

Function and significance of certain electrolytes and their informative value: sodium (Na), potassium (K) and chloride (Cl)

Sodium (Na)

Sodium is the most important cation in the extracellular fluid. Hence, it is essential for maintaining osmolality or the distribution of water between the extracellular space (ECS) and the intracellular space (ICS). Hypo- and hypernatraemia occur when the sodium/water ratio in the ECS shifts towards water or sodium. This is often caused by an increase or decrease in total body water without an absolute change in electrolytes. But since Na is the main component of osmotically active substances in the ECS, hypo- and hypernatraemia are associated with changes in osmolality.

Na is the major electrolyte in the ECS and potassium (K) in the ICS. This asymmetric distribution of the electrolytes across the cell membrane requires active exchange of both cations by the Na/K-ATPase.

As a result, body fluids are in osmotic equilibrium. The distribution of water between the ECS and the ICS is normally constant and only shows slight fluctuations of just 1 – 2%. Acute changes in serum Na concentration which are not accompanied by a corresponding change in intracellular K concentration cause permeation of water from the ECS to the ICS. Cellular oedema occurs.

Concentrations of Na in serum and in the interstitial fluid are almost identical. Na causes about 95% of the osmotic pressure. The organism regulates the plasma Na concentration by adjusting the water content of the ECS and keeping the total body Na and Na in serum constant within a narrow range. This is done by drinking or by renal excretion of free water.

Examples of diseases and causes which can lead to hyponatraemia

cardiac insufficiency	reduced cardiac output and decreased circulating blood volume cause activation of the renin-angiotensin system and release of arginine-vasopressin (AVP) = water and Na retention, development of hypervolaemic hyponatraemia
osmotic diuresis	loss of Na and water with reduction of extracellular fluid volume (ECFV) = hyponatraemia, e.g.: diabetes mellitus with glucosuria
renal tubular acidosis (RTA), metabolic alkalosis	excretion of bicarbonate \uparrow , causes osmotic excretion of cations such as Na, K and Ca \uparrow = bicarbonaturia, urine Na concentration \uparrow , cause of RTA is defect in bicarbonate reabsorption in the prox. tubule
pancreatitis, peritonitis, vomitus, diarrhoea, blood loss	extrarenal loss of water and Na = ECFV \downarrow = fluid loss into the third space, e.g. into the abdominal cavity in case of peritonitis, into the intestinal lumen when suffering from pancreatitis, highly concentrated urine, serum Na \downarrow

hypothyroidism	non-osmotic release of AVP, cardiac output per minute ↓ = less osmotically active substances in diluting segments (tubules)
ketonuria	in poorly controlled diabetes mellitus there is excretion of negatively charged ketone bodies = excretion of Na, K and NH ₄ = hyponatraemia with reduction of the ECFV, urine Na concentration increased
chronic kidney disease	formation of oedema and hyponatraemia, causes: glomerular filtration rate (GFR) ↓ = reduced excretion of water and Na, resulting in a dilution effect, consequently hyponatraemia
glucocorticoid deficiency/ hypoadrenocorticism	AVP secretion despite hypoosmolality, renin concentration often unchanged, normal cortisol concentration required for AVP secretion

Examples of diseases and causes which can lead to hypernatraemia

diabetes insipidus (DI)	DI results from decreased central AVP secretion (hypothalamus) or end organ resistance (nephrogenic) = polyuria (PU), sec. polydipsia (PD), neurogenic DI = necrosis of AVP-secreting neurons, nephrogenic DI = rare, often caused by drugs, tubulointerstitial diseases, obstructive nephropathies, urine Na osmolality is low but higher than in serum
ileus, intestinal obstruction	loss of water and Na, with the loss of water being higher than the loss of Na resulting in concentration of Na
primary hyperaldosteronism	water and Na retention due to excessive production of aldosterone in the adrenal cortex = increase in volume of the ECS and rise in serum Na concentration

Potassium (K)

In terms of quantity, potassium is the most important intracellular cation. More than 98% of the potassium in the body is found inside the cells. The serum potassium concentration is regulated within narrow limits. It is very important for membrane potentials; disorders of potassium balance lead to dysfunctions of skeletal muscles, heart and nerve cells. Potassium homeostasis is regulated by oral intake, distribution between the ECS and the ICS and renal elimination.

The regulation by the Na/K-ATPase is an important control mechanism for the movement of potassium between the ECS and the ICS. About 90% of the potassium is excreted via the kidneys, only a small part via the intestines.

Even though the plasma K concentration is only a moderate indicator of total body potassium, it is physiologically important for assessing the transmembrane electrochemical gradient.

