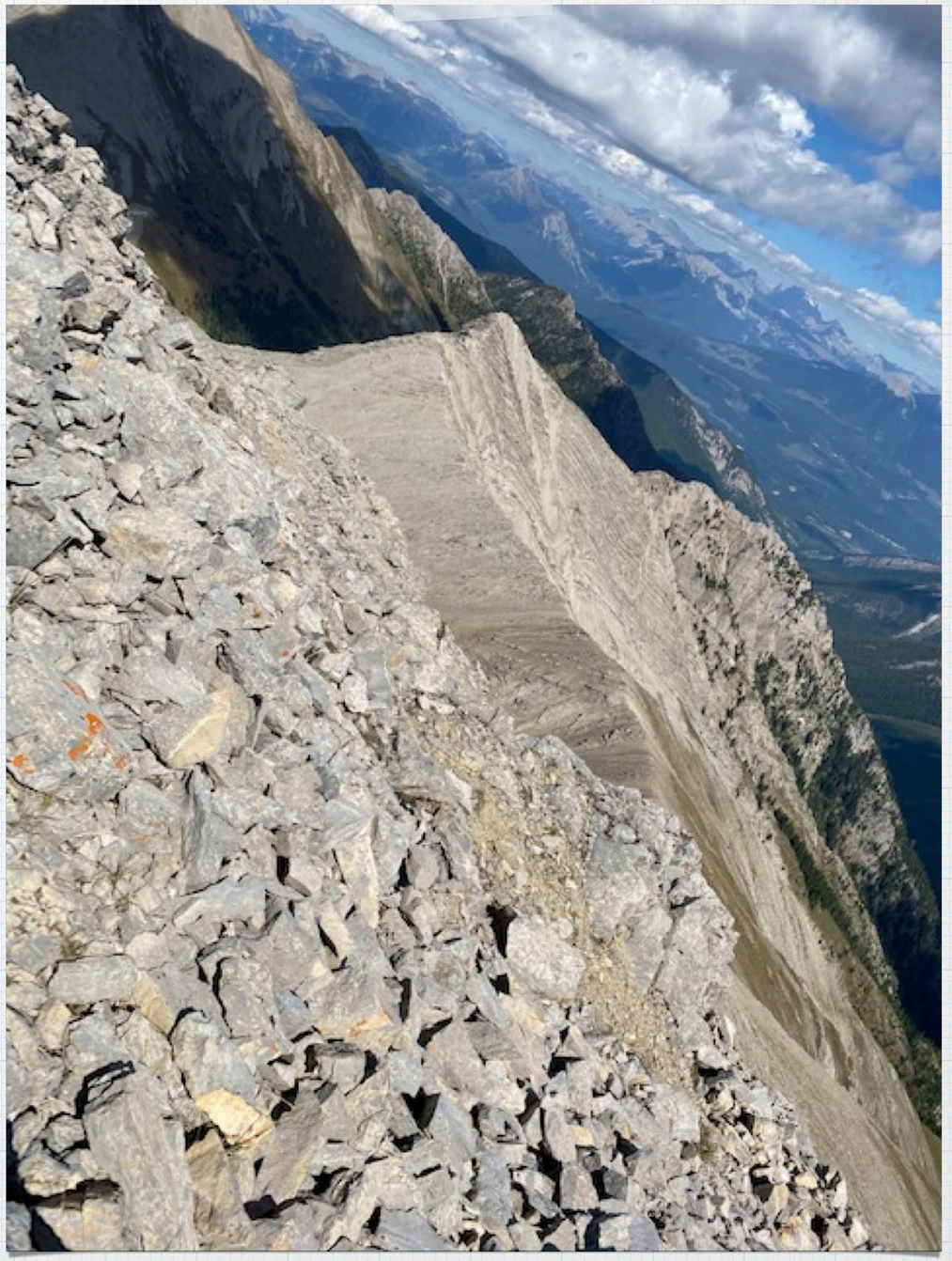
