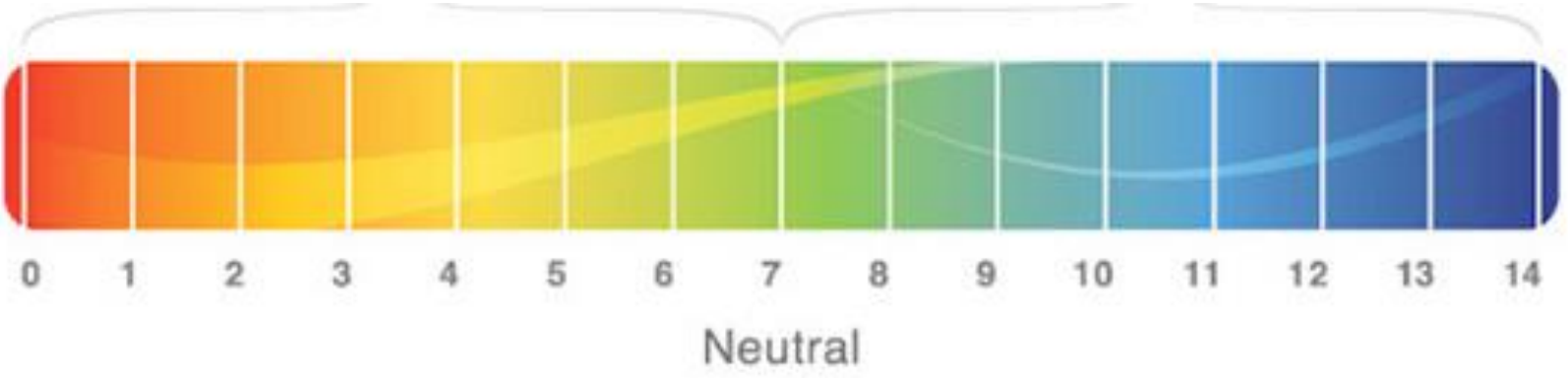
800 : 1 : 0.03

Lungs (intensity of exhalation CO_2)

Kidneys (excretion of H^+, synthesis HCO_3^-)

Hydrogencarbonate (HCO_3^-)

- Deficiency (decrease in concentration) → acidosis
- Excess (increase in concentration) → alkalosis



Compensation of AB dysbalances

The compensation of alkalosis and acidosis (the body's reaction to deflection) takes place in the opposite way to the one that triggered the pathological condition.

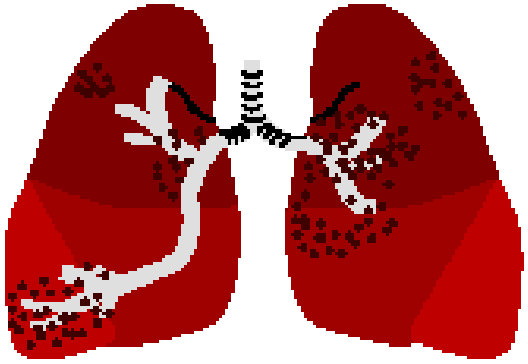
Respiratory disorders are compensated metabolically (by the kidneys) and vice versa.

Pulmonary compensation

Change in the pCO_2 → change in concentration of H_2CO_3

$$HCO_3^- + H^+ \rightleftharpoons H_2CO_3 \rightleftharpoons H_2O + CO_2$$

Excretion of CO_2 by lungs drives reaction to right.

