

# Sodium

**Sodium** ( $\text{Na}^+$ ) or **sodium** is the most abundant metal in the human body; is **the major cation of extracellular fluids**. The daily requirement of sodium is about 1 g, its intake is safely ensured by salt ( $\text{NaCl}$ ) in the diet (5-15 g per day). The total amount of body sodium in an adult is about 60 mmol / kg body weight. About 30% of sodium is immutable in bone and cell structures. It is excreted mostly in the urine, followed by sweat and faeces.

## Reference limits:

- **plasma** : 132-142 mmol/l;
- **urine**: 120-140 mmol / day.
  - Sodium has <sup>1</sup> element (alkali metal).
  - As a pure metal, sodium is a highly reactive reducing agent, reacting violently with water, contact with skin and mucous membranes can cause burns (reactivity) and corrosion of the resulting hydroxides (strong bases).

## sodium and water management

The  $\text{Na}^+$  content in the body determines the size of the ECT:

- increased intake of  $\text{Na}^+$  → hyperhydration (weight gain, swelling);
- reduced  $\text{Na}^+$  supply → increased water losses;
- serum  $\text{Na}^+$  concentration is not a measure of  $\text{Na}^+$  content in the body (eg in heart failure there is often hyponatremia, even if the total sodium content in the body is increased → therapy: not  $\text{Na}^+$  supply, but dehydration of the body);
- abnormal serum  $\text{Na}^+$  levels may also indicate a water management disorder, an osmoregulatory disorder.

## sodium regulation

- The renal countercurrent mechanism is involved in regulation, together with osmoreceptors in the hypothalamus, which control ADH secretion and thus maintain water content in the body, which is very closely related to  $\text{Na}^+$  concentration in the ECT, so that sodium is maintained in the range of 132-142 mmol / l. that water intake can fluctuate considerably.
  - A defect in the renal capacity of the kidneys, when associated with inadequate water intake, leads to hyponatremia.
  - A defect in concentration capability results in hypernatremia.
  - **Thus, serum osmolality is related to  $\text{Na}^+$  concentration.**
    - Hypernatremia always means hyperosmolality.
    - However, normal natremia or hyponatremia does not always mean euosmolality resp. hypoosmolality; there are clinical situations (eg hyperglycaemia) where a direct relationship between S- $\text{Na}^+$  and osmolality does not apply.
    - It depends on the nature of the solutes whether or not they contribute to increasing the effective osmolality; those that are membrane permeable, such as urea, ethanol, methanol, ethylene glycol, can cause hypertonicity without dehydration; Insulin - deficient glucose does not enter the cells and its presence in the ECT causes water to move from the ICT to the ECT, resulting in intracellular dehydration and hyponatremia (so-called translocation), as is infused mannitol.
- 

## disorders of natremia

### Prerequisites for fault assessment:

- **clinical information:**
  - knowledge of current weight, comparison with standard
  - we observe hydration
  - clinically obvious dehydration - 2.5-5% of body weight
  - hyperhydration - 2.5-7.5%
  - vomiting, diarrhea, diuresis, sweating
- **laboratory information:**
  - serum  $\text{Na}^+$
  - serum glucose
  - serum and urine osmolality
  - $\text{Na}^+$  urine losses
- Na dose calculations need to be considered more broadly

**Therapeutic doses of water and salts** - 2 components:

1. *corrective* - they correct the existing deficit in the body
2. *substitution* - we cover measurable and non-measurable losses from the organism