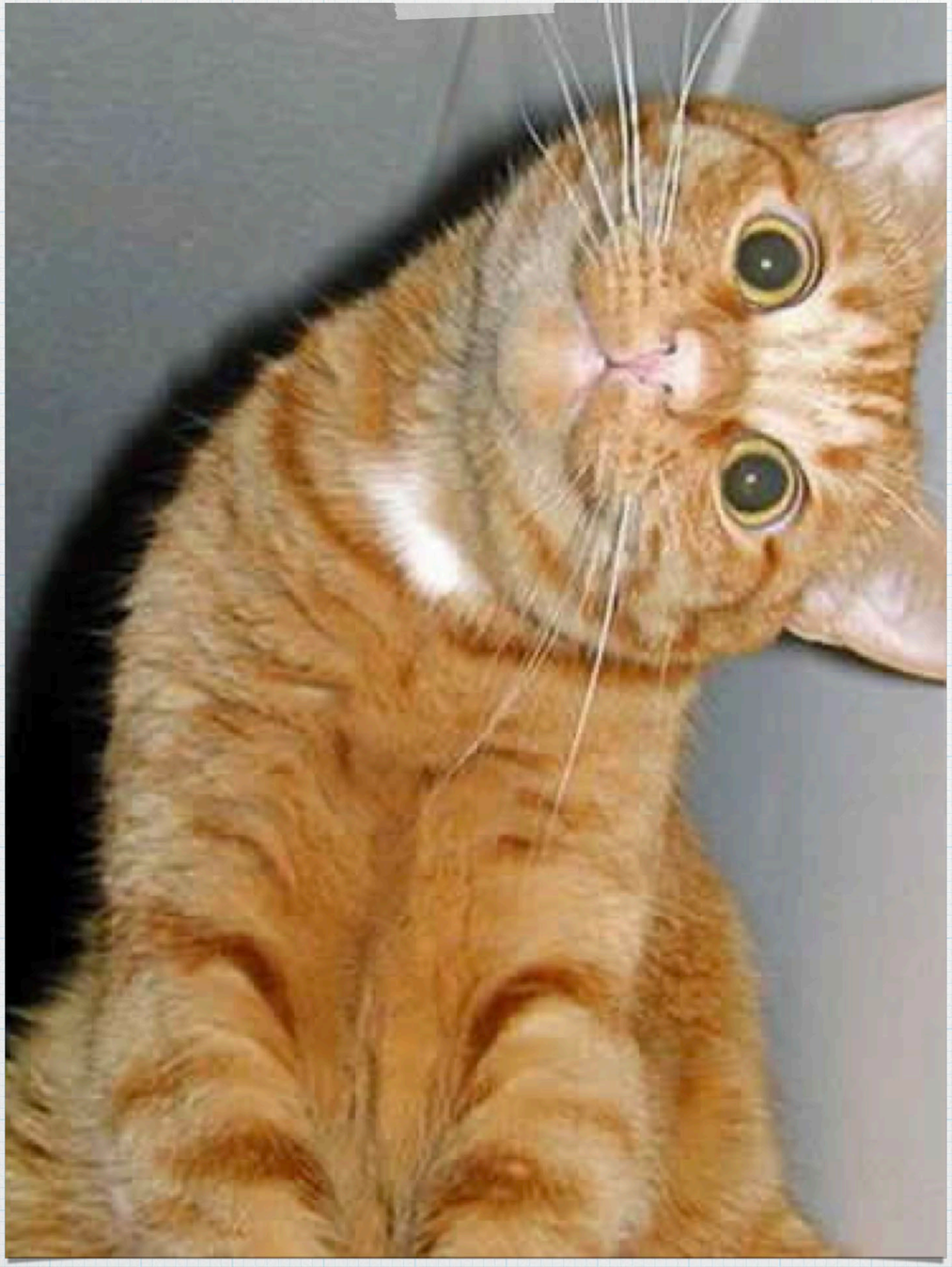
CME rounds