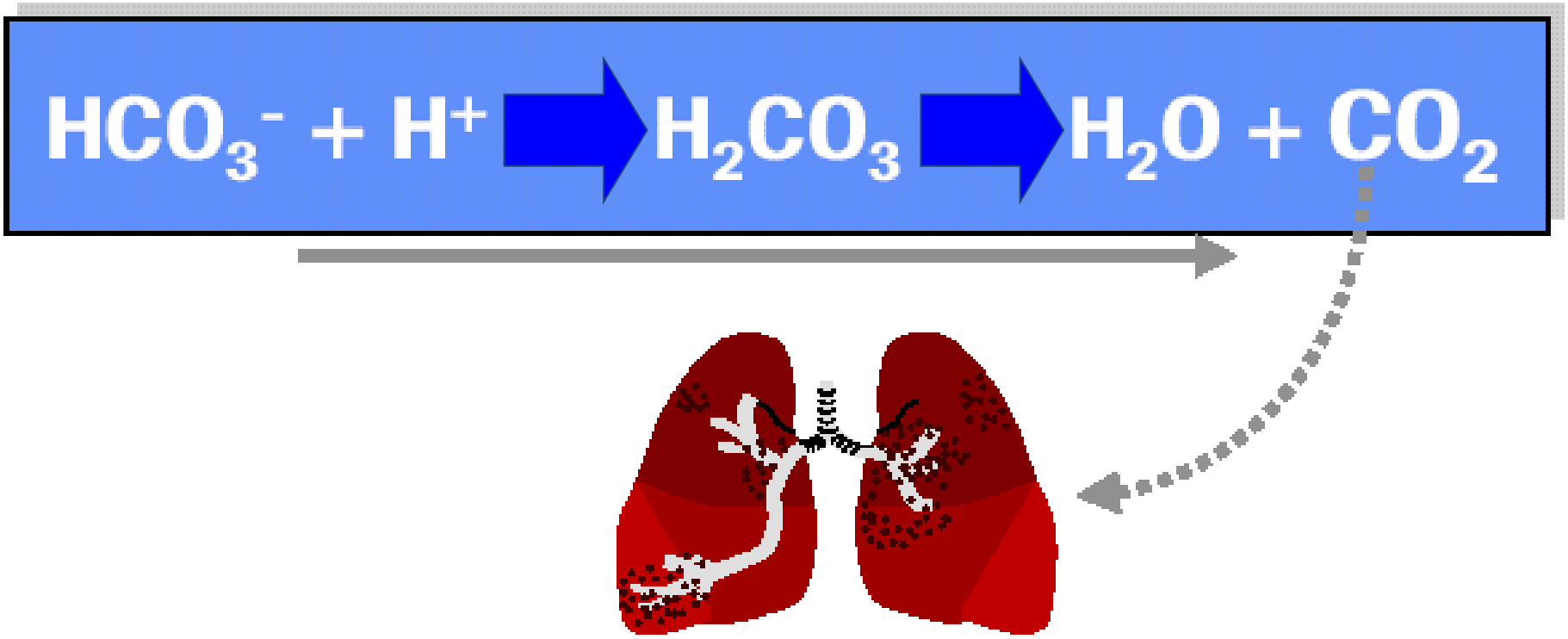


Pulmonary compensation for metabolic acidosis

Reaction: hyperventilation, Kussmaul's breathing

exhalation CO_2, ↓ H_2CO_3

- effective mechanism

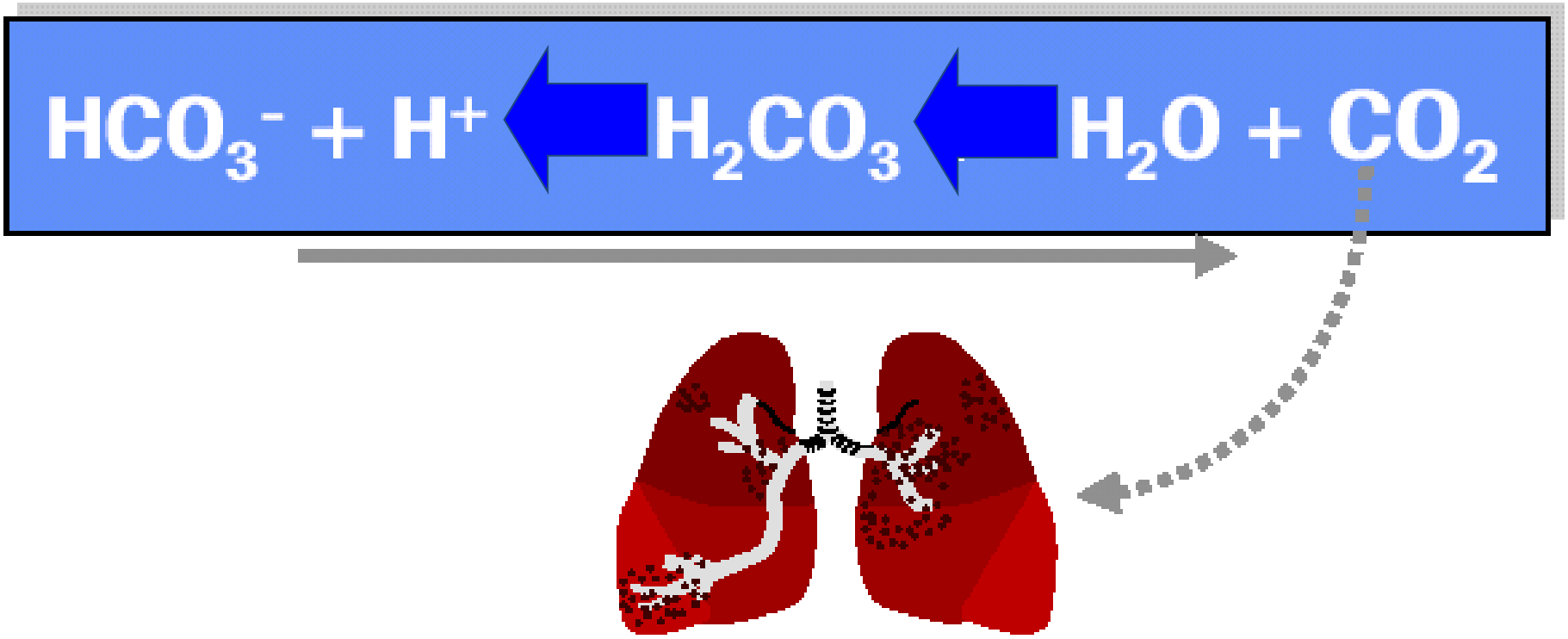


Pulmonary compensation for metabolic alkalosis

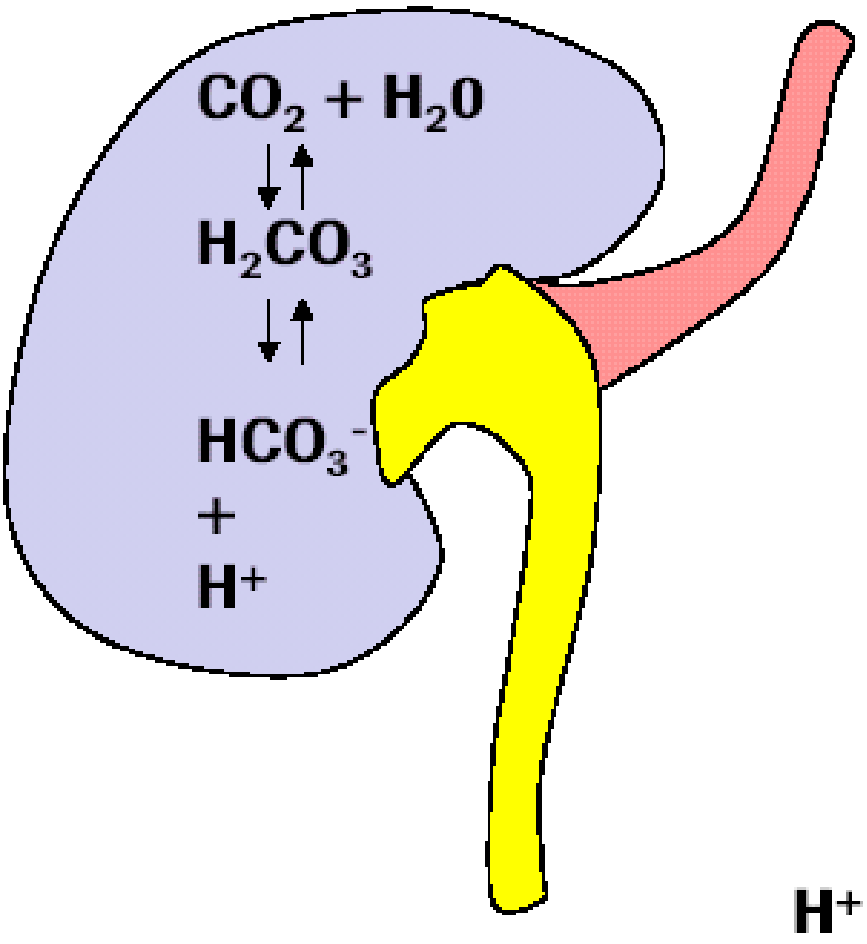
Reaction: hypoventilation

↑ pCO_2 , ↑ H_2CO_3 but ↓ pO_2, hypoxia

- low-effective mechanism



Kidneys



HCO_3^- returned to blood

Bicarbonate regeneration

Exclusion
hydrogen ions

Kidney compensation

Acidosis

- ↑ synthesisHCO_3^-
- ↑ synthesis and excretion NH_4^+, $H_2PO_4^-$

Alkalosis

- ↓ resorption HCO_3^-
- ↓ synthesis NH_4^+ (↓ exkrece H^+), ↑ synthesis HPO_4^{2-}

AB balance parameters examination

- pH Measurement [H+]
- pCO_2 Measurement of the resp. component