## Physiological hydration with hyponatremia

- weight unchanged
  - **Causes:**
    - *Acute conditions:*
      - water losses were covered by glucose infusion or drinking water
      - an osmotic gradient between ECT and ICT is evolving with the transfer of water to ICT
      - clinical signs of hyposmolality in acute conditions occur when  $S-Na^+$  falls below 127 mmol / l.
      - isotonic saline solutions are usually sufficient for correction:
- $$\text{correction dose} = (\text{To target} - \text{To detected}) \times F \times CTH$$
- F - factor for calculation of distribution space (♀ 0.55; ♂ 0.6; children 0.65–0.75)  
CTH - patient weight
- *Chronic conditions:*
    - atrophy and catabolism of cellular structures, decreased tonicity (ICT) of cells → transfer of water to ECT → dilution of ECT
    - supply correction will not solve anything, it would just be more excreted
    - causal treatment, securing energy intake
  - **Therapy:**
    - Sodium should only be used in symptomatic hyponatraemia, depending on the clinical condition

## Physiological hydration with hypernatremia

- Cause: usually iatrogenic - administration of concentrated salt solutions.
- patient weight unchanged, ECT is hyperosmolar, compared to ICT, water transfer from cells to ECT; risk of pulmonary edema.
- correction: estimation of excess  $Na^+$  in the organism and calculation of water deficit

$$Na^+ \text{ excess} = F \times m (\text{patient weight in kg}) \times [\text{measured } S-Na^+ - 137 \text{ mmol / l}]$$

$$H_2O \text{ deficit} = F \times m (\text{patient weight in kg}) \times [(\text{measured } S-Na^+ \div 137) - 1]$$

- **Therapy:**
  - we administer diuretics in an infusion of 5% glucose
  - fluid administration would lead to hyperhydration
  - in severe cases by hemodialysis.

## Dehydration with normonatremia

- Cause: proportional loss of water and electrolytes (GIT, kidneys, transfer to the third space - eg peritoneum)
- Weight reduced, no shifts.
- Dangerous mainly because the loss only affects extracellular fluid ! - There is a risk of collapse
- Oliguria develops,  $Na^+$  in urine is very low, but urine osmolality increases (U-urea rises)
- Prerenal uremia occurs
- 
- **Therapy:**
  - we supply isotonic solutions with a weight that is missing - we give 2/3 and further according to the patient's reaction.

## Dehydration with hyponatremia

- Caused by renal or extrarenal losses of NaCl and water, which are insufficiently covered, but more by water (non-electrolytes) than NaCl
- Weight reduced, water transfer
- Extrarenal losses caused by e.g. diarrhea, skin burns or moving to the third area induces hypovolemia, which stimulates the regulatory system renin-angiotensin and increases the secretion of aldosterone and ADH with subsequent changes in renal hemodynamics, the amount of urine is small,  $U-Na^+$  and  $U-Cl^-$  is below 20 mmol / l
- Renal losses due to diuretic overdose, primary adrenal insufficiency or nephropathy have  $U-Na^+$  and  $U-Cl^-$  increased (above 20 mmol / l)
- **Therapy:**
  - we supply water according to the difference in weight and ions according to the equation above

## Dehydration with hypernatremia

- **Three situations:**

1. water and a small amount of isotonic fluid are missing → relative increase in sodium concentration
2. sodium concentrations did not change, only water was lost
3. there was a loss of water and an increase in sodium concentration

- however, water passes from intracellular fluid (ICT) to extracellular fluid (ECT)

- **Therapy:**

- loss of mostly water - compensation of water deficit
- water loss and  $\text{Na}^+$  - treatment in two steps:
  1. administration of glucose solutions or hypotonic electrolytes;
  2. replenishment of NaCl with isotonic NaCl solution (even in isotonic solution is hypotonic → corrects)
- water loss + increase in  $\text{Na}^+$  concentration - infusion of 5% glucose.