Invermere

2022 October 22



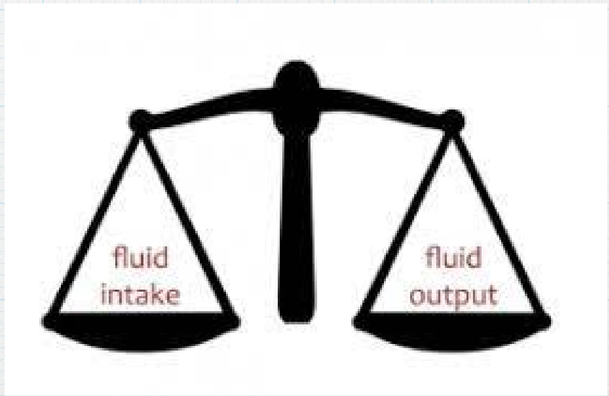


Hypo Natraemia

Made simple

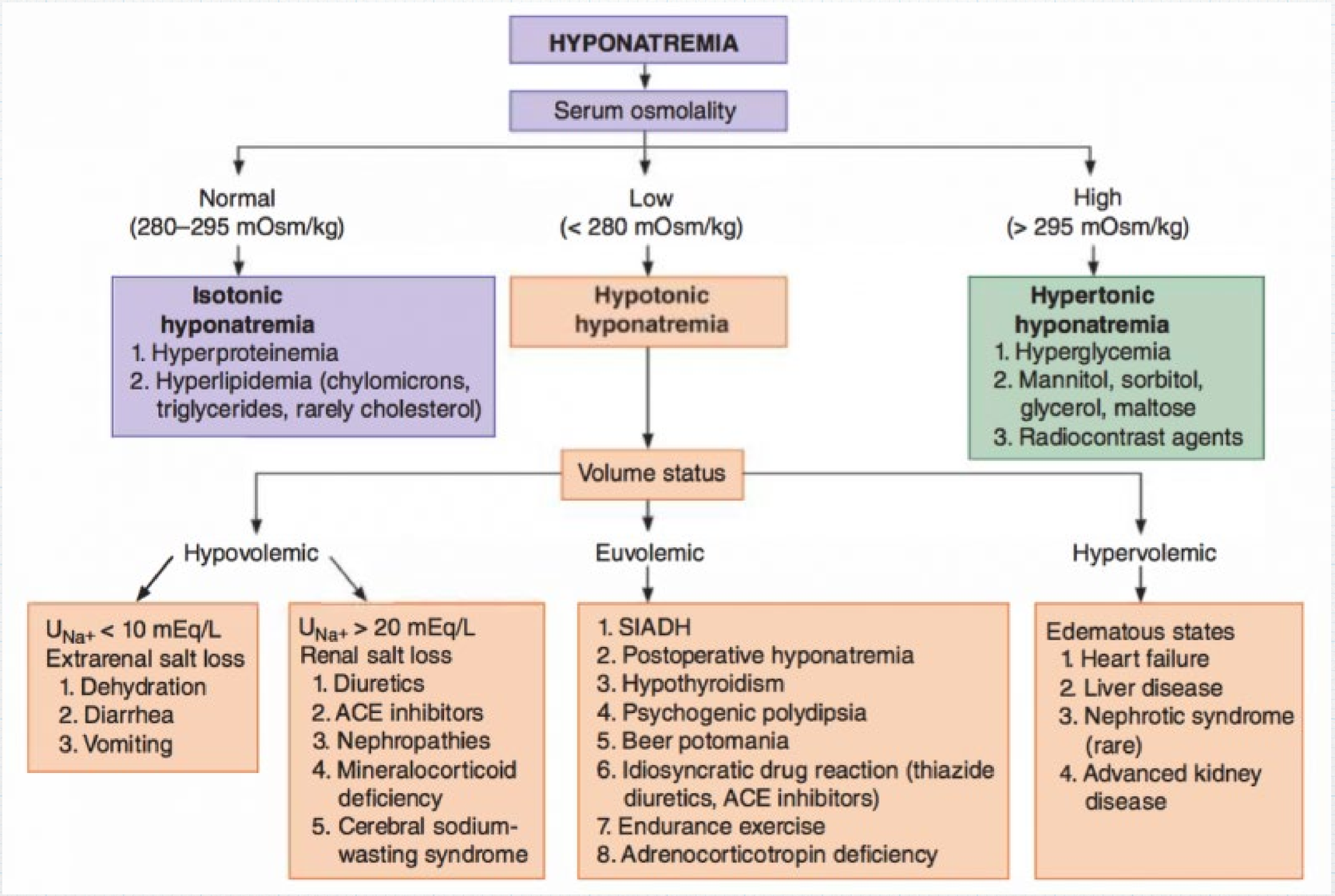
Na problem

- Is not Na problem but a water problem
- Water IN > Water OUT
  - Increased intake
  - Decrease output
  - Combination of 2



Concepts

* Low sodium does not equals low osmolality / tonicity (dissociation)
* Na is needed to produce urine volume, excrete water
* ADH - “hydration hormone”