- HCO_3^- Measurement of metabolic component

AB balance parameters

Anion gap (AG)

- difference between the main cations and plasma anions $(Na^+ + K^+) – (Cl^- + HCO_3^-)$
- is used to assess the proportion of lactate, ketosubstancces, oxalate in the AB disorder.

Strong ion difference (SD)

- $(Na^+ + K^+ + Ca^+ + Mg^+) - Cl^-$
- used to assess Cl ions on the AB dysbalance

When to examine AB balance parameters

Metabolic disorders

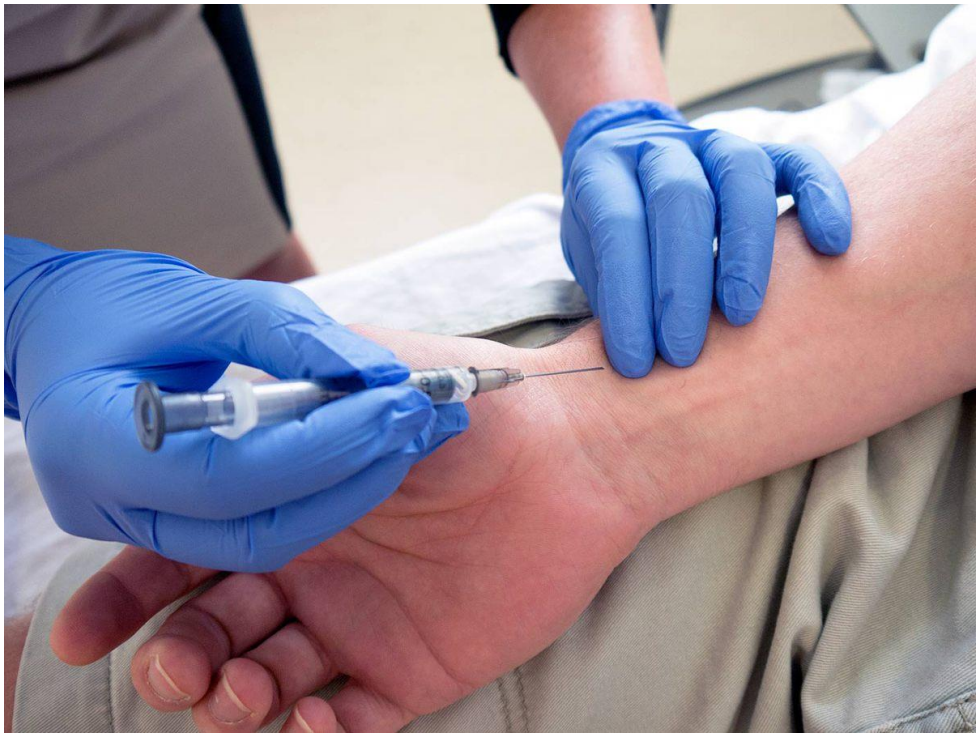
- Metabolic disorders (ketoacidosis, DM not well compensated)
- Poisoning drugs
- Ion dysbalance

Respiratory disorders

- Respiratory insufficiency
- COPD

Taking the blood sample

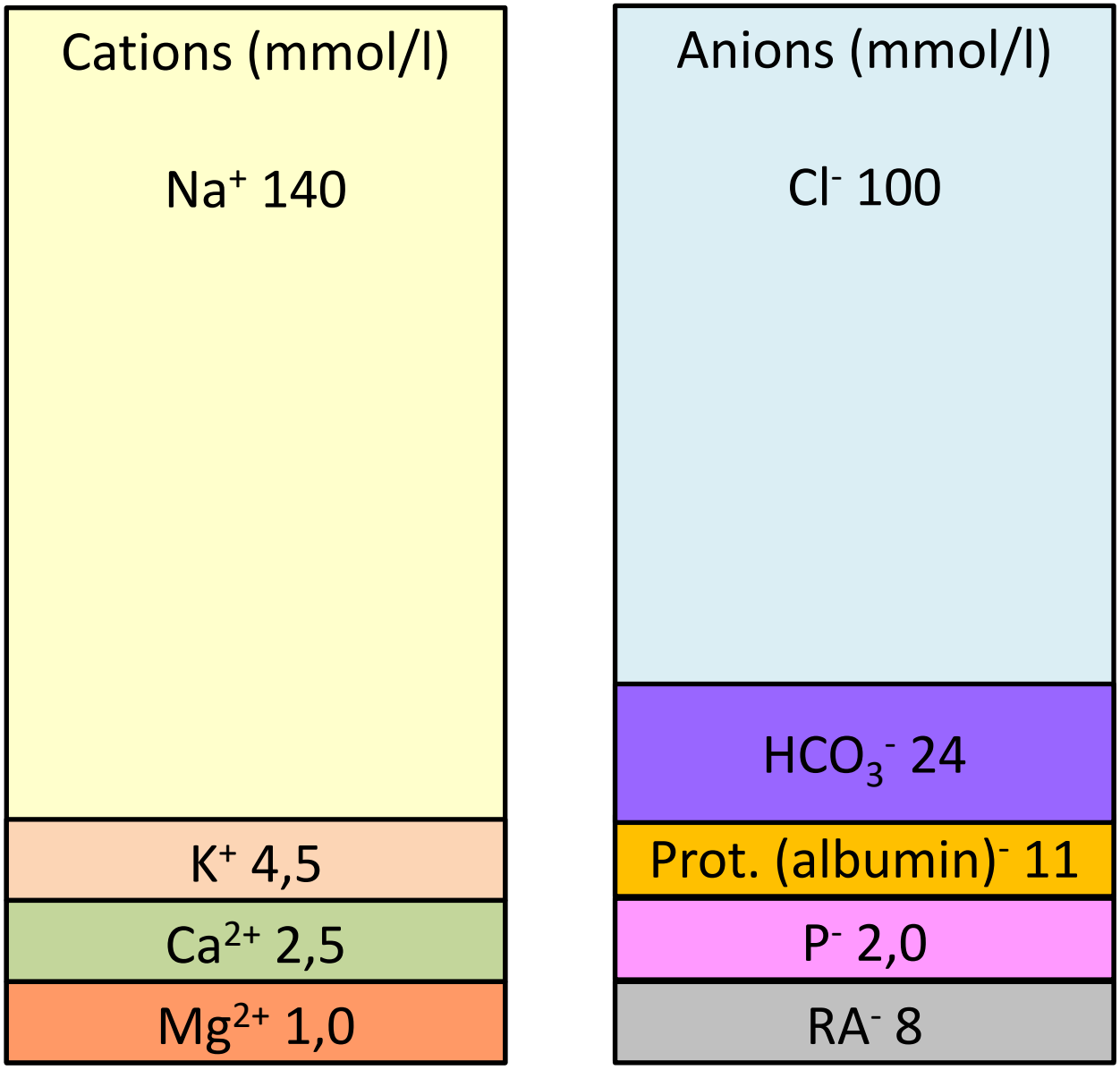
- The sample is taken from the artery without the access of the air



