Water & electrolyte disturbances

Copotoiu Ruxandra
Total body water TBW

- 60% men, if 70kg TBW = 600ml/l = 42kg
- 50% women
- Age dependent
Shigechyo Izumi born June 1965, lived 120 years + 237 days

- Worked until 105 yrs
- Drank sake
- started to smoke at 70yrs
TBW age dependent
Growing old
TBW compartments

- Intracellular 2/3 total water = 40% total body weight
- Extracellular 1/3 = 20% total body weight
  - Intravascular 5% total body weight
  - Interstitial fluid 15% total body weight
  Transcellular – part of extracellular 1-10l

Plasma + red cells = blood volume = 7% total body weight
Osmolarity = tonicity = nr of particles in solution = 280-295 mOsm/l

Osmolality = mOsm/kg

Measured osmometers
   Depression of the freezing point
   Vaporization

Calculated
   2xNa⁺ + BUN/2.8 + glicemia/18 = 280-295
Urine osmolality

50 – 1200 mOsm/kg
Hel imbalances

- Compartment volume – regulated by aldosterone
- Fluid concentration – regulated by ADH
Tonicity regulation

AVP arginine vasopressin = ADH

Dependency:
- Water intake
- Hormone output
  - Small changes of osmolality 1-2% osmoreceptors ant hypothalamus
  - Mean BP/blood volume baroreceptors – Ao arch, carotid bodies
ADH→↑AQP-2 channels:

- Free water reabsorption
- Antidiuresis
Secretin & oxytocine contribution to water regulation
ICF concentration of solutes # ECF concentration of solutes
70kg adult, masculine, TBW = 42l

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<td>Na+ 10; K 150+; Ma 2+ 40; Fosfates 107; proteins 40; sulfates 43</td>
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<td>Circulating BV = 4.9 (7% x 70kg)</td>
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As today, we are unable to assess intracellular electrolytes at the bedside.
Fluid flux between intravascular compartment and interstitium Q

Plasma proteins/interstitial fluid proteins 16/1 = oncotic pressure difference.

\[ Q = K_f \{ (P_c - P_i) - \sigma (\pi_c - \pi_i) \} \]

Q = net flux
Pc-Pi = hydrostatic pressure capillary/interstitium
\( \pi_c - \pi_i \) = oncotic pressure difference
Kf membrane filtration coefficient ml/min/mmHg
(capillary surface area x capillary hydraulic conductance)
\( \sigma = \text{permeability factor} \) (0 = completely permeable, 1=impermeable)
Fluid flux between intravascular compartment and interstitium $Q$

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(capillary surface area x capillary hydraulic conductance)

$\sigma = \text{permeability factor} (0 = \text{completely permeable}, 1 = \text{impermeable})$

$\sigma$ Explains why in capillary leak (shock, ARDS), colloids cannot maintain the oncotic pressure difference and leak into the interstitium.
Principles of fluid resuscitation

- Intravascular hypovolemia should be replaced with isotonic fluids which tend to distribute in the ECF (3:1) intravascular: interstitium.
- Hipotonic fluids will distribute evenly between all body compartments.

- The endpoint of fluid resuscitation !?
  - Surrogate markers: BP, HR, urine output, parameters of perfusion and cardiac function.
Hyponatremia ser Na⁺<135mE/l

Water>>>/Na
Clinical signs: Cerebral edema

Mild: 130-135 - asymptomatic
Moderate: 125-130: fatigue, malaise, nausea, usteadiness
Severe: 115-120 headache, restlessness, obtundation, lethargy, seizures, coma, brainstem herniation, respiratory arrest, death
Truisms about Na

• Na is a relative fixed solute 136-140
  – K follows…should be closely monitored and replaced or opposed

• Disturbances in serum Na reflect disorders in water balance

• Administration of water to a patient with impaired water excretion can lead to hyponatremia.
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<th>Thirst stimulated $\rightarrow$ ↑water intake</th>
<th>Free water retention</th>
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<td>ADH release $\uparrow$</td>
<td>Concentrated urine</td>
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<td>ADH suppressed $\rightarrow$ Dilute urine</td>
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States of impaired water excretion in the ICU leading to Hyponatremia