HYPONATREMIA
Serum osmolality
Normal
(280–295 mOsm/kg)
Low
(< 280 mOsm/kg)
High
(> 295 mOsm/kg)
Isotonic hyponatremia
1. Hyperproteinemia
2. Hyperlipidemia (chylomicrons, triglycerides, rarely cholesterol)
Hypotonic hyponatremia
Hypertonic hyponatremia
1. Hyperglycemia
2. Mannitol, sorbitol, glycerol, maltose
3. Radiocontrast agents
Volume status
Hypovolemic
Euvolemic
Hypervolemic
U_{Na+} < 10 mEq/L
Extrarenal salt loss
1. Dehydration
2. Diarrhea
3. Vomiting
U_{Na+} > 20 mEq/L
Renal salt loss
1. Diuretics
2. ACE inhibitors
3. Nephropathies
4. Mineralocorticoid deficiency
5. Cerebral sodium-wasting syndrome
1. SIADH
2. Postoperative hyponatremia
3. Hypothyroidism
4. Psychogenic polydipsia
5. Beer potomania
6. Idiosyncratic drug reaction (thiazide diuretics, ACE inhibitors)
7. Endurance exercise
8. Adrenocorticotropin deficiency
Edematous states
1. Heart failure
2. Liver disease
3. Nephrotic syndrome (rare)
4. Advanced kidney disease

Osmosis

Movement of water across a membrane

Solute
Ex. Salt

Solvent
Ex. Water

Solute +Solvent Solution
Saltwater

TONICITY

Measure of the effect of osmotic pressure gradient

Hypertonic Solution

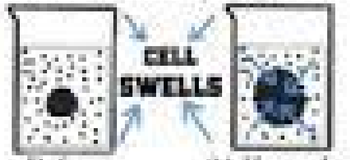
solutes in solution > solutes in cell

Isotonic Solution

solutes in solution = solutes in cell

Hypotonic Solution

solutes in solution < solutes in cell



Hypo Na
Normal serum osmolality

- Pseudohyponatraemia
- Isotonic - lab error, the way Na is measured
- Clinical pearl: ABG will measure true [Na]
- Causes:
  - Increase protein: MM, Waldenstorm, IVIG
  - Hyperlipidemia

Hypo Na
High serum osmolality

- Hyperosmolar / Hypertonic
- Osmolar active substances, solutes that MOVE water:
  - Glucose
  - Mannitol, Sorbitol
  - Contrast Dye
- Water moves OUT of cells, dilutes Na

* NB - concept on Hyperosmolar hypotonic hyponatraemia
* Increased osmolality and hyponatraemia caused by independent processes
* Osmolar non active substances, solute that DOES NOT move water:
  * Ethanol
  * Urea

Hypo Na
Low serum osmolality

* Hypoosmolar/ TRUE
* Water moves INTO cells as a result of hyponatraemia
* ADH dependent/ independent