Selected ions and their relationship to AB dysbalance

Ions in blood and cells

	ECF (blood) mmol/l	ICF (cells) mmol/l
Na	140	10
K	4,0	155
Cl	102	8
Ca	2,2	0,001
Mg	1,0	15
P	1,0	65


Cations (mmol/l)
Na^+ 140
K^+ 4,5
Ca^{2+} 2,5
Mg^{2+} 1,0
Anions (mmol/l)
Cl^- 100
HCO_3^- 24
Prot. $(albumin)^-$ 11
P^- 2,0
RA^- 8

The most significant ions in connection with AB dysbalance

- Potassium
- Chlorides
- Calcium

Potassium

- Physiological concentration K = 3,7 - 5,1 mmol/l
- Main ICF ion (98 % protein binding and polysaccharides), stock about 3 500 mmol

Concentration

- plasma 3,7 – 5,1 mmol/l
- cells 110 - 160 mmol/l (ery 95 mmol/l)

→ in strongly hemolytic samples we do not measure the concentration of potassium.

Potassium

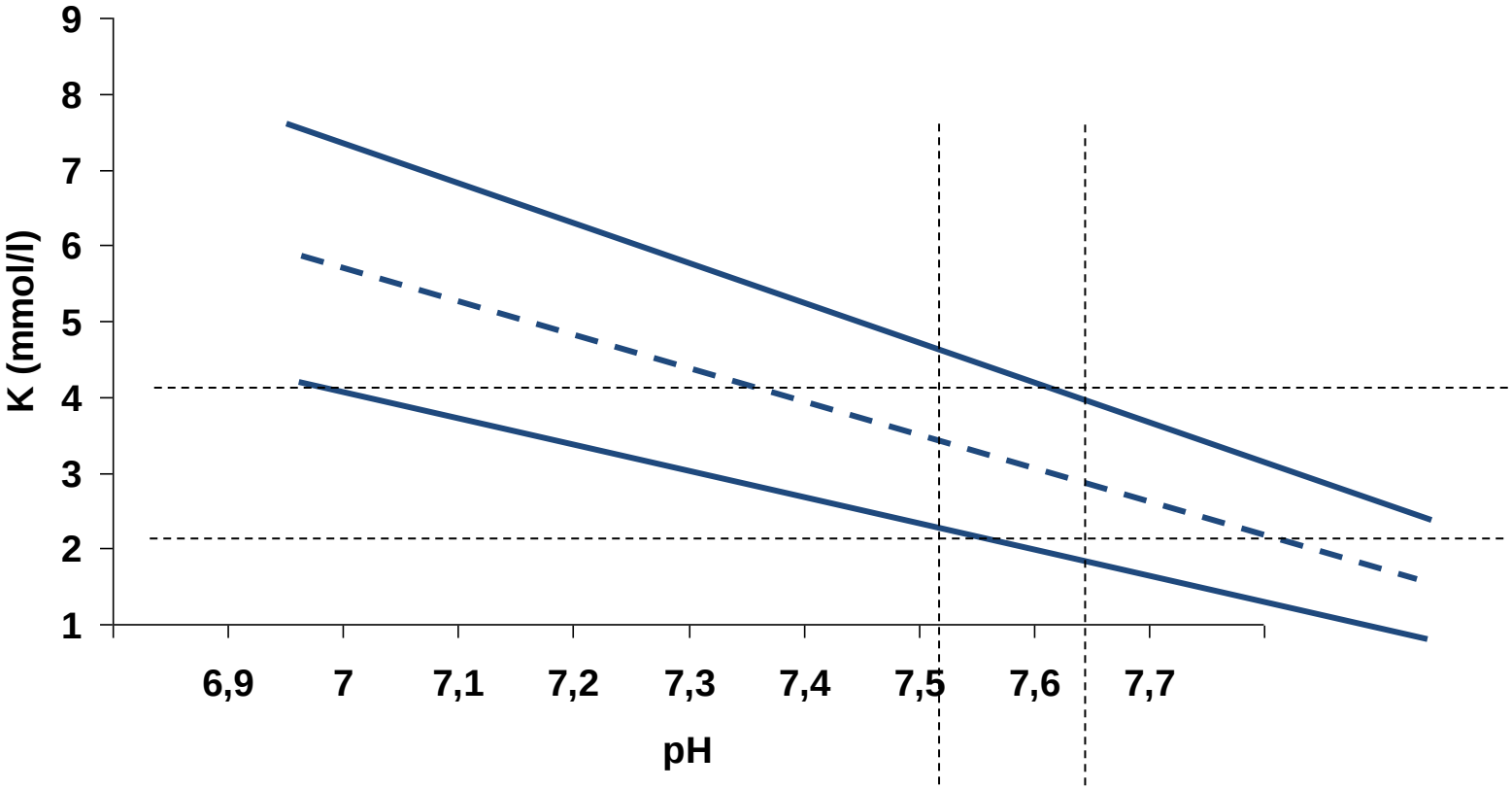
Source: plant-based diet

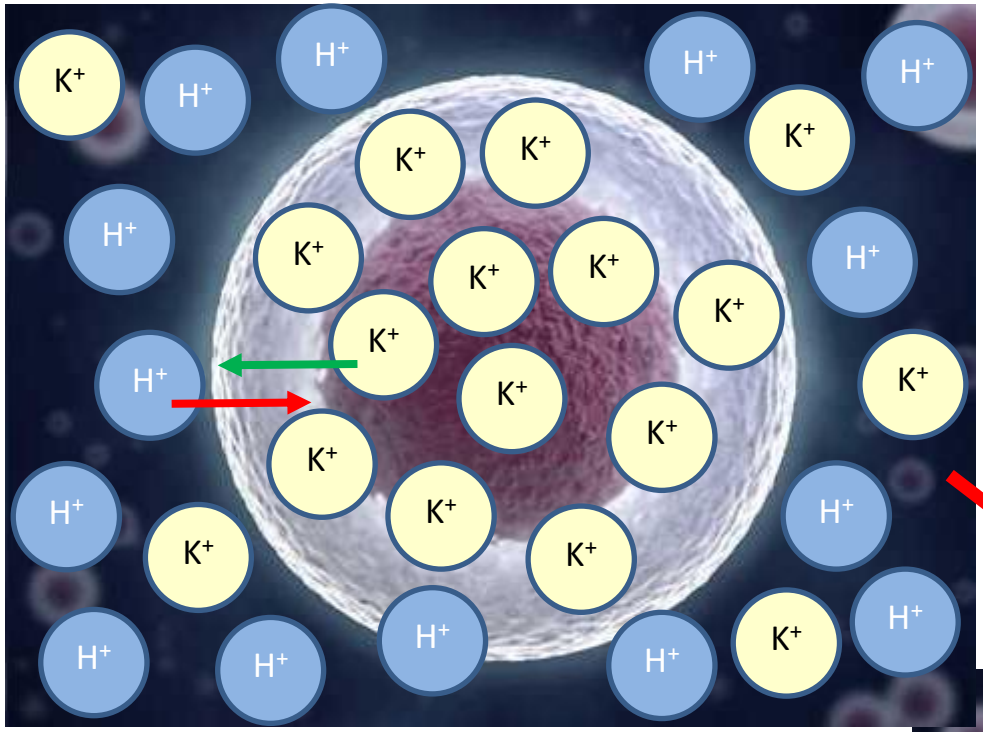
Losses

- Urine: 45 - 90 mmol/24 hrs
- Faeces: 5-10 mmol/24 hrs

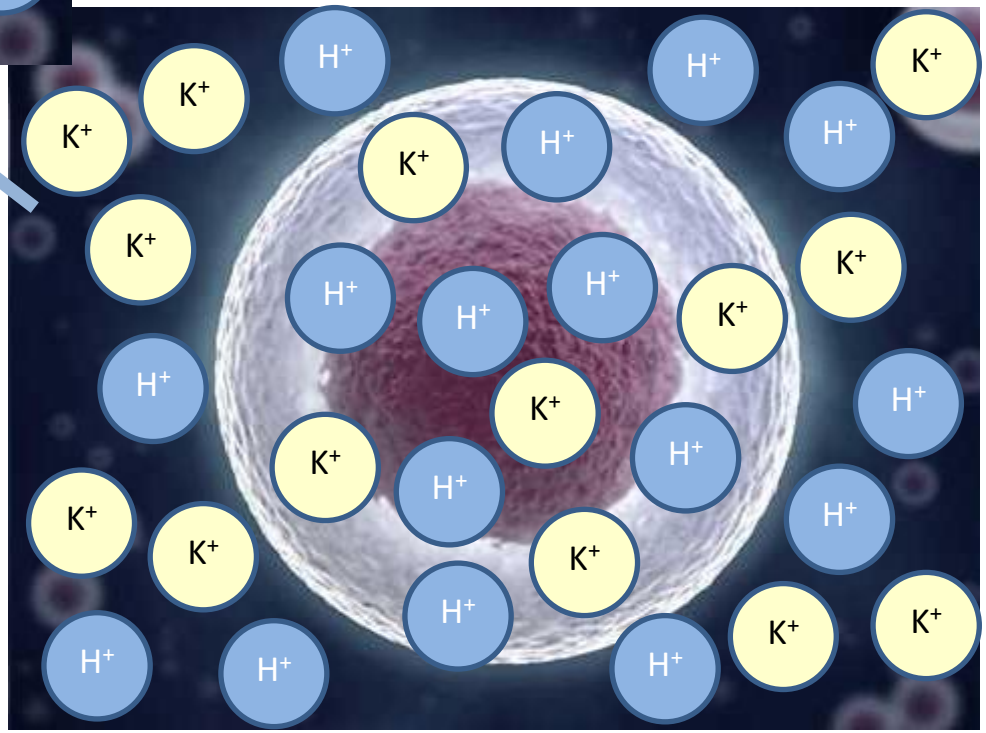
Fundamental relationship to the pH of the organism.

K-pH dependency





Acidosis (↑[H⁺] → ↓pH)
Increase in extracellular concentration of H⁺. H⁺ move into the cell in exchange for K⁺ → hyperkalemia



Alkalosis (↓[H⁺] → ↑pH)
Reduction of extracellular concentration H⁺ - opposite process → hypokalemia

Attention!

- Decompensated diabetics experience diabetic ketoacidosis and hyperkalemia (see the mechanism above).
- When you start to treat the diabetes, the situation reverses (K^+ returns to the cell, and H^+ out). In the meantime, however, due to osmotic diuresis (hyperglycaemia), a significant amount of K^+ leaves the urine → hypokalemia

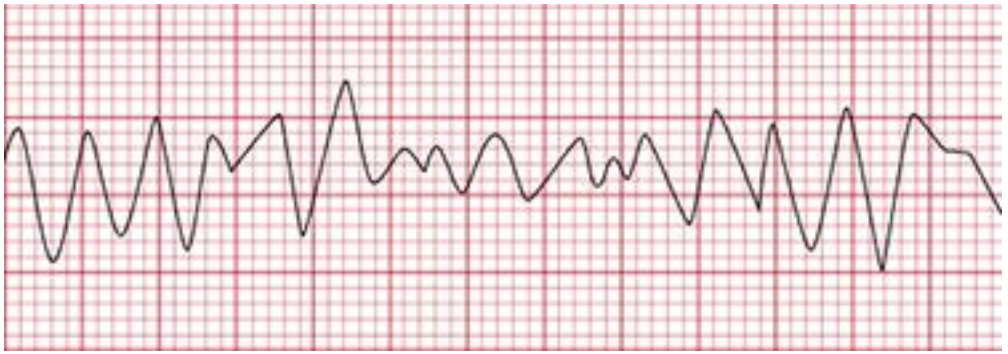
While treating the diabetes, it is necessary to check the ions and substitute eventual hypokalemia

Hyperkalemia

- Increased intake (also iatrogenous)
- Reduced renal excretion (oliguria, anuria)
- K^+ is leaving the cells when: acidosis, haemolysis, catabolism.

Symptoms

- Arrhytmia



Dangerous values:

- > 6,5 mmol/l
- > 9-10 mmol/l → ventricular fibrillation
- HD is required

Hyperkalemia - treatment

Treatment in the patients with functional kidneys:

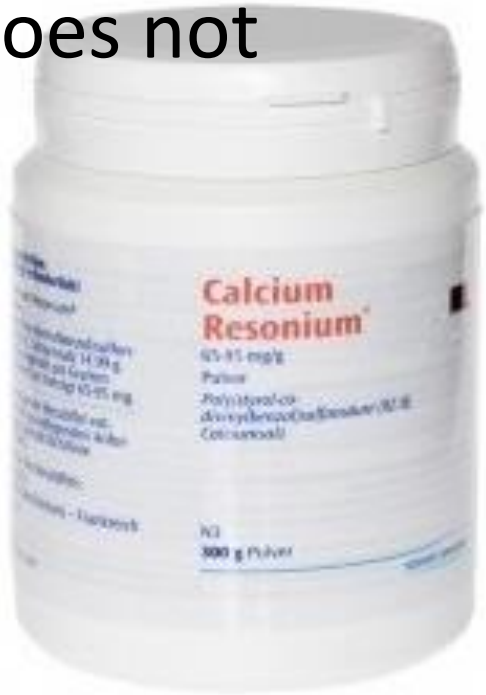
- Diuretics (furosemide)

Treatment if renal failure

- Glucose infusion with insulin (insulin promotes glucose entry into cells together with K^+)
- Ion – Exchange (Calcium Resonium - CaR)
- Hemodialysis

Calcium Resonium (CaR)

- Contains calcium polystyrene sulphonate
- Redundant K^+ is exchanged in the body for Ca^{2+} (especially in the large intestine). CaR resorption to systemic circulation does not occur
- Redundant K^+ is excreted by faeces
- KI: ileus, hyperCa, hyperPTH, multiple myeloma, K < 5 mmol/l



Hypokalemia

- Increased losses: diuretics, GIT causes (diarrhoea)
- Reduced intake (long-term)
- Move into the cells (alkalosis, anabolism)

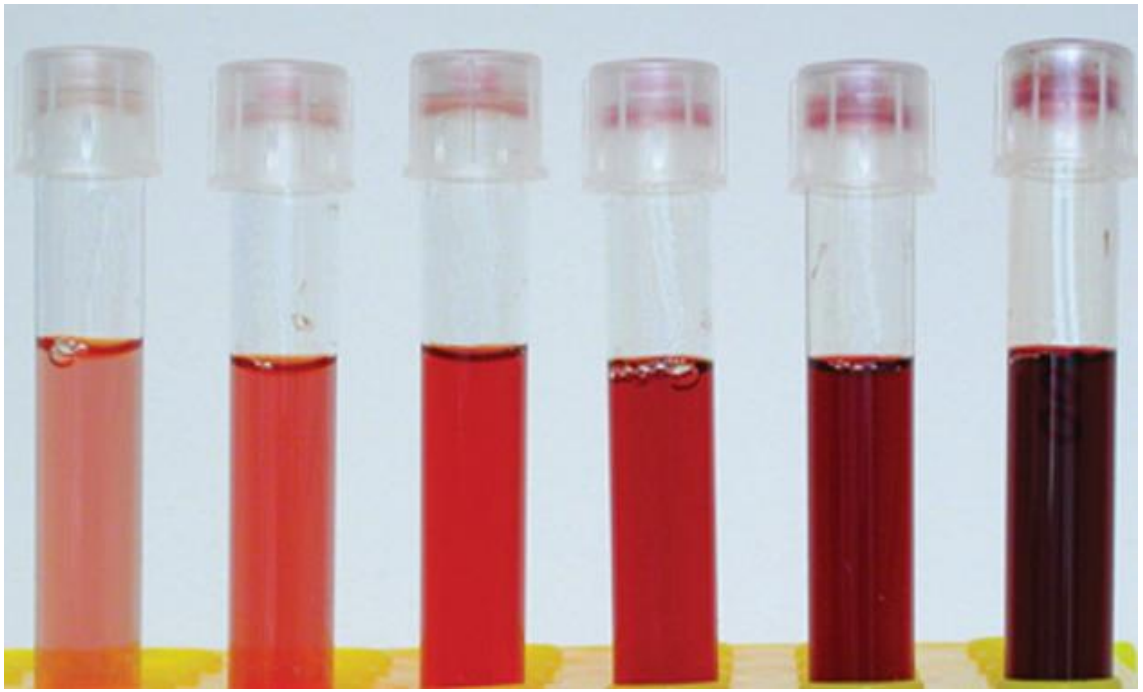
Symptoms:

- Arrhythmia
- Muscle weakness, ileus

Hemolysis

Examination K^+ (erythrocytes!)

