Fluid volume and electrolyte disorders

MUDr. Rudolf Metelka
Disorders of fluid volume and electrolyte balance

Important major characteristics

• Frequent consequence of derangements of renal, pulmonary and cardiovascular function reflected in changes in body weight and altered laboratory values

• Homeostatic mechanisms may be redundant and cause the "maladaptation" and "circulus vitiosus" in organ's insufficiency
Water = basic fluid medium

TBW = total body water men 60%, women 55% of total body weight

ECV = extracellular volume(water 20%  
  e.g.ISV = interstitial volume 15%  
  e.g.IVV = intravascular volume 5%

ICV = intracellular volume 40-35%

Solids (muscles, bones,..) 40-45%

Fluid (water) volume and its distribution depends on: age, fat, gender (over 75y. 54% men, 46% women) and actual state (temperature, load),

The key is effective arterial blood volume = volume of blood delivered to the volume sensitive organs, predominantly brain and kidney
**Water intake**

- total 2,500-3,000 ml/day
- per os: over 1,000 ml/day, food: 1,000 ml
- metabolic water by nutrients oxidation: 300 – 500 ml

**Water output**

- total 2,000-2,500 ml/d

- Diuresis: 1,000-1,500 ml/d
- Perspiratio insensibilis - skin: 300-600 ml/d
  - lungs: 200-400 ml/d
- temp. 39st: over 1,000 ml, profuse perspiration: 2,000 ml
- Feces: 100-200 ml

- Anuria under 150 ml
- Oliguria under 500 ml/d
- Polyuria = over 2,500 ml/d
<table>
<thead>
<tr>
<th></th>
<th>Plasma</th>
<th>Range</th>
<th>Urine</th>
<th>Range</th>
<th>Intracellular</th>
<th>Range</th>
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<tbody>
<tr>
<td>Sodium</td>
<td>130 – 140 mmol/l</td>
<td>(Ø 137)</td>
<td>120-140 mmol/l</td>
<td></td>
<td>30 – 35 mmol/l</td>
<td></td>
</tr>
<tr>
<td>Chlorines</td>
<td>96 – 106 mmol/l</td>
<td>(Ø 100)</td>
<td></td>
<td></td>
<td>30 mmol/l</td>
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</tr>
<tr>
<td>Potassium</td>
<td>3,8 – 5,1 mmol/l</td>
<td>(Ø 4,45)</td>
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<td></td>
<td>115 – 160 mmol/l</td>
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<tr>
<td>Calcium</td>
<td>Plasma total 2.15-2.65 mmol/l</td>
<td></td>
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<tr>
<td>Glucose</td>
<td>Plasma 3-5mmol/l</td>
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Osmolality

> osmolality = osmotic pressure in 1 kg solvents
> main osmotic activity = sodium, urea, proteins, glucose

Main regulator of osmolality is water retention by antidiuretic hormone (vasopresin = ADH). Production is stimulated by changes in:

1. Serum osmolality (regulation between values 272-282 mmol/kg)
2. 10-20% drop in circulating volume
3. Hypotension

Normal value: Serum 275-295 mmol/kg

Assessment: by osmometry

by calculation: mOsm/kg = 2(sodium) + urea + glucose (all in mmol/l)

Concentration of Urine

Specific weight normal up to 1025

Urine osmolality up 600 – 1200 mmol/d

Osmolality index: osmolality Urine/osmolality Plasma over 1.2

Urea U/P normal over 5

Prerenal cause of renal insf.: Uosm/P-osm over 1.5

Renal cause of renal insf.: Uosm/P-osm under 1.1

Normal value = 1.2

df.dg. hypotonic polyuria (d.insipidus x psychogenic x renal). df.dg. by administration of (desmopresin - Minirin nas.spr, gtt, = ADH)
Effective osmotic pressure is made by gradient between individual osmotic compartments (extracellular(intravascular+interstitial) x intravascular).

Caused by different transmembrane diffusibility of active osmotic matters (sodium, potassium, proteins, glucose, urea). Water diffuses to the space with higher osmotic power.
Hyperosmolar conditions

1. **Simple dehydration** – depletion free water (without solutes): diabetes mell., koma, burns, renal failure, sepsis, intoxication - alcohol, diabetes insipidus, nephrogenic d. insipidus., drowning in salt water

Key: increased effective osmolality in ECV, water transfer ICT → ECT

2. **Acute katabolism**, shock

Key: cumulation osmotic active catabolits in cells, resulting in hyperosmolality and transfer water to cells

ICT ← ECT

3. **Iatrogenic added osmolality**: bad infusion administration (hyperosmotic nutrition bag „all in one“)

Symptoms: encefalopathy with neuronal functional disorders

thirst (absent in seniors), headache .... consciousness, delirium..coma.

Histology: hemorrhagic encefalopathy by Acute elevation Sodium over critical level 150mmol/l (osm nad 310), chronically sodium over 160mmol/l (330osm/kg).
Hypoosmolar conditions

Cause- metabolic reaction on trauma, repletion isotonic waste only by glukose or free water (in hospital or sports iv, p.o.), chron. katabolism, freshwater drownig, inadequate sekretion of vasopresin (SIADH)

Symptoms: weekness, nausea, apathy, headache

Braindedema is rising with danger of herniation of brainstem (CT-edem)

Acutely: sodium under cruitical level 125mmol/l, osmolality under 265 mmol/kg
Chronically sodium under 120/osm pod 250, resp. pod 250mmol/kg

osmolality disturbances should be corrected very slowly

should be made slowly +- 20-30mmol/kg /24hod

e.g. Osm 2-4mmol/kg/hod....Sodium 1-2mmol/l/hod
The solid and orange lines from volume depletion - positive mechanisms activated when volume depletion is either modest or severe, respectively.
The dashed lines - negative feedback mechanisms.
## Integrated volume response to hypovolemic condition (fluid loss)

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<tr>
<th>Response</th>
<th>Systemic hemodynamic changes</th>
<th>External Salt and water balance</th>
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<tbody>
<tr>
<td></td>
<td>• Tachycardia</td>
<td>• Thirst</td>
</tr>
<tr>
<td></td>
<td>• Peripheral resistance</td>
<td>• Renal Na, water retention</td>
</tr>
<tr>
<td></td>
<td>• Venous capacitance</td>
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</table>

<table>
<thead>
<tr>
<th>Onset</th>
<th>Systemic hemodynamic changes</th>
<th>External Salt and water balance</th>
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<tbody>
<tr>
<td></td>
<td>• Minutes</td>
<td>• Hours, days</td>
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<table>
<thead>
<tr>
<th>Major activators</th>
<th>Systemic hemodynamic changes</th>
<th>External Salt and water balance</th>
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<tbody>
<tr>
<td></td>
<td>• Catecholamines</td>
<td>• Catecholamines</td>
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<tr>
<td></td>
<td>• ADH</td>
<td>• Aldosterone</td>
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<tr>
<td></td>
<td>• Angiotensin II</td>
<td>• ADH</td>
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<tr>
<td></td>
<td>• Prostaglandin H2</td>
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<td></td>
<td>• Thromboxane A2</td>
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<th>Major inactivators</th>
<th>Systemic hemodynamic changes</th>
<th>External Salt and water balance</th>
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<tbody>
<tr>
<td></td>
<td>• Prostaglandin E2</td>
<td>• Prostaglandin E2</td>
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<tr>
<td></td>
<td>• Atriopeptin</td>
<td>• Atriopeptin</td>
</tr>
<tr>
<td></td>
<td>• Nitric acid</td>
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</tbody>
</table>
• **absolute** depletion: fluid generally intake-output (perspiration, central-ADH, renal, gastrointestinal, other) /plasma-burns/ whole blood loss by bleeding

• **relative** depletion = redistribution = low effective circulation

eg. -anafylactic reaction = vasodilation
   - end stage of cardiac failure
   - hepatic cirrhosis+ ascites
Extrarenal causes of volume depletion

Hemorrhage

Cutaneous losses
- sweating
- burns

Gastrointestinal losses
- vomiting
- diarrheal disorders
- gastrointestinal fistulas
- tube drainage
Renal causes of volume depletion

Hormonal deficits
*Pituitary diabetes insipidus*

*Aldosterone and kortisol insufficiency*
- Addison disease
- hyporeninemic hypoaldosteronism

Renal deficits
*Specific tubular nephropathies*
- renal tubular acidosis (proximal, distal, gradient limited)
- Bartter’s syndrome
- nephrogenic diabetes insipidus
- diuretic abuse
- postobstructive polyuria

*Excessive filtration of nonelectrolytes* (osmotic diuresis)

*Generalized renal disease*
- chronic renal failure
- interstitial nephritis
Redundant volume repletion reaction

Cause-example: low cardiac contractility-chronic heart failure - low effective circulating volume-activation

**Renin Angiotensin Aldosteron System + vasopressin + other**

Resulted in the same neurohumoral effect as absolute volume depletion:
1: besides further fluid retention with edema, asthma cardiale, ascites pleural effusion

2: reduction of renal perfusion (catecholamine, angiotensin II)
(by centralization of circulation for brain + heart with ischemization of kidney → prerenal azotemia)

3. **metabolic disturbances** (cortisol catabolism for neoglucogenesis
(anaerobic processes instead of oxidative etc.

note: treatment of CHF: inhibition of angiotenzin converting enzyme (ACE-I) or receptor for A - sartans
• Complexity in diagnostics and monitoring is necessary and targeted:
  to watter
  osmotic homeostasis
disease in the background)
Evaluation water and minerals disturbances in praxis

I. clinical (history+somatic) exam

?= is there any problems

key:     ? symptoms of water disturbances?

     ? what about osmolality

II. weight, history and measure water balance

III.lab - confirmation of volume disturbances

- of osmolality status

- mineral and acidobasia disturbances

- others (anemia, endocrinopathy, renal, hepatic, cardiac exam.)
in details:

**History** - state of body organs, nutrition, metabolic diseases (diabetes)
- pharmacological history (diuretics, insulin absence in diabetes
- fluid turnover (fluid income-diuresis, vomiting, diarrhea, fistula, drains.

**Somatic examination** (turgor, dry mucous membrane and tongue, muscle tonus, edema, blood pressure, pulse, venous filling on the neck, central venous pressure, urine colour)

**Laboratory parameters**

daily monitoring: Blood-hematokrit, hemoglobin, sodium, potassium, chlorines, calcium, blood and urine osmolality, glucose, acide-base balance, specific marker of organ’s damage – troponin, TSH,

index urine/plasma (value over 1.5 is for water depletion - dehydration

urine turnover of sodium
Symptoms of volume deficit

– mainly dehydration
(others: bleeding or plasma loss,..)

Thirst, weakness, letargy
Tachycardia Postural giddiness -hypotension Circulatory collaps
Decreased skin turgor Decreased moistness of mucous membranes

Weight loss
filling of neck veins, low central venous pressure

Oliguria 250-500ml/den- Anuria under 150ml/d

Extrarenal disturbance- urine osmolality over 600 mosm/l(spec.w. 1015-1030)
* index osmol. U/P over 1.5

df.dg. Renal disease izo – hypotonic urine osm= under 250mmol/kg, spec.w. 1010-1012, Sodium norm

Hematocrit over 0.5 / or pseudonormal -
Total plasmatic proteins are elevated or pseudonormal df.dg. myeloma..over 100g/l

Sodium plasma over 145mmol/l
Evaluation water and minerals disturbances in praxis

I. clinical (history+somatic) exam  II. weight, water balance  III. lab

A. 1 Normal hydration with sodium in normal range

Cause: state after operation.... the need of substitution of output

Th: isotonic solutions/ alternately glukonic solution

2. Normal hydration with low sodium

Cause: cover the loss of fluid only by G5% or free water, hypothalamic trauma

Result: water transfer ICT ← ECT

Th: usual isotonic solution (Na CL 0.9% = physiological solution

Sodium correction calculation Na+ (mmol) =

\[(\text{targeted Na} - \text{measured Na}) \times F(0.6) \times \text{Total Body Weight (TTW)}\]

F = men 0.6, women 0.55pozn. F.

Chronical state (often nutritional, end stage of cardiological and metabolic states):
very slowly 20-24mmol/l Na/day. often with hypoproteinemia
3. Normal hydration, high sodium

cause: hypertonic infusion therapy (vacs all in one, nasogastric nutrition without water/tea)

result: přesun vody  ICT → ECT

... diuretics and infusion  G5% (glucose is metabolized)
Dehydration

B.1. Dehydration, sodium in normal range = Izotonic dehydration

Balanced cumulative deficit Sodium+water...no transfer ICT x ECT

Cause: Diuretics, ascites puncture, vomiting, diarrhoe, burns, fluid loss to „third spaces“ (ascites, hydrothorax, ileus)

Dg: laboratorně normal osmolality + clinice dry skin and mucous membrane, venous pressure, circulatory colaps with oliguric renal failure,

Th: Izotonic solutions (correction dose and substitution dose - renal, diarrhoe, drainage)

..administered only se 2/3 of expected /calculated volume frequent control is the best marker of restitution.
2. **Dehydration, low sodium** (Hypotonic dehydration)

... output Sodium > water ... přesun vody z ICT ← ECT

Cause: Substitution of ECV loss by drinking or infusion by free water (or glucose) without minerals (sodium), hypokortisolism –m. Addison, diuretics, spirolakton

DG: weight loss, dehydration, low Sodium <130 + other

Th. correction:

1. correction by 2/3 volume by physiologic solutin and lab. + clinical control (in hours

2. correction resulted sodium deficit (it is not necessary – start spontaneous recovery possibility: moderately hypertonic solution mírně hyperton.r.(+10% NACl)

Dose of sodium mmol = (Target sodium - measured sodium) x F(0.6) x TTW
Dehydration, high sodium  (Hypertonic dehydration)

Cause: isolated deficit of free water, or > Sodium output

water transfer ICV ➔ ECV

perspiration, Diabetes mell. with glycosuria, diabetes insipidus, infusion- mannitol, hyperventilation, drinking of seawater, only mineral water by high perfusion...

DG: clinically dehydration, hyperosmolality, high sodium over 145mmol/l, proteins, hematocrit

Th:

1. water deficit by glucose 5%  (glucose is metabolised, resulted free water)

   sodium (liters) = (1 – 137/ measured sodium) ) x TTW x F(0.6)  or

   correction to known weight  or the best monitoring clinical status of hydration
   (CVP, mucous memb. etc.)

2. continued correction according lab. control of mineralogram  (physiolog. solutin
   alone or alternately glucose 5-10%)

3. by persistent high sodium and or heart failure or by seniors with low cardiac
   reserve, in pts with existing or expected renal function impairment
   (ischemia, acute tubular necrose) administer together glucose+ diuretics:

   after correction by 1-2 liters of G5%  we can add low diuretics, monitoring
   diuresis, plasma potassium etc.
A. DEHYDRATACE S HYPERNATRÉMÍ
Tři možné situace ve vztahu dehydratace a zásoby Na

B. KOREKCE DEHYDRATACE S HYPERNATRÉMÍ

I. KROK KOREKCE

DEFICIT VODY (litry) = \( \frac{137}{\text{Na zvlášť}} \) - 2,5 P. CTH

II. KROK KOREKCE

Litr izotonické tekutiny, které je nutno přidat (+) nebo ubrat (-) k deficitu při výpočtu korekce pokles hmotnosti - deficit vody vypočtěný výše

Pozn.: deficit vody hradí 1/2 až 2/3 solným roztokem, pokud po úhradě přetrvává hypernatremie, podávají se diuretika a 5% glukóza
• sodium containing solution for treatment

<table>
<thead>
<tr>
<th>Solution</th>
<th>Volume (ml)</th>
<th>Sodium (mmol)</th>
<th>Chloride (mmol)</th>
<th>Carbonate (mmol)</th>
</tr>
</thead>
<tbody>
<tr>
<td>NaCl 10%</td>
<td>1</td>
<td>1.7</td>
<td></td>
<td></td>
</tr>
<tr>
<td>NaCl 5.8%</td>
<td>1</td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>NaHCO3 4.2%</td>
<td>1</td>
<td>0.5</td>
<td>0.5</td>
<td></td>
</tr>
<tr>
<td>NaHCO3 8.4%</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td></td>
</tr>
</tbody>
</table>

1 g NaCl contains 17.0 mmol Na
1 g NaHCO3 contains 12.0 mmol Na

physiological solution 0.9% = 154 mmol/l NaCl (sodium chloride)
abreviation = F l/l
C. **Hyperhydration, normal sodium** (free hyperhydration)  
... no change of effective osmolality, no transfer ICV x EVC  

DG: edema, rapid (days) weight increase, lab.-normal sodium, osm, organ´s diagn.  
Cause: cardiac or renal failure, infusion overload by isotonic solution  

Th: stop mineral solution, adding the diuretics,  
symptomatic treatment of organ´s failure

**Hyperhydration:** symptoms: edema, anasarc, dyspnea, weight++
Hyperhydration, low sodium  (hypotonic hyperhydration)  
... water transfer z ECT → ICT  

Cause:  
- end stage of heart failure, of ascitic diseases (cirrhosis)  
- water intoxication (overload by G5%), inadequate secretion of vasopresin, oxytocin  

DG: edema, anasarca, weight increase, low sodium under 125mmol/l  

Th: decrease fluid intake (p.o. 500-1000ml), iv, dg. and th of SIADH  
forced diuresis 10-30mg furosemide/h. plus suppl. sodium (slow)
B.3. **Hyperhydration, high sodium** (hypertonic hyperhydration)

...water transfer z ICT → ECT

DG: weight increase, edema, high sodium over 145mmol/l

Calculation: sodium over = F(!0.2) x CTH (sodium measured - minus 137)

Th: edema - forced diuretics - hyperosmolar condition - correction by infusion by G5% together
appropriately versus inappropriately increased ADH concentrations

<table>
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<tr>
<th>Appropriate</th>
<th>ADH secretion</th>
<th>Inappropriate (SIADH)</th>
</tr>
</thead>
<tbody>
<tr>
<td>N ↓</td>
<td>Plasma osmolality ↓ &lt; 280 mosm/kg</td>
<td></td>
</tr>
<tr>
<td>↓</td>
<td>Plasma sodium ↓ &lt; 125 mmol/l</td>
<td></td>
</tr>
<tr>
<td>↑</td>
<td>Urine osmolality ↑ &gt; 500 mosm/kg</td>
<td></td>
</tr>
<tr>
<td>↓</td>
<td>Urine sodium ↑ &gt; 20-40 mmol/l</td>
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</tr>
<tr>
<td>↑</td>
<td>Plasma uric acid ↓</td>
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Potassium is the main intracellular kation, key in production and degradation of macroergic phosphates (ATP-AMP). It takes place in all phosphorylation actions, e.g. energy management.

### Concentration
- **Plasma**: 3.8 – 5.1 mmol/l (Ø 4.45)
- **Intracellular**: 115 – 160 mmol/l

### Elevation:
- **Light**: 5.5 – 6.5
- **Moderate**: 6.6 – 7.5
- **Severe**: Over 7.5

Potassium concentration depends on actual plasma pH!
- pH 7.1: K 5.5 – 6.0
- pH 7.5: K 3.8
- pH 7.7: K 3.5

Decrease pH by 0.1 leads to potassium elevation by 0.3–0.7 mmol/l.
Potassium

plasma potassium concentration is influenced by total body potassium stores,
and
influenced by factors influencing its distribution between extra- and intracellular spaces.

- pH
- catabolism x anabolism
- damaged cell membrane
Potassium

pH and potassium dependency

\[ A: y = 4.8x + 38.75 \]
\[ B: y = 3.08x + 26.4 \]
Major Causes of Hypokalemia

1. Excess renal loss
2. Gastrointestinal losses
3. ECV to ICV shifts (recovery – anabolism, metabolic alkalosis)
4. Inadequate low intake (starvation)
Major Causes of Hypokalemia

- Factors causing increased urinary loss of potassium
  - Increased mineralocorticoids
  - Increased delivery of sodium to collecting duct
  - Increased fluid flow to distal tubule
  - Metabolic and respiratory alkalosis
  - Increased excretion of nonreabsorbable solutes
Major Causes of Hypokalemia

1. Excess renal loss
   - Mineralocorticoid excess
   - Bartter’s syndrome
   - Diuresis: diuretics with a pre- late distal locus
     - Osmotic diuresis
   - Chronic metabolic alkalosis
   - Antibiotics (Carbenicilin, Gentamicin, Amphotericin B)
   - Renal tubular acidosis: Distal, gradient limited
     - Proximal
   - Liddle’s syndrome
   - Acute leukemia
   - Ureterosigmoidostomy
<table>
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<tr>
<th>Diuretic</th>
<th>Primary Effect</th>
<th>Secondary Effect</th>
<th>Complications</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>1. Proximal Diuretics</strong>&lt;br&gt;Acetazolamide&lt;br&gt;Metolazone</td>
<td>↓ Na⁺/H⁺ exchange&lt;br&gt;↓ Na⁺ absorption</td>
<td>↑ H⁺ loss, ↑ HCO₃⁻ loss&lt;br&gt;↑ K⁺ loss, ↑ Cl⁻ loss</td>
<td>Hypokalemic, hyperchloremic acidosis&lt;br&gt;Hypokalemic alkalosis</td>
</tr>
<tr>
<td><strong>2. Loop Diuretics</strong>&lt;br&gt;Furosemide&lt;br&gt;Bumetamide&lt;br&gt;Ethacrynic acid</td>
<td>↓ Na⁺: K⁺: 2Cl⁻ absorption</td>
<td>↑ K⁺ loss, ↑ H⁺ secretion</td>
<td>Hypokalemic alkalosis&lt;br&gt;Hearing deficits</td>
</tr>
<tr>
<td><strong>3. Early Distal Diuretics</strong>&lt;br&gt;Thiazide&lt;br&gt;Metolazone</td>
<td>↓ Na⁺ absorption</td>
<td>↑ K⁺ loss, ↑ H⁺ secretion</td>
<td>Hypokalemic alkalosis&lt;br&gt;Hyperglycemia, hyperuricemia</td>
</tr>
<tr>
<td><strong>4. Late Distal Diuretics</strong>&lt;br&gt;Aldosterone antagonists&lt;br&gt;Spironolactone&lt;br&gt;Nonaldosterone antagonists&lt;br&gt;Triamterene&lt;br&gt;Amiloride</td>
<td>↓ Na⁺ absorption</td>
<td>↑ K⁺ loss, ↑ H⁺ secretion</td>
<td>Hypokalemic alkalosis</td>
</tr>
</tbody>
</table>
Major Causes of Hypokalemia

2. **Gastrointestinal losses**
   - Vomiting
   - Diarrhea, particularly secretory diarrheas

3. **ECV to ICV shifts**
   - Acute alkalosis
   - Hypokalemic periodic paralysis
   - Barium ingestion
   - Insulin therapy
   - Vitamin B12 therapy
   - Thyrotoxicosis (rarely)
Hypokalemia- symptoms

- Clinical Manifestation:
  1. muscular weakness... paralysis (hypokalmic periodical paralysis), obstipation, paralytic ileus hypokalemic nephropathy

  2. spasmophilic state in combination alkalosis and hypocalcemia org hypomagnesemia

  dg: convulsion (hands, legs), Chvostek sign, lab. potassium under 3mmol/l, ECG flattened T waves (non-specific sign)
Correction

calculation of hypocalemia (potassium)

deficit potasium mmol = TTW x 0.6 x (4.4- K measured) x 3

deficit both ECV and ICV!!!

+substitution of gastrointestinal, renal loss/day

The best: 1. administration of 1/3 of calculated dose

2. than according to lab. monitoring the dose specification!!

too quick adjustment... potasium doesn´t go intracellular, danger of hyperkalemie with cardiac disturbances, often in catabolic, dehydrated seniors or in renal impairment

Substitution

of potassium: diuresis x urine potassium mmol/l /day
Correction of hypokalemia (low potassium under 3.5 mmol/l)

- 1 g KCl = 13.5 mmol K
- 1 g KHCO3 = 10.0 mmol
- 1 g Kalc. citr. = 9.0 mmol
- 10 ml 7.5% KCl = 10 mmol K
- 10 ml 10% KCl = 13.5 mmol K
- KH2PO4 13.8% 1 ml = 1 mmol K to 1 mmol P

- Administration rules:
  - speed - max. 10 mmol potassium/h
  - peripheral vein - up to 40 mmol potassium/l, eg 20 ml/500 ml inf.
  - central vein - higher doses possible
  - maximal 150 mmol/d
  - to advance intracellular metabolism - anabolic infusion with glucose
  - better together with magnesium, calcium (spasmophilia, acc. labor.)
  - combination with p.os kalium chlorratum, spirolacton (temporarily
Hyperkalemia - major causes of and symptoms

1: **RENAL** Diminished renal excretion by acute/chronic renal failure

2: **FROM CELLS** - transcellular shifts
   - Crush syndrome (from muscles + renal overload by myoglobin)
   - Catabolism (trauma, inflammation-infection, intoxication)
   - Other -

3. **Bad ADMINISTRATION** of potassium, together ACEi + potassium + potassium saving diuretics (spirolactone)

Clinical manifestation:
- Often oligosymptomatic:
  - Neuromuscular (hyperpolarization on neuromusc. disc) - fasciculation, paresthesia - tingling hands, mouth, tongue, paresis
- **ECG** (over 6.5-7.5 mmol/l): high T waves, alteration of cardiac excitability: bradycardia, AV blocks, asystolia or ventricular fibrillation
Clinical manifestation of hyperkalemia
- alteration of cardiac excitability

- A: Serum K\(^+\) = 6.8 mEq per liter:
  - peaked T waves
  - normal sinus rhythm.

- B, C: Serum K\(^+\) = 8.9 mEq per liter:
  - peaked T waves
  - absent P waves.

- C: Serum K\(^+\) = 8.9 mEq per liter:
  - absent P waves
  - marked prolongation of QRS
  - peaked T waves.

All of the illustrations are from lead V
Acidosis

Cell destruction
(trauma, burns, rhabdomyolysis, hemolysis, tumor lysis)

Hyperkalemic periodic paralysis

Diabetic hyperglycemia
(insulin dependence plus aldosterone lack)

Depolarizing muscle paralysis
(succinylcholine)
pH and potassium dependency

A: $y = 4.8 \times + 38.75$
B: $y = 3.06x + 36.4$

Přerušované jsou značeny referenční mez kalémie a pH
Correction of hyperkalemia

Causal treatment is necessary – rhabdomyolysis – crush or „position trauma“ (often seniors or etylics lying alone- hours- after crash in cold condition at home,outdoor )

stopping inappropriate pharmacology

Th: shift to intracellular space G10%+insulin, rehydration!

renal elimination by diuretics

changing the ion potassium/ calcium (Calcium resonium -2scoop/4hod)

dialysis by critical conditions ecg, heavy acidosis, reansl failure, potassium corrected on acidosis is over 7mmol/l

CAVE monitoring ekg, acidobasia (ABR,eg. pH x potassium -to prevent hypokalemia (arrhythmisas,convulsion) with ABR correction towards to alcalosis
CALCIUM

The main regulator is parathormon increases the plasma level – resorption GIT, releasing from bones, vitamin D3 (calcitriol has additive effect)

Kalcitonin decreases the plasma level aktivní mtb vit.D3

Concentration
- Plasma 2.15-2.65 mmol/l
- Ionized difusible 1.0-1.3 mmol/l (50%)
- Bound on proteins, vázané na bílkoviny, non-difusible (43%)

Reserve: 99% bones

Intake: 20-35 mmol/24 hod.

Output: 25% urine 0.6-5.5 mmol/24 hod., urine balance: dU-Ca = kg x 0.1 mmol/24h

75% feces
Calcium- disturbances

Hypercalcemia: myelom, metastatic processes, bones polytrauma, hyperparathyreosis primary or secondary

Danger of failure of vital function (central nervous s., heart, kidney)—over 3.5 mmol/l.

Symptoms: weakness, letargia, confusion, emotional lability, psychotic behavior, koma anorexia, vomiting, polyuria, thirst (chronically-weight loss).

DG: thinking over hyperkalcemia, plasma calcium, osmolality, plasmatic proteins (paraprotein), B –Jones proteinuria
Hypercalcemia - therapy

- Th:
  - Causal: (myeloma therapy, adenoma operation)

- Symptomatic:
  - **rehydration** through central venous vein!, monitoring CVP, high volume overload
  - + paralel forced **diuretics** (after ECV supply) 10-20mg furosemide /h
  - + i.v. **biphosphonates**
  - (pamidronate 20-60mg/3hod), after that tbl p.o.70mg/d
  - + corticosteroids (usual by meoplasma)
  - + kalcitonin (miacalcic 1-2x 200jed./den nasal spr.
  - + **sensitizer of calcium receptors for PTH**
    (cinakalcet=Mimpara tbl)
  - dialysis in critical conditions (calcuium over 4.5mmol/l or with vital organ impairment)
Hypocalcemia

**Cause:** deficit vit D, m. Crohn, after bowel resection, diarrhoe gastrointestinal loss, pancreatitis necroticans, postoperative (thyroid) hypoparathyreosis in 24-48h after oper.

**Symptoms** usually in case of complex ion disturbances-

- K x P04 x HCO3 alkalosis
- Ca x Mg x H acidosis

**Symptoms:** Paresthesia, hyperreflexia (Chvostek), dysartria, fascikulation x **tetanic convulsion**, emotional lability - excitability

**Lab:** together Sodium x potassium x calcium x magnesium x acidobasia x proteins, ecg ion. hydration and nutritional status
Hypocalcemia-therapy

Th: Calcium gluconicum 10%... 0.25mmol Ca, 10ml obtains 90mg element. calcium
tj. 2.2mmol Ca/10ml... maxima speed od administration is 50mg/min (symptoms limited administration-feeling hot, nauzea, urge to urinate)

1. acute administration
   - 10-20ml 10% Ca gluc up to symptom disappearance,
   - repeated administration up to target Ca = 1.9-2.25mmol/l
   - with monitration ecg, danger of arrhytmias together with digitalis

2. prolonged infusion: up to 0.45mmol/l/kg tj. 2ml/kg TTW. (...14 amp/24h usually G5% 500+10-30ml Ca cluc/6-8h)

3. anticonvulsion th: fenytoin, fenobarbital, benzodiazepin

4. Suplementation and physiological support – kalcitriol (aktive D3), tachystin 3x5-10gtt(sled. Ca!), Rocaltrol 0.5-1-2 g/den
   p.o. calcium 200mg až 500mg CaCO3 /d
Thank for Your attention