## Hyperhydration with normonatremia

- Cause: e.g. excessive infusions with reduced water and electrolyte excretion
- There is no osmotic gradient between ECT and ICT
- Circulating volume increases, edema occurs
- **Therapy:**
  - according to the differences in weight, we influence the supply of fluids or diuretics

## Hyperhydration with hyponatremia

### Acute conditions:

- Causes: water intoxication
- The risk increases after administration of drugs with antidiuretic effect ( barbiturates , opiates ), also during childbirth (antidiuretic effect of oxytocin ), in conditions with chronic water excretion, in schizophrenia (ADH release
- **Danger of increased intracranial pressure**
- Treatment: slow correction with NaCl solutions - min. 12-24 h

### Chronic conditions

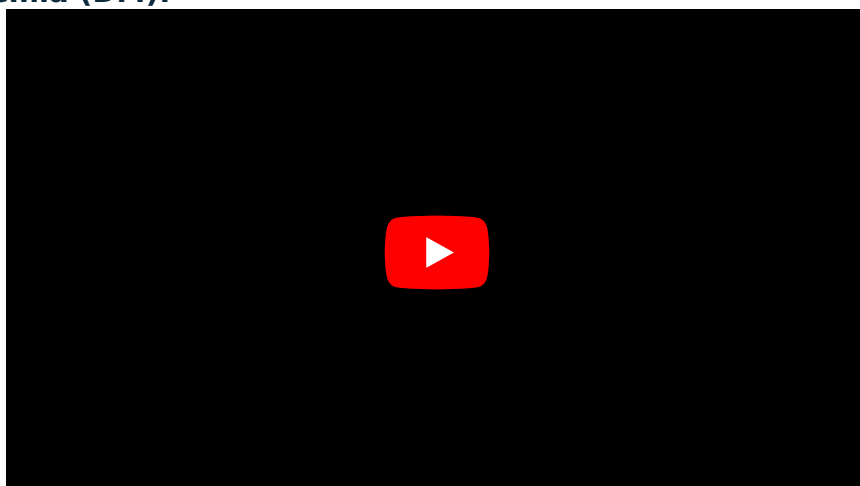
- Causes: edema caused by heart failure, nephrotoxic syndrome, liver cirrhosis
- Hyponatremie je asymptomatická, pokud  $\text{S-Na}^+$  nepoklesne pod 120 mmol/l.
- Treatment: water supply must be limited (less than 1 l / 24 h); NaCl intake must be balanced

## Hyperhydration with hypernatremia

- Causes:
  1. *Na* - hyperaldosteronism ( Conn's disease ), decrease in filtration
  2. *hypertonic fluid uptake* : eg drinking seawater, administration of concentrated saline solutions
- Hyperosmolarity of ECT versus ICT → transfer of water from ICT to ECT → expansion of ECT - **danger of pulmonary edema**
- **Treatment** : diuretics.

## Pseudohyponatremia

### Pseudohyponatremia (DM):



- This condition occurs when **the content of solutes in the plasma increases** (hyperproteinemia, hyperlipidemia)

- Under normal circumstances, the content of solutes in plasma is 7%, if e.g. to 14%, with a constant  $\text{Na}^+$  concentration in the plasma water, the  $\text{Na}^+$  concentration in the whole plasma decreases , which causes misinformation about the  $\text{Na}^+$  state in the ECT
- Delivery of saline solutions may result in dangerous hypernatremia
- **The true concentration of  $\text{S-Na}^+$  is determined by examination on instruments with an ion-selective electrode**

## Links

### Related

- Sodium in urine
- Dysbalance natria • Dysbalance natria (pediatrics))
- Dehydration

*Other chapters from the book **MASOPUST, J., PRŮŠA, R. :: Pathobiochemistry of metabolic pathways***  
 Template:Patobiochemie metabolických drah (Masopust)

### External links

- Sodium (Czech wikipedia)
- Sodium (English wikipedia)

### Resources

- - MASOPUST, Jaroslav and Richard PRŮŠA. *Pathobiochemistry of metabolic pathways*. 2nd edition. Charles University, 2004. 208 pp. 172–174.

### References

- - SCHNEIDERKA, Petr, et al. *Chapters from clinical biochemistry*. 2nd edition. Prague: Karolinum, 2004. ISBN 80-246-0678-X .