- watch out for hemolysis (ery contain a lot of potassium)



Chlorides - Cl

- Physiological concentration 97 - 105 mmol/l
- Main anion of ECF
- ICF 3 - 10 mmol/l

Function:

- osmolality
- maintaining AB balance (change in concentration Cl^- → change in concentration HCO_3^-)
- gastric juice - HCl

Intake in NaCl

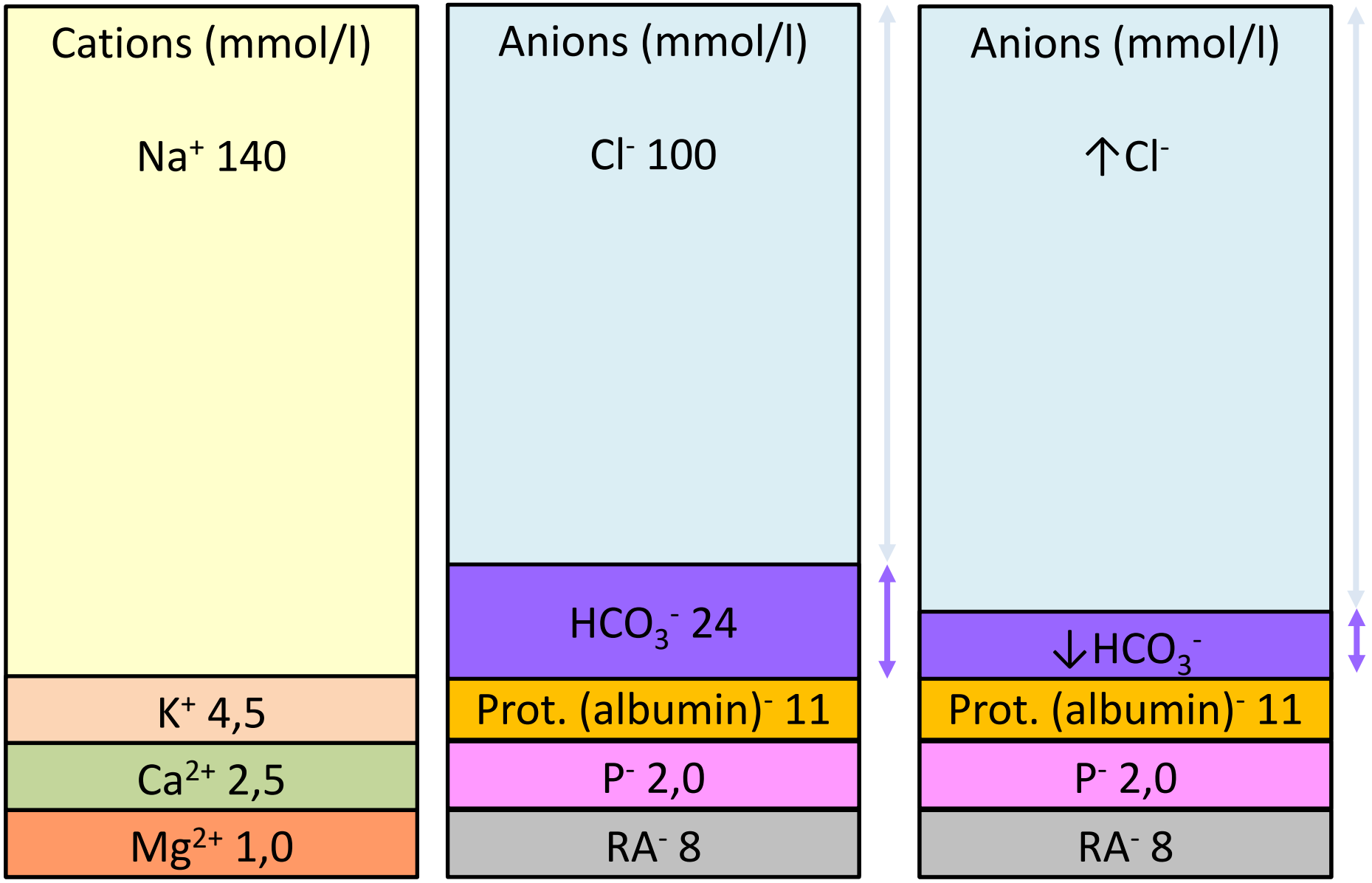
Losses

- Urine 120 - 240 mmol/24 hrs
- Faeces 10 mmol/24 hrs, sweat 10 - 20 mmol

Hyperchloridemia

- Reduced excretion — renal disease
- Increased intake (NaCl) in renal disease
- Increased NaCl by iatrogenous supply (cave FR)

↑Cl^- → ↓HCO_3^- (buffering system limited – is unable to bind H^+) → accumulating H^+ → ↓ pH (development of acidosis)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Mg^{2+} 1,0
Anions (mmol/l)
Cl^- 100
HCO_3^- 24
Prot. (albumin)$^-$ 11
P^- 2,0
RA^- 8
Anions (mmol/l)
↑Cl^-
↓HCO_3^-
Prot. (albumin)$^-$ 11
P^- 2,0
RA^- 8

Hyperchloridemia

Hyperchloridemic metabolic acidosis

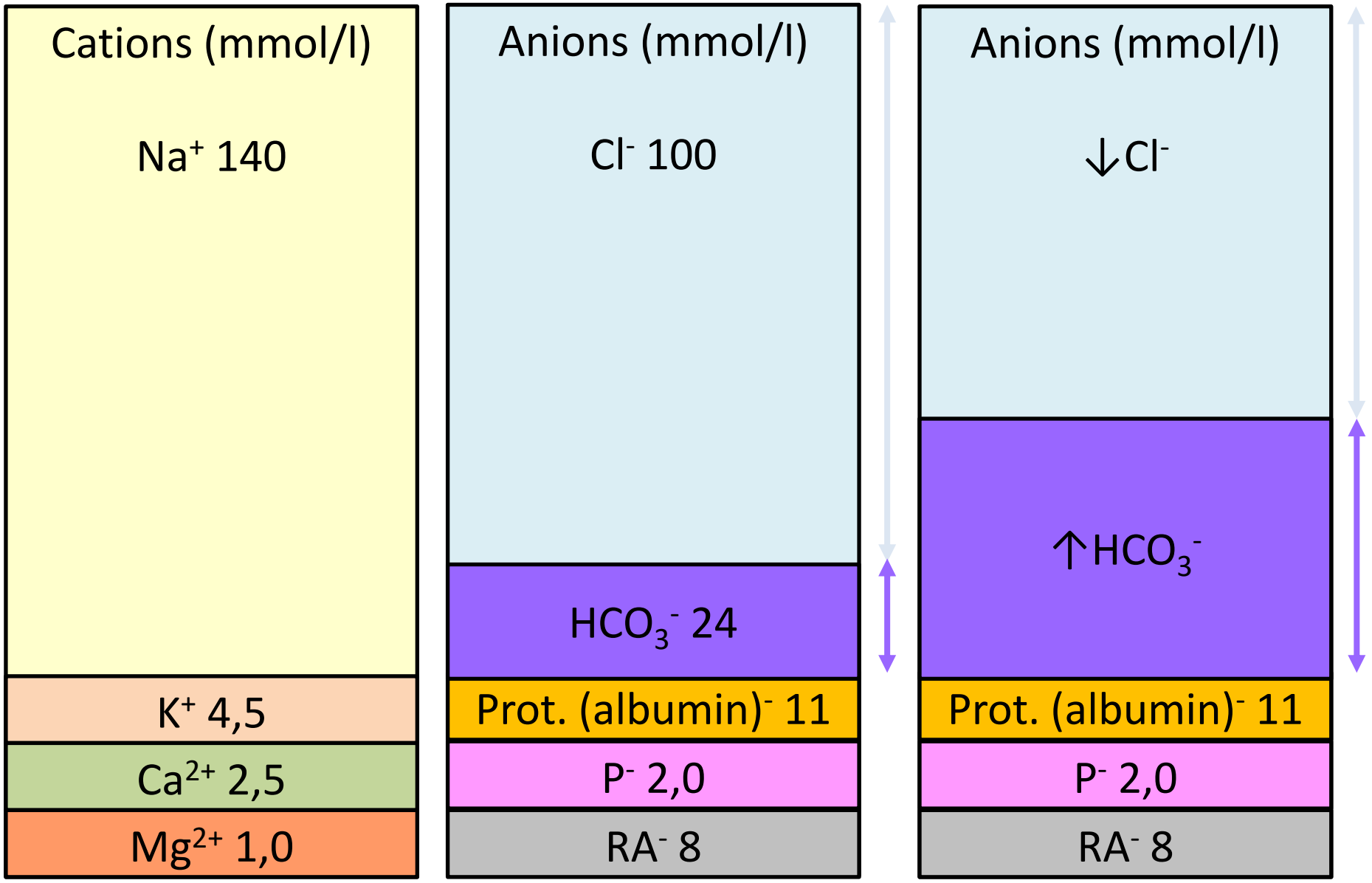
- Beware of long-term saline therapy (when hyperNa, hyperCl do not give more) – a more suitable is glucose infusion if there is no contraindications
- HyperNa (water movements), hyperCl (MAc)