Urine osmolality

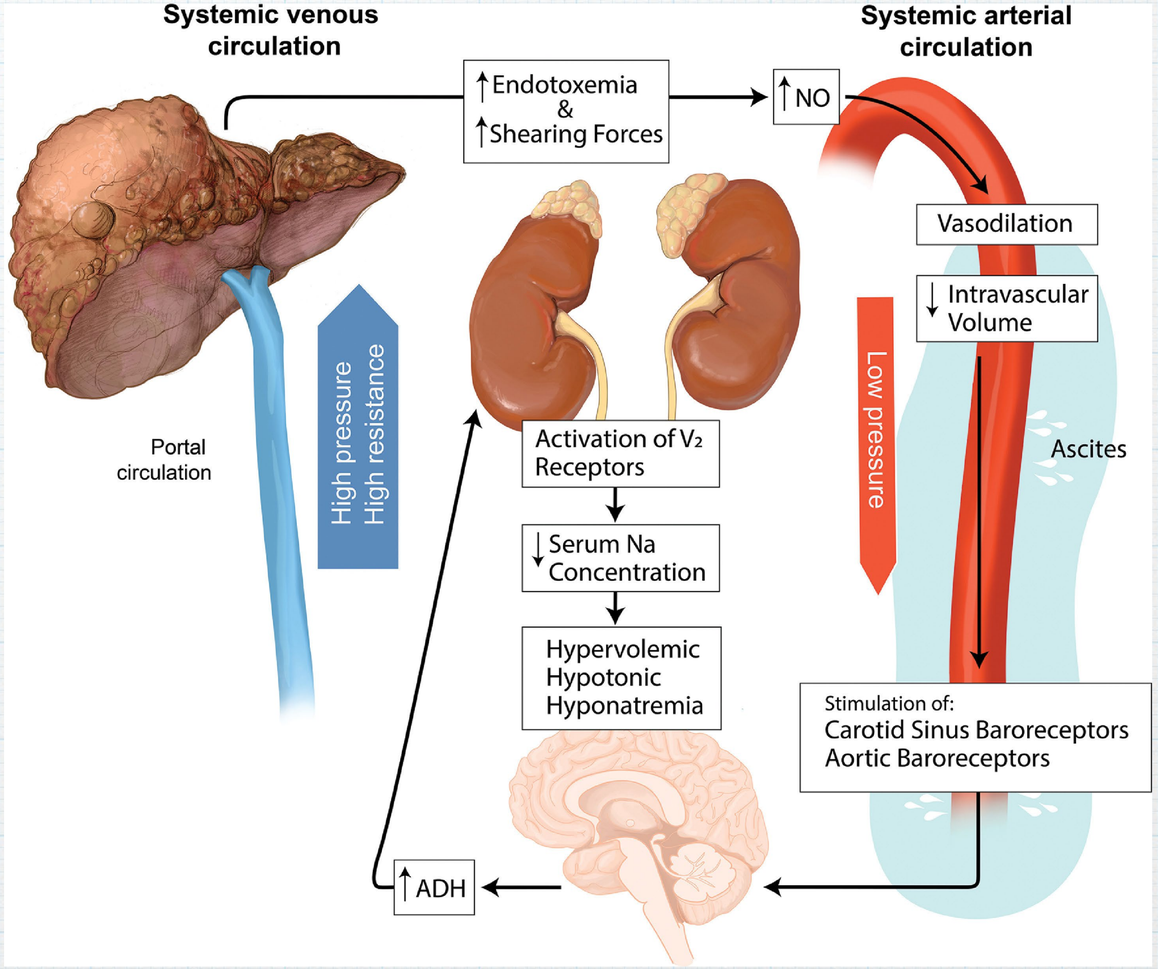
* Lowest - 50 mmol/ L
* Highest 1200 mmol/ L
* Urine volume 0.5 - 14 L on usual diet
* Low solute diet - can’t dilute less than 50 mmol/ L

ADH independent
Urine osmolality LOW

- Tea-toast
- Beer potomania
- Primary polydipsia
- Kidney dysfunction

ADH

- “Hydration” hormone
- Triggers:
  - high serum osmolality
  - Non osmotic release - hypotension/ hypovolemia
- Retains water, drops UO, concentrates urine



ADH dependent
Urine osmolality is HIGH

* Volume status
* Hypovolemic: renal/ extra renal losses
* Euvolemic: SIADH, hypothyroidism, AI
* Hypervolemic: HF, liver cirrhosis, kidney failure

SIADH

- Pulmonary disease - PPV, asthma, PNA, Ca, emphysema
- CNS disease
- Pain, N/V - transient
- Meds: SSRI, opioids, antiSZ

Case #1

* 60 F presented with SZ as a hot stroke, CT head N
* Na 117
* Serum Osmolality - low - TRUE
* Urine Osmolality - low - ADH independent
* By Hx - alcohol abuse, camping on a hot day, increase water intake
* K 2.7

Case #2

* 16 M Hx of N/V, labs - Na 104!
* Serum osmolality low - TRUE
* Urine osmolality high, ADH dependent
* Started on Tx
* Further labs - cortisol of 14!
* Further imaging - paper thin adrenal glands

Case #3

* 70 M with Na admitted after labs showing Na 115
* Serum osmolality - low - True
* Urine osmolality - high, ADH dependent
* Euvolemic, urine Na high - DS of SIADH
* Medication review - no culprits
* By Hx - active long term smoker, COPD
* Imaging with CT head and chest - only emphysema

Treatment
How normal is normal saline?

* What happens if you give 0.9% NaCl to a patient with hyponatraemia?

Hypoosmolar hyponatraemia ADH independent

* 1 L of 0.9% NaCl - solute load - can produce 6 L of urine!
* Quick correction/ overcorrection!

Hypoosmolr hyponatraemia ADH dependent, hypovolemic

* 1 L of 0.9% NaCl will shut off ADH secretion (NO trigger) leading to acute increase in UO
* Quick correction/ overcorrection!

