HYPONATRAEMIA

The Basics.

- Na < 135 mmol/L
- Results from primary H2O gain &/or Na loss greater than that of H2O.
    - Can also result from alteration in body-water distribution & laboratory error.
- Symptoms are usu. related to *rate of change* in Na-levels.
    - <120 mmol/L more likely assoc. w/ symptoms
- Symptoms include;
    - N&V, anorexia, muscle cramps, confusion lethargy.
    - Severe cases - coma & seizures.

Pathophysiology.

CNS.

- ↓ serum Na creates a concentration-gradient across BBB. Draws H2O into brain.
    - Alters consciousness, agitation, headache, seizures & coma.
    - Slower onset associated w/ more non-specific symptoms.
- Brain susceptible to injury with Na correction.
    - Rapid ↑ in serum Na (ahead of brain solute recovery) results in fluid shift out of CNS cells → ***Central Pontine Myelinolysis.***

CVS.

- In volume-deplete patients, hyponatraemia further ↓s intravascular volume by shifting fluid from ECF to ICF spaces.
- ADH release occurs in all hyponatraemic patients [to preserve intravascular volume by vasoconstriction].

Renal.

- Kidneys response to hyponatraemia is to *produce dilute urine.*
    - Hindered by the presence of ↑ ADH concentrations.
- Urinary Na < 10mmol/L usu. indicates *normal renal handling of sodium.*
    - Urinary Na > 20mmol/L indicates tubular damage or *natriuretic response to hypervolaemia.*

Diagnosis.

First Step - Osmolalities.

- True hyponatraemia = ↓ plasma osmolality.
- Factitious hyponatraemia = normal or ↑ plasma osmolality.

Hypertonic hyponatraemia [Osmol > 295]*;*

- Hyperglycaemia
- Mannitol excess
- Glycerol therapy

Isotonic hyponatraemia [Osmol 275-295]*;*
"pseudohyponatraemia"

- Hyperlipidaemia
- Hyperproteinaemia (eg. myeloma, Waldenstrom)

If HYPOTONIC, proceed to next step.

BOX 123-1 CAUSES OF HYPONATREMIA

Sampling error
Pseudohyponatremia
  Hyperlipidemia
  Hyperproteinemia
Redistributive type
  Hyperglycemia
  Mannitol
Hypovolemic type
  Renal losses
  Gastrointestinal
  Third-space losses
  Excessive sweating
  Addison's disease
Euvolemic type
  SIADH
  Psychogenic polydipsia
Hypervolemic type
  Congestive heart failure
  Hepatic cirrhosis
  Nephrotic syndrome

Second Step - Volume Status.

- Euvolaemia vs Hypovolaemia vs Hypervolaemia
- Consider *predisposing factors.*

Results from intracellular volume expansion w/ consequent derangement of cell functions.

- Disproportionate loss of Na & H2O.
- Can be further assessed by *urinary sodium.*

Third Step - Urinary Sodium.

- > 20mmol/L - usu. renal loss of Na
- < 20mmol/L - usu. extrarenal loss of Na & H2O.

Hypovolaemic hyponatraemia.

- Disproportionate loss of Na & H2O.
- Often inadequate H2O replacement (via oral intake or hypotonic fluids).
- Loss of Na can be renal or extrarenal.

Renal	Extrarenal
Diuretic use	Volume replacement (hypotonic fluids)
Salt-wasting nephropathy (RTA, CRF, interstitial nephritis)	GIT losses (vomiting, diarrhoea, fistula, NGT)
Osmotic diuresis (glucose, mannitol)	Third-space losses (burns, pancreatitis)
Mineralocorticoid deficiency	Sweating (eg. cystic fibrosis)

Hypervolaemic hyponatraemia.

- Total body water excess.
- Impaired ability to excrete a H2O-load in excess of Na-retention.
- 1st group: generalised oedematous states w/out advanced renal insufficiency.
- 2nd group: acute or chronic renal failure

Urinary Na < 20 mmol/L	Urinary Na > 20 mmol/L
Congestive cardiac failure (low-flow to kidneys → stimulates ADH)	Renal failure (inability to excrete free H2O)
Nephrotic syndrome	
Cirrhosis	

Euvolaemic hyponatraemia.

- Combination of normal volume state & hyponatraemia.
    - Not clinically oedematous & nearly normal total-body Na.
- Typically, urinary-Na is > 20mmol/L (or higher) in states of *ADH-excess.*

DDx for Euvolaemic Hyponatraemia
SiADH - *see other table*
Hypothyroidism
Drugs
Water intoxication [psychogenic polydipsia]
Glucocorticoid excess
Porphyria

Diagnostic Criteria for SiADH
Hypotonic hyponatraemia
Clinical euvolaemia
Inappropriate urine concentration [Ur Osm > 100 mmol/kg]
↑ Urinary Na^+ [> 30 mmol/kg]
Normal adrenal, renal, cardiac, hepatic & thyroid function
Correctable w/ H2O-restriction

Causes of SiADH			
CNS	**Pulmonary**	**Tumour**	**Drugs**
Tumour or trauma	Tumour	Lung	Diuretics
CVA, SAH	Tuberculosis	Pancreatic	Cyclophosphamide
Meningoencephalitis or abscess	Pneumonia, abscess or aspergillosis	Thymoma	Exogenous vasopressin
Guillain-Barre	COPD	Ovarian	Vincristine
Multiple sclerosis	Cystic fibrosis	Lymphoma	many many more...

<u>Emergency Management.</u>

- The vast majority of cases will require little urgency in their management.
- If severe (Na < 115 mmol/L) or if symptomatic (coma, seizures, focal neurological findings) then treatment is required...

Rule of 3's (Acute)

- 3mL/kg of 3% Saline over 30mins
- Aim to ↑ 2-6 mmol acutely.
- Avoid correcting by 0.5-1 mmol/hour (Max: 10-12mmol in 24 hours) !!

Rule of 6's (Chronic)

- Six-a-day makes sense for safety. (Do not ↑ by > 6mmol in 1st 24 hrs)
- Six in six hours for severe symptoms and stop !!