Hypochloridemia

Losses

- Gastric juice (vomiting, suction by NGT)
- Kidneys (diuretics, polyuria)
- Excessive sweating

↓ Cl^- → ↑ HCO_3^- (buffering system in excess), ↓ H^+ (bound with buffer) → ↑ pH (development of alkalosis)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RA^- 8
Anions (mmol/l)
↓Cl^-
↑HCO_3^-
Prot. $(albumin)^-$ 11
P^- 2,0
RA^- 8

Hypochloridemia

Hypochloridic metabolic alkalosis

- Patients with dyspepsia and vomiting
- Suction of gastric juices by NGT

Calcium

- The largest depo in the bones (1,2 kg in the form of hydroxyapatite)

Ca in the blood:

- Ca bound to proteins (undiffusible), mainly albumin (46 %)
- Ca free, ionized[1)] (48 %) – biologically active fraction
- Ca in complex compounds[1)] (6 %), citrates, phosphates, lactate, sulfate

Function: nerves, formation of bone mass

[1)] Diffusible forms of Ca

Total vs. ionized calcium

Total calcium is the sum of:

- Ca ionized (cca 1/2 of total Ca)
- Ca protein-bound (mainly albumin)
- Ca bound to complex compounds

Ionized calcium

- Only ionized Ca has physiological effects (hypo/hyperCa symptoms therefore occur when this fraction changes)
- Hypoalbuminemia → reduced concentration of total Ca (but normal levels of ionized Ca are often present, therefore the patient may not have typical symptoms).

Together with the total calcium, ionized calcium should be examined as well

Calcium

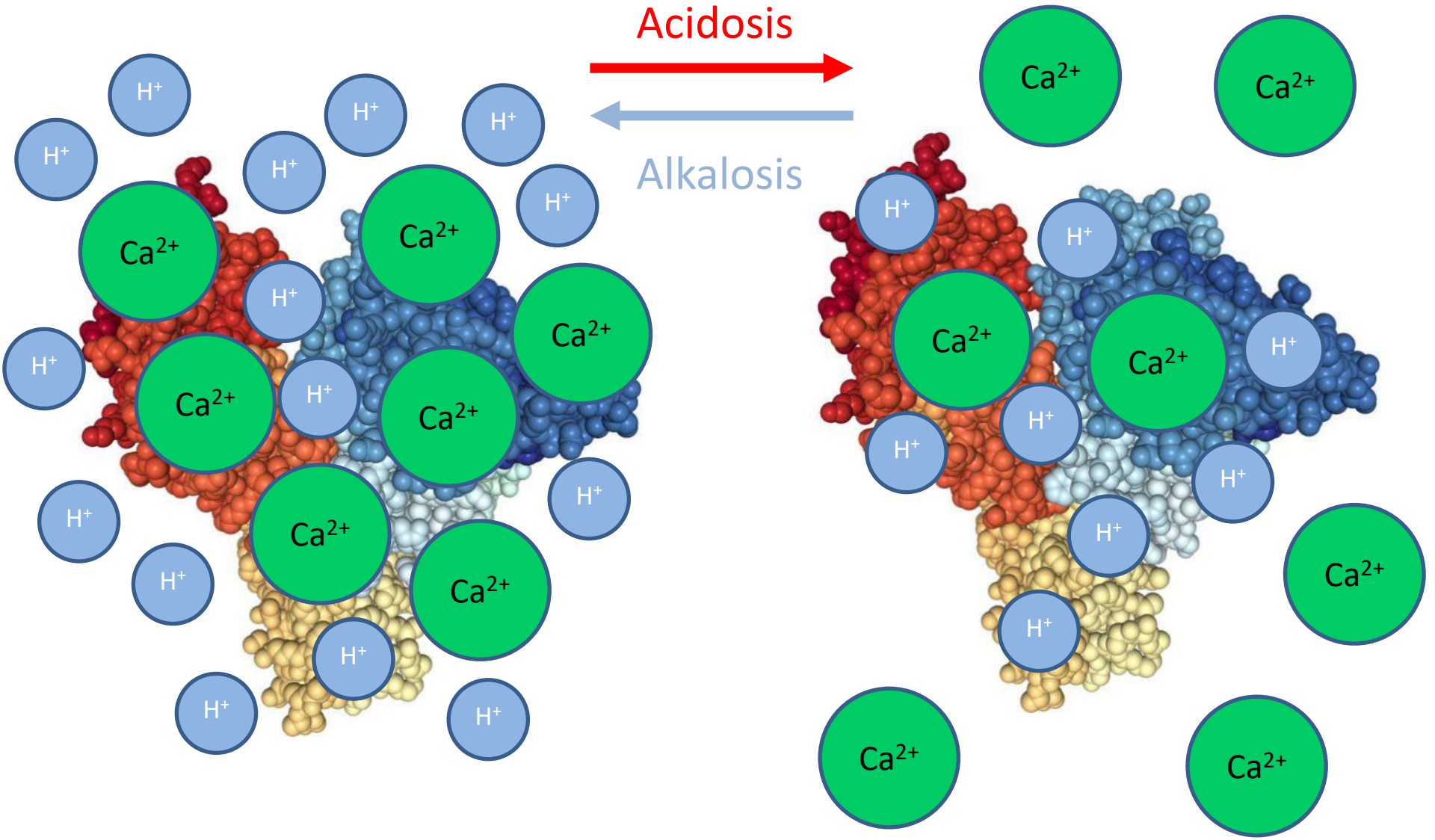
- Physiological concentration: 2,1 - 2,6 mmol/l
- Strict regulation in the organism (very strict reference range)
- Relatively small dysbalance can lead to potential life-threatening conditions

Ca protein binding

Affected by:

- Protein concentrations, in particular albumin (hypoalbuminemia → ↓total Ca)
- pH value
  - ✓ Acidosis → Ca release, free fraction increase
  - ✓ Alkalosis → Ca binding, decrease in free fraction

Changes in calcemia in AB dysbalances



Ca changes in different situations

- Albumin decrease of 10 g/l = Ca decrease of 0.25 mmol/l
- decrease Ca
  - ✓ drugs (furosemid, bisphosphonates)
  - ✓ hyperP, hypoMg
  - ✓ malabsorption, kidney disease, tumours

Changes to AB balance

- pH decrease (acidosis) → hyperCa
- pH increase (alkalosis) → hypoCa

Sampling kits: not into tubes with EDTA or Na-citrate (they bind Ca)

Regulation of Ca levels in the blood

- Calcitonin – parafolicular cells if thyroid gland, reduces Ca levels in the blood
- Parathormon (PTH) – increases Ca levels in the blood (by releasing from the bones, increases reabsorption of Ca in the kidneys, stimulates the formation of calcitriol in the kidneys)
- Vitamin D (active form – calcitriol) – support for resorption of Ca from the intestine

Hypocalcaemia

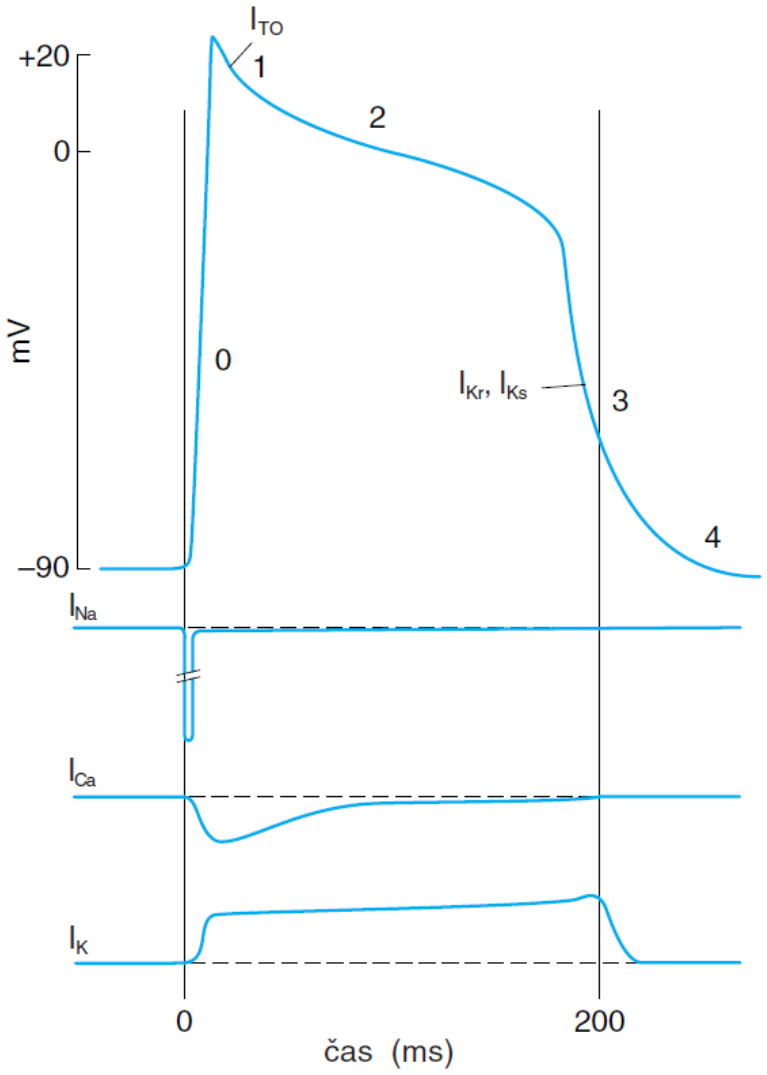
Causes:

- Long-term depletion Ca
- Absorption disorder: vitamin D deficiency (lipophilic vitamin, malabsorption syndromes)
- Parathormone deficiency (iatrogenously, when the thyreoidectomy may accidentally remove the parathyroid glands) – a necessary check of Ca and PTH after surgery.

