Presenting problems in disorders of sodium balance and water balance

DISORDERS OF WATER BALANCE:

Daily water intake can vary from about 500 mL to several litres a day. While a certain amount of water is lost through the stool, sweat and the respiratory tract ('insensible losses', approximately 800 mL/day), and some water is generated by oxidative metabolism ('metabolic water', approximately 400 mL/day), the kidneys are chiefly responsible for adjusting water excretion to maintain constancy of body water content and body fluid osmolality (reference range 280–295 milliosmol/kg).

Presenting problems in disorders of water balance:

Disturbances in body water balance, in the absence of changes in sodium balance, alter plasma sodium concentration and hence plasma osmolality. When extracellular osmolality changes abruptly, water flows rapidly across cell membranes with resultant cell swelling (during hypo-osmolality) or shrinkage (during hyperosmolality).

Cerebral function is very sensitive to such volume changes, particularly brain swelling during hypo-osmolality, which can lead to an increase in intracerebral pressure and reduced cerebral perfusion.

The diagnosis of hypovolaemia is based on characteristic symptoms and signs.

16.4 Clinical features of hypovolaemia and hypervolaemia

	Hypovolaemia	Hypervolaemia
Symptoms	Thirst Dizziness on standing Weakness	Ankle swelling Abdominal swelling Breathlessness
Signs	Low JVP Postural hypotension Tachycardia Dry mouth Reduced skin turgor Reduced urine output Weight loss Confusion, stupor	Peripheral oedema Raised JVP Pulmonary crepitations Pleural effusion Ascites Weight gain Hypertension (sometimes)

(JVP = jugular venous pressure)

Intravenous fluid therapy:

The choice of fluid and the rate of administration depend on the clinical circumstances, as assessed at the bedside and from laboratory data.

Notes...

16.6 Basic daily water and electrolyte requirements

	Requirement per kg	Typical 70 kg adult
Water	35–45 mL/kg	2.5–3.0 L/day
Sodium	1.5–2 mmol/kg	100–140 mmol/day
Potassium	1.0–1.5 mmol/kg	70–100 mmol/day

16.7 Composition of some isotonic intravenous fluids

Fluid	D-glucose	Calories	Na^+ (mmol/L