Examples of diseases and conditions which can lead to hypokalaemia

renal tubular acidosis (RTA)	RTA is associated with renal loss of K and hypokalaemia, kaliuresis because of increased bicarbonate excretion = cations such as Na, K are excreted more often due to osmotic effect, loss of Na leads to activation of the renin-angiotensin system, since reduction of volume = reduction of K in plasma
ACTH-producing tumours/ primary hyperaldosteronism	tumours produce paraneoplastic ACTH (ectopic production of ACTH) = cortisol and aldosterone production↑ = renal loss of K
feline hypokalaemia	autosomal recessive inheritance, cats show muscle weakness, especially in neck muscles, but leg muscles also affected, serum hypokalaemia, elevated creatinine kinase (CK), Labogen offers genetic testing for example for Burmese, Cornish Rex, Devon Rex, Sphynx and Tonkinese!
primary hyperaldosteronism	increased autonomous aldosterone production due to hyperplasia, adenoma or carcinoma of the adrenal cortex, resulting in Na retention and increased K excretion

Examples of diseases and conditions which can lead to hyperkalaemia

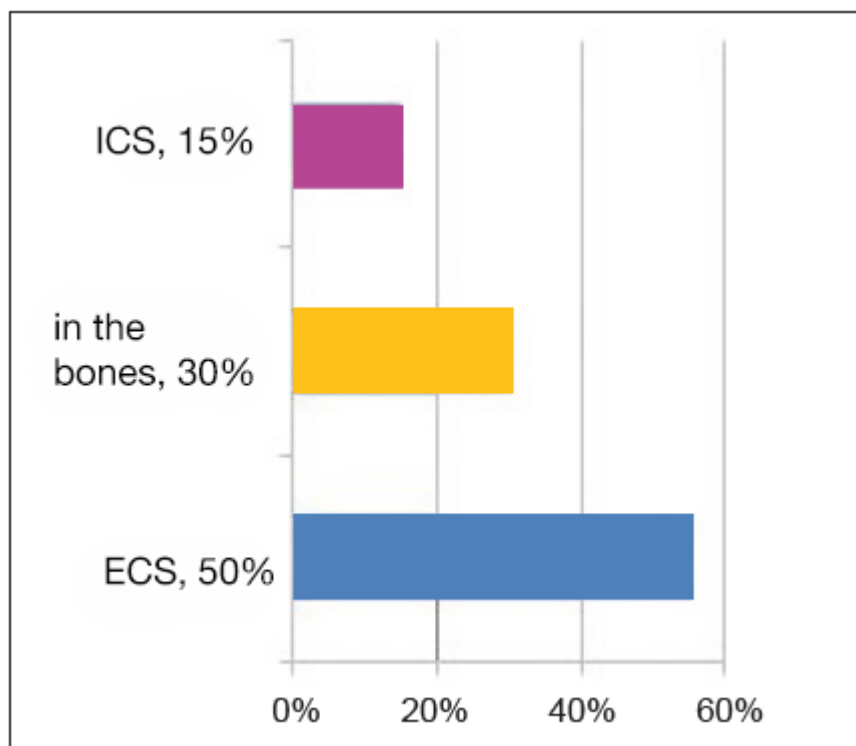
Addison's disease	loss of function of the adrenal cortex = cortisol↓ and mineralocorticoids (aldosterone)↓, ACTH production significantly increased in prim. Addison's disease, hormone deficiency and increased ACTH cause hypotension, acidosis, hyponatraemia, hyperkalaemia
digoxin toxicity	digitalis compounds inhibit effect of the renal tubular Na/K-ATPase = less K is transported into the ICS
non-steroidal anti-inflammatory drugs (NSAIDs)	NSAIDs interfere in prostaglandin metabolism by inhibiting cyclooxygenase. They also act as vasodilators = renin and aldosterone synthesis↑ because of Na deficiency due to drop in blood pressure = hyperkalaemia because of redistribution of fluid between the ECS and the ICS caused by Na/K-ATPase
K-sparing diuretics	for example: Spironolactone, Amiloride Spironolactone: aldosterone antagonist = K secretion is inhibited Amiloride: inhibition of Na reabsorption of the tubular cells = also inhibition of K secretion

Chloride (Cl)

Chloride is one of the most important anions in the ECS. To a large extent, it is bound to sodium and present as common salt (NaCl). As a counterion of Na, it plays an important role in maintaining the water distribution between the ECS and the ICS, hence, in the plasma osmolality.

As a vital electrolyte, more than half of the chloride is found in the ECS (about 55%), approximately one third in the bones (about 30%) and only a small part inside the cells (about 15%).

Chloride fractions



Chloride is mainly taken in through common salt (sodium chloride) in food. It is excreted via the kidneys and regulated by the hormone aldosterone, which causes reabsorption of the anion if there is a lack of chloride.

Example of diseases associated with an increase in serum chloride

metabolic acidosis	increase in Cl, lactate and other anions due to insufficient renal excretion or loss of bicarbonate = in the laboratory Cl is increased, HCO ₃ is decreased
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Examples of diseases associated with a decrease in serum chloride

metabolic alkalosis	increase in HCO ₃ concentration because of conditions that prevent the kidney from excreting or when gastric juice is lost: loss of H and Cl ions, ECFV decreased in gastrointestinal diseases (vomiting), ECFV increased in Cushing's disease, hyperaldosteronism
diuretics	reduction of Na reabsorption = hypochloraemia
hyperaldosteronism, Cushing's disease	Cl low if metabolic alkalosis is present, cannot be corrected by common salt, correlates with extent of hypokalaemia