Symptoms:

- Paresthesia
- Tetany, arrhythmia
- Dyspnoea

Phase of AP of the heart muscle fibers (changes in membrane conductivity for individual ions)

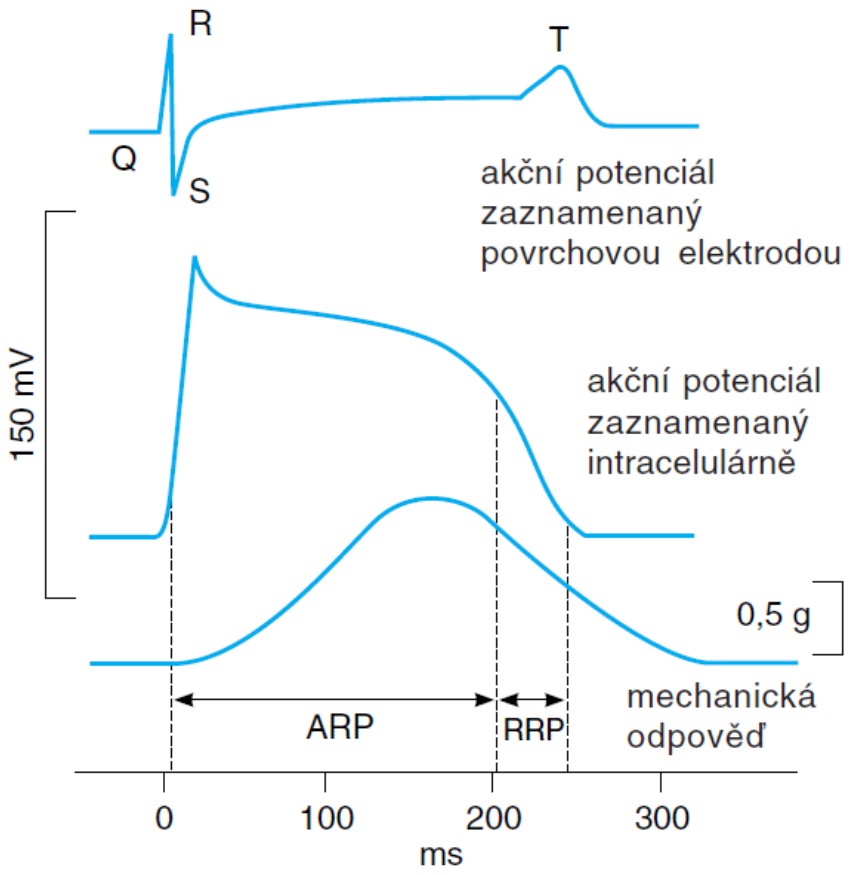


- 0 - depolarisation
- 1 - initial rapid repolarization
- 2 - plato phase
- 3 - late rapid repolarization
- 4 - resting potential

The plato phase (2) is triggered by a slow opening VOC Ca^{2+} (–30 až –40 mV)

The final repolarization (3) on the resting potential (4) is the closure of VOC Ca^{2+}.

AP and mechanical response of heart muscle fibers



ARP – absolute refractory period

RRP - relative refractory period

- During phases 0-2 and half of phase 3, the heart muscle cannot be re-excited (ARP). The RRP then takes until phase 4.
- Therefore, unlike skeletal muscle, tetany (under physiological circumstances) cannot be developed in the heart muscle → protection from malignant arrhythmia.

Strict regulation of Ca in the body is absolutely crucial

- Ca^{2+} ions play an essential role in maintaining ARP. HypoCa may lead to cardiac muscle tetany and life threatening arrhythmias
- The need to maintain an optimal pH with regard to Ca concentration protects from malignant arrhythmia

Hypercalcaemia

Causes:

- Increased absorption (excess vitamin D)
- Excess parathormone (adenomas parathyreoid glands)

Symptoms:

- Myasthenia
- Nausea
- Polyuria

Another ions (Na, P, Mg) + RA

Minimal relationship to AB balance influence

- Na – deviations lead to water dysbalance
- P – phosphates affect Ca concentration - product [Ca] x [P] = konst.
- Mg – nerve irritation

RA = residual anions (S, organic acids)

Sodium

Physiological concentration: 135 - 145 mmol/l

- ECF 50 %
- Bone tissue 40 %
- ICF 10 %

Na is osmotically active → water binding (retention Na → water retention).

Intake NaCl 8-11 g/day (however, 1 g/day is sufficient)

Losses:

- Urine: 120 - 240 mmol/l
- Sweat: 10 - 20 mmol, faeces 10 mmol

Meaning of examination Na: hydration, osmolality

Sodium

- Na^+ has an essential relationship to influencing the distribution and balance of water
- The relationship with AB balance is negligible
- Concentration disturbances Na^+ → water management disorders (hyper/dehydration)
- 3 regulatory systems: ADH, aldosterone, natriuretic peptide

Phosphorus

- Stock 600 g (85 % - bones, 15 % - soft tissues)
- Main ion of ICF: organic phosphates (phospholipids, phosphoproteins, ATP, nucleic acids)
- Inorganic phosphates (serum - mono and dihydrophosphate, protein binding, P – buffer), hydroxyapatite in the bones

Fluctuations in phosphatemia

- Increase - chronic renal failure
- Reduction - absorption disorders, antacids

Calciophosphate product

$$[Ca] \times [P] \leq 4{,}4\ mmol^2/l^2$$

Increased product Ca x P in plasma:

- Leads to the precipitation of calcium salts in soft tissues → HypoCa
- Inorganic P inhibits 1-hydroxylation → reducing creation 1,25-dihydroxyvitamin D →↓ resorption of Ca in the intestine → HypoCa

In patients with CKD who are supplemented with vitamin D(↑ Ca) ectopic calcification is a common complication if hyperphospatemia correction is not sufficient.

Magnesium - Mg

- 55 % in the bones (25 g)
- 45 % intracellular (main ICF ion - ATP, GTP)
- Blood
  - ✓30 % protein binding
  - ✓55 % ionized fraction
  - ✓15 % complexes: citrates, phosphates, other anions
- Function: nervous-muscle irritation, bone mass, enzyme cofactor. In the context of AB balance minimal importance.

Residual anions

- RA includes organic acids
- Lactate (product of anaerobic glycolysis – examination is commonly available in the labs) – respiratory arrest, shock, post-resuscitation conditions, biguanides (metformin), etc.
- Ketosubstances (acetoacetate, β-hydroxybutyrate, aceton)