SIADH

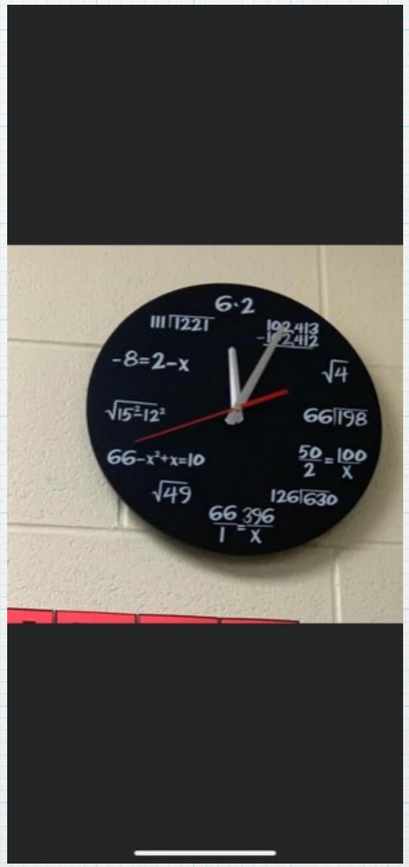
- 1 L of 0.9% NaCl is 99.1% of water that will be left behind due to functioning ADH
- Worsening of hyponatraemia!

Hypoosmolar hyponatraemia ADH dependent hypervolemic

* 1 L of extra fluid - inability to excrete water
* Worsening clinical status!

Treatment

- Acute Tx / subsequent correction of Na
- Formulas do not work!
- Concept of DDAVP clamp
- Frequent labs
- Treat etiology



Acute Tx

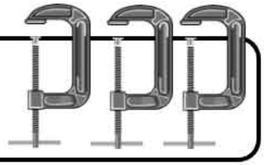
* If CNS symptoms: SZ/ Altered LOC
* 3% NaCl - 100 ml, may repeat
* Will increase Na by 2
* Clinical pearl:
    * NaHCO3 - chloride free hypertonic saline, 7.5%

DDAVP Clamp-Bolus technique

Candidate for DDAVP clamp

- General criteria:
  - For patients at risk for sodium over-correction (rapidly reversible cause of hyponatremia).
  - Especially if high risk of osmotic demyelination (e.g., chronic hyponatremia, cirrhosis, alcoholism, malnutrition, hypokalemia, severe hyponatremia).
- Clamp may be applied pre-emptively (to prevent over-correction), or in a delayed fashion (to treat over-correction).

Placing the clamp on



- Order DDAVP 2 micrograms IV q8hr.
- Restrict free water intake (<1 liter) & avoid hypotonic IV fluids as able.

Plot a course

- Determine the trajectory that you want the patient's sodium to take. The usual trajectory is a gradual increase of 6 mM per day.
- If the patient has *deviated* from an ideal trajectory (e.g., over-corrected), then the goal is to get the patient *back* on the ideal trajectory (osmotic demyelination may take days to manifest, so just because the patient looks OK doesn't mean that they aren't at risk for osmotic demyelination).

Check sodium q4hr or q6hr

Make ongoing, iterative corrections to the patient's sodium level:

- If sodium is below target, bolus with 3% NaCl or hypertonic bicarbonate to increase the sodium to goal.
- If sodium is above target, bolus with free water (e.g., D5W IV) to reduce the sodium to goal.

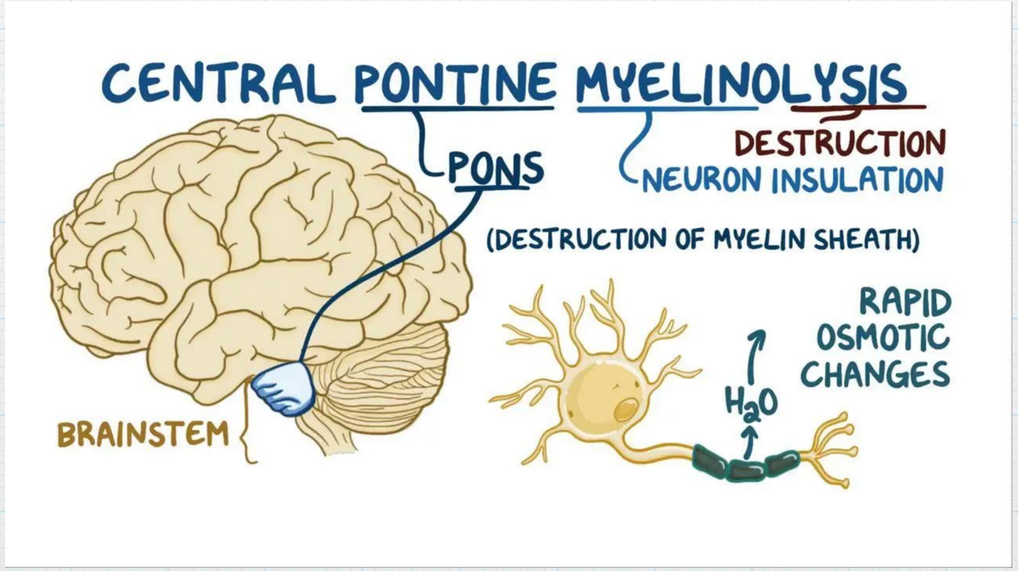
Key components of this strategy:

- Calculate exactly how much 3%, isotonic bicarbonate, or D5W is required to achieve the desired change in sodium.
- Administer the full bolus to correct the sodium *before* rechecking it, in order to generate a tight and closed feedback loop.

-The Internet Book of Critical Care, by @PulmCrit

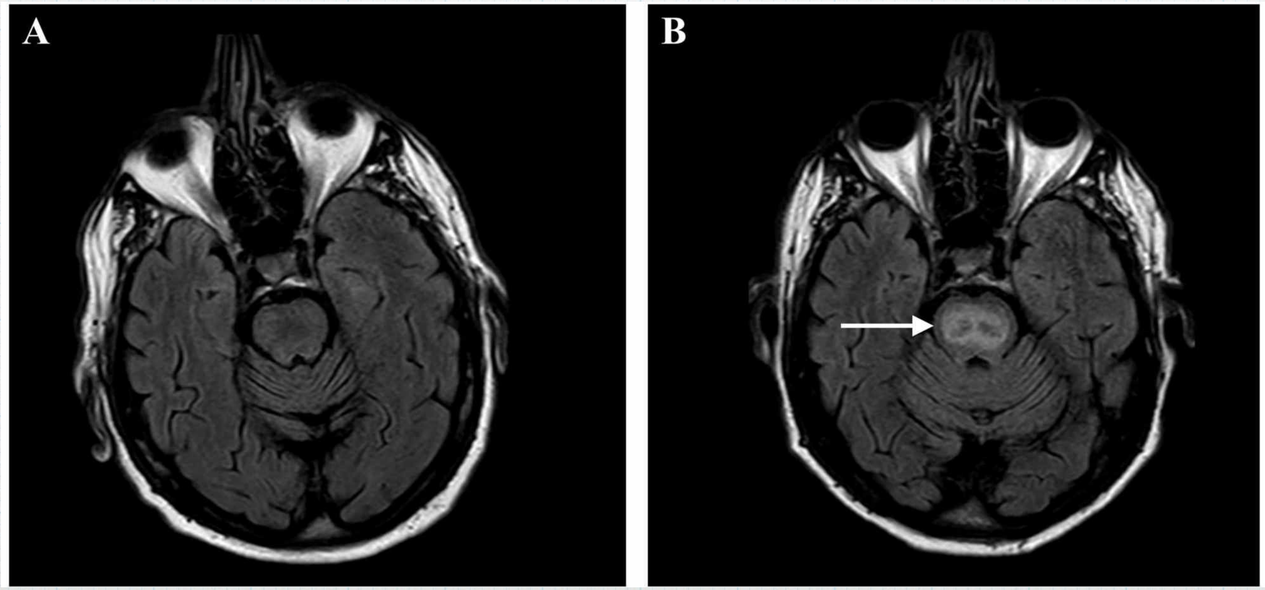
Na correction

* NB risk of osmotic demyelination - shrinking of the brain with quick Na correction
* No more than 6 in first 6 hrs



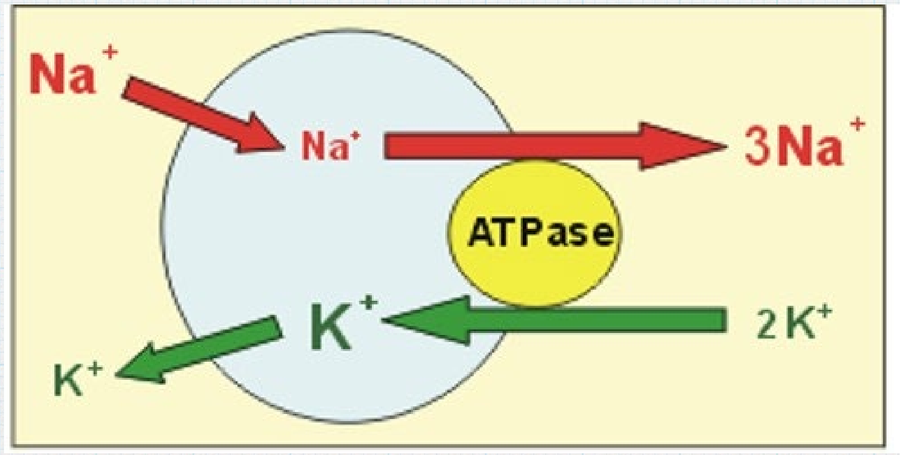
Case #4

- 30 F known alcohol abuse, presentation with AWD
- Na normal
- CT head carried out given delirium - lesion of demyelination