**Volume depleted states**
- Volume depletion
- Diuretics

**Normal volume states**
- SIADH
- Pain
- Postoperative state
- Nausea
- Hypothyroidism

**Volume-expanded states**
- Congestive heart failure
- Renal failure
- Cirrhosis
Hypo-osmolar hyponatremia

ECV

Hypovolemic
Simultaneous loss of solute & water →↓ECV→nonosmotoc release of ADH. If water intake →hNa
Cerebral salt-wasting syndrome

Isovolemic
SIADH ser Osm<275, Urine>100mOsm/l
Adrenal insufficiency – nonosmotic ADH release due to cortizol deficiency
Pregnancy - chorionic GDT ↑

Hypervolemic
Congestive heart failure
Cirrhosis
Chronic kidney disease
Dehydration

- Dehydration – ambiguous term, unable to differentiate between simple water loss and Na loss
- A simple water deficit proportionally reduces ECF and ICF
- A NaCl deficit always reduces ECF

Water deficit (l) = 0.6 x G x Na plasmatic / (140 – 1)
Hyperosmolar Hyponatremia

↑↑osmotically active particles in plasma

Water efflux from ICS→ECS

hNa⁺ & Hyperosmolality
Hyperosmolar Hyponatremia

↑↑osmotically active particles in plasma

Water efflux from ICS→ECS

hNa⁺ & Hyperosmolality

Hyperglycemia, mannitol, glycerol, ethanol, sorbitol infusions
Iso-osmolar hNa

- ↑↑ECS by isotonic non-Na containing fluid
- ↑↑ serum proteins & lipids
Hypernatremia >145mmol/l

- Brain shrunked, collapsed → vascular damage + intracerebral or SAH

- Osmotic myelinosis = late, but following rapid hNa correction:
  Lethargy, muscle weakness, nausea, hyperreflexia, seizures, coma
HNa

Diabetes insipidus

• **Central diabetes** = ADH deficiency: TBI, pituitary surgery, brain death, aneurismal SAH, autoimmune

• **Nephrogenic**
  renal resistance to ADH

• **Osmotic diuresis** – excess nonresorbable urinary solute
  – Hyperglycemia, mannitol, ↑↑serum creatinine, hypertonic medication
HNa

- Sea water ingestion
- Use of hypertonic saline for cerebral edema
- Use of NaHCO3 iv
- Overdose of tricyclic antidepressants
K⁺

- 98% IC = 3 000mEq 140-150mmol/l
- EC <2%  60ml ser 3.5-4.5mEq/l
- Cellular access; active Na+, K⁺-ATP ase
- Cellular exit: passive diffusion
hK <3.5

- ↓ K intake
- ↑renal losses
- Redistribution ECS → ICS
HK+>5.0

- Exogenous
- Endogenous
Myoglobinuria syndromes

- Paroxistic rhabdomyolysis: severe muscle pain, exercise collapse
- Malignant hyperthermia
- Rhabdomyolysis due to heroin overdose

(Kumar, BJA 1999;83:496-498)
PPF periodic familial paralysis

Hypokalemia. Uncontrolled diuretic or steroid use, fluid loss (vomiting, diarrhea, etc.), or aldosteronism with hypertension may induce potassium depletion, resulting in weakness or even paralysis, areflexia and/or arrhythmias.

Hyperkalemia. Addison’s disease (primary adrenocortical insufficiency), characterized by bronzing of skin, weakness, weight loss and hypotension, is associated with elevated serum potassium. Manifestations may be mild in early stages, with weakness predominating.
HEL status assessment

Volemia assessment
BP supine + sitting
HR
Mucosal Humidity
Skin fold
Urinary output

Assessment of plasmatic concentration
Na serum
serum osmolality

Assessment of electrolyte composition
Serum electrolites, BUN, glycemia, ABG, pH