Production of ketosubstances during the decompensation of DM

Absolute (DM1) / Relative (DM2) Insulin deficiency

↓

Glucose entry failure in cell → hyperglycaemia

↓

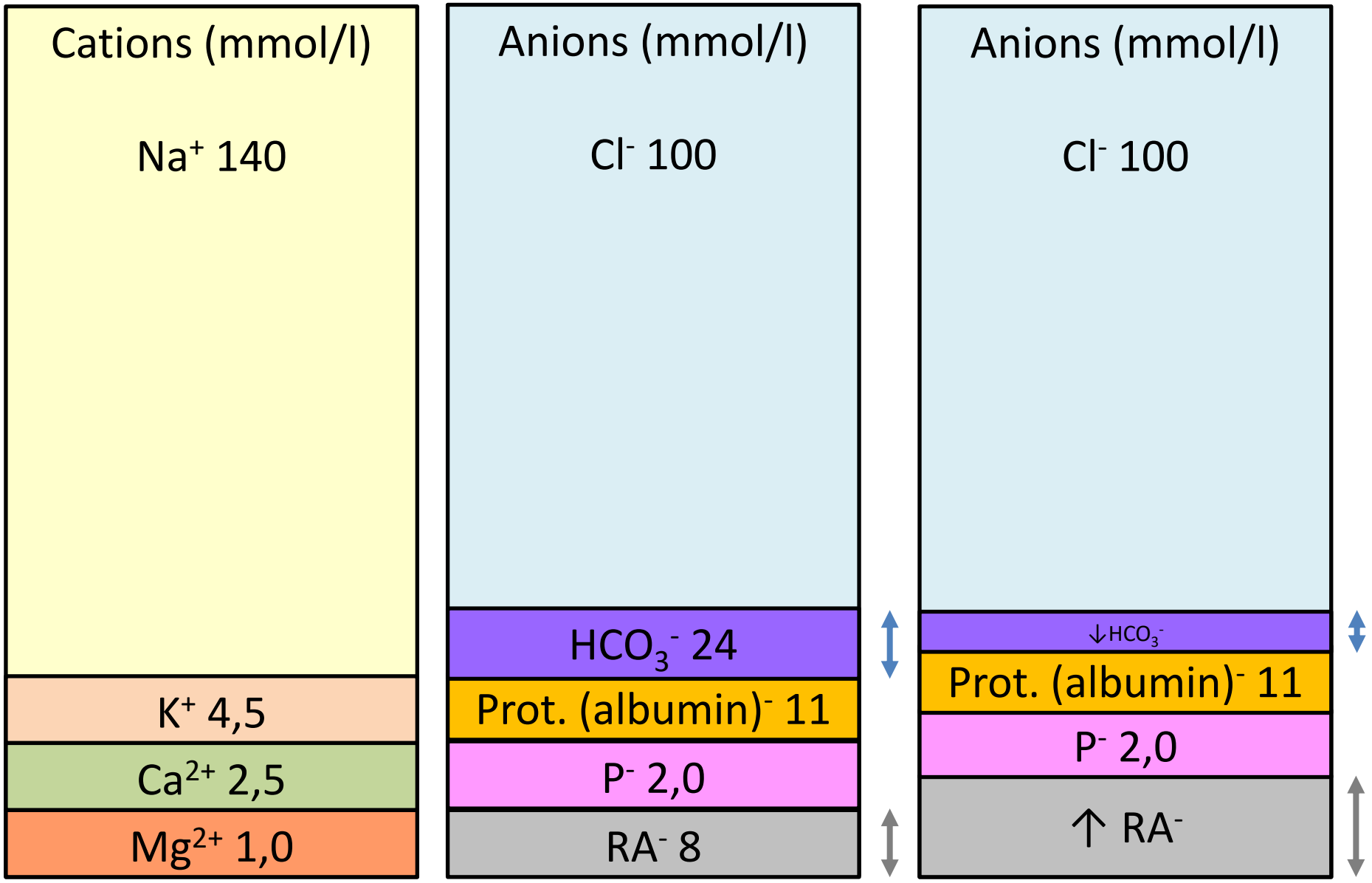
Instead of glucose, cells utilize FA (β-oxidation)

↓

Production of ketosubstances →↓pH

↓

Diabetic ketoacidosis (complications - DM coma)


Cations (mmol/l)
Na+ 140
K+ 4,5
Ca2+ 2,5
Mg2+ 1,0
Anions (mmol/l)
Cl- 100
HCO3- 24
Prot. (albumin)- 11
P- 2,0
RA- 8
Anions (mmol/l)
Cl- 100
↓HCO3-
Prot. (albumin)- 11
P- 2,0
↑ RA-

Combined AB disorders (1)

The patient vomits for several days. What changes can I expect in ABB?

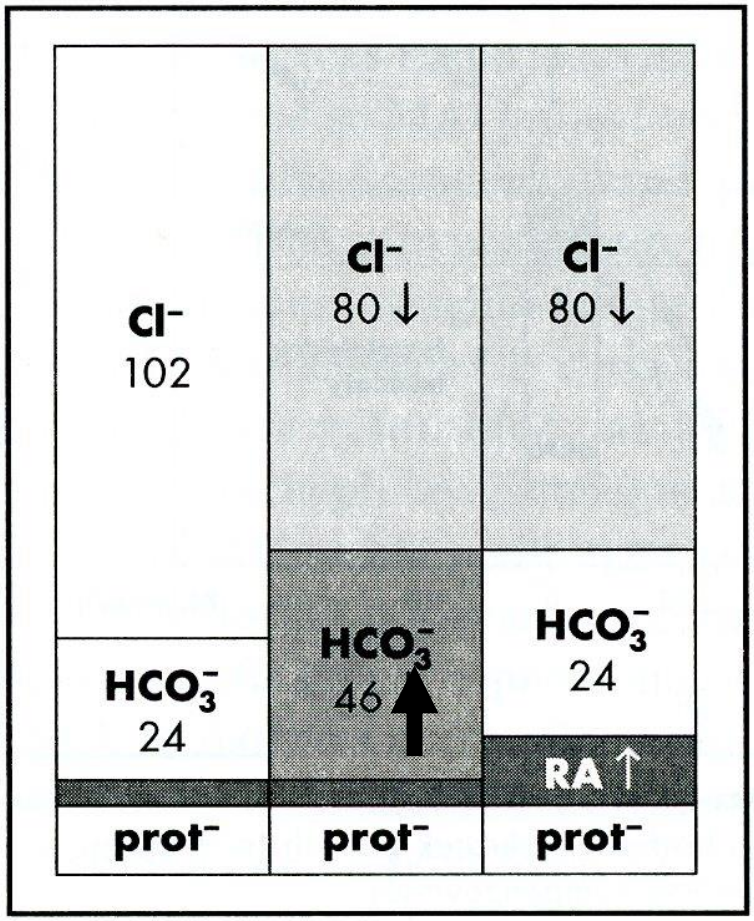
- Loss of HCl → hypochloridemic MAlk

+

Due to nausea, the patient starves and does not want to receive food.

- β-oxidation of FA → production of ketosubstances → metabolic ketoacidosis

Development of combined AB disorder



Type of AB disorder

- Combination of metabolic acidosis and metabolic alkalosis
- pH can be normal, only pH is not enough to investigate
- The proportion of ions must be determined (in particular Cl^-) on AB disorder → always examine the ions!

Combined AB disordes (2)

Non-compliant patient, diabetic who forgets to inject insulin. What changes can I expect in ABR?

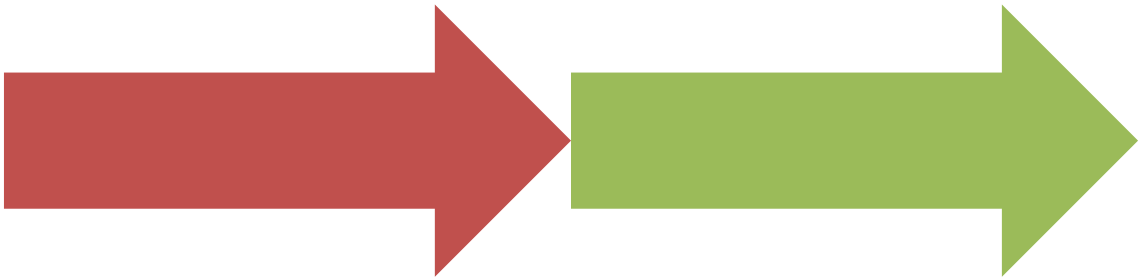
- Hyperglycemia → β-oxidation of FA → DM ketoacidosis

+

- Hyperglycemia → osmotic diuresis → polyuria and dehydration (hypovolaemia) → tissue acidosis → lactic acidosis

Type of AB disorder

- Combination of two metabolic acidosis (ketoacidosis from DM + lactic acidosis from tissue hypoxia)



Combined AB disorders (3)

Patient with CP arrest. What changes can I expect in ABR?

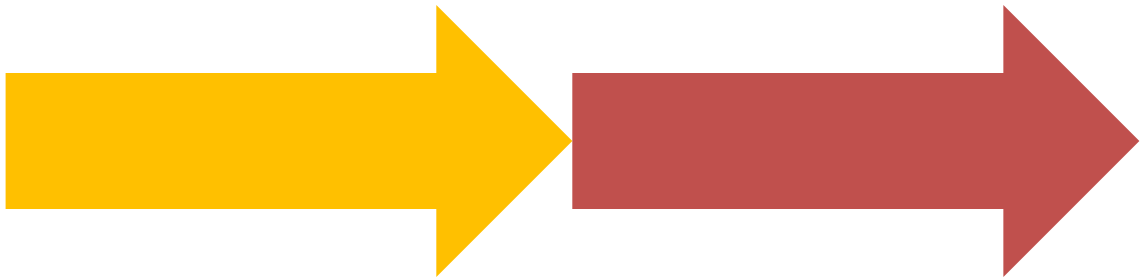
- Increase of CO_2 due to respiratory insufficiency → respiratory acidosis

+

- Tissue hypoxia → lactic acidosis

Type of AB disorder

- Combination of respiratory acidosis and lactic acidosis



Take home message

- The stability of pH is a prerequisite for maintaining physiological processes in the organism
- ABB disorders are associated with the movement of ions between the compartments as well as with changes in their concentrations
- K^+, Cl^-, Ca^{2+} have a significant relationship to ABB disorders (K^+ /pH dependency, Cl^- /HCO_3^- and Ca^{2+}/pH relationship)
- Na^+, P^-, Mg^{2+} dysbalances are primarily associated with other disorders (transfer of the water between compartments, neuromuscular excitability etc.)
- Residual anions – their contribution to ABB disorders is very important and should be clarified as well

Take home message

- In case of AB dysbalances the basic ions should be examined: Na, K, Cl + better Ca, P, Mg. Without this, (especially combined ABB disorders) cannot be evaluated at all.
- The measurement of lactate should be performed (it has a contribution to lactic acidosis)
- Beware of hypokalemia when compensating DM, hyperglycemia should be trated very slowly (changes of K^+ when pH changes) + brain edema
- Do not forget to examine the ionized Ca (biologically active form)