Hypokalaemia

* Correcting K will increase extracellular Na!

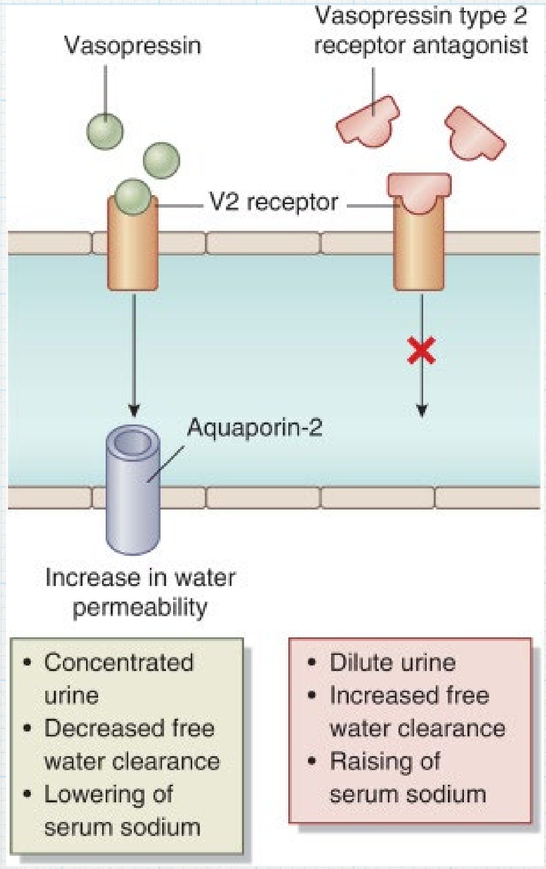


Treatment SIADH

- NB - transient causes
- Medication review
- Fluid restriction: as can't excrete more than set amount of water
- Salt tablets: to increase solute and allow more water excretion

Vaptans

* ADH antagonists
* Liver toxicity
* I don’t use - Nephrology



Case #1, Tx

* Acute Tx with 3% NaCl (Seizure)
* Correct potassium
* Frequent labs
* Advocated patient to eat salt containing foods
* In 48 hrs Na autocorrected close to normal without 0.9% NaCl infusion
* Patient education: diet with higher solutes, stop/ decrease alcohol intake

Case #2, Tx

* DDAVP clamp
* 3% NaCl infusion to correct Na in controlled fashion
* Initiation of hydrocortisone - new DS of AI
* Patient had episode of confusion on steroids, for the concern of osmotic demyelination, CT head done - normal

Case #3, Tx

* No transient causes or culprit medications
* Water restriction
* Avoid 0.9% NaCl infusion!
* Na still low, addition of salt tablets

Management STEPS

* Check serum osmolality: FALSE vs TRUE hyponatraemia
* Other labs: TSH, Cortisol, Cr, Urea, Ethanol
* Acute Tx if CNS symptoms: SZ/ altered LOC
* Urine studies: osmolality, electrolytes: ADH dependent vs not
* Foley IN, monitor UO - key!
* DO NOT empirically give 0.9% NaCl
* Tx as per etiology

Questions?

Thank you!

* Clinical practice guideline on diagnosis and treatment of hyponatraemia, J Spasovski, R Vanholder, B Allolio, European Journal of Endocrinology, March 2014
* Hyponatraemis, review article, H J Adrogue, N E Madias, NEJM, May 2000
* The fluid, electrolytes and acid base companion, S Faubel, J Topf, 1999
* Treating profound hyponatremia: a strategy for controlled correction, R H Sterns, J K Hix, S Silver, Am J Kidney, Oct 2010
* Medication-induced SIADH: distribution and characterization according to medication class, D Shepshelovich, A Schechter, B Calvarysky, T Diker-Cohen, B Rozen-Zvi, A Gaffer-Gvili, BJCP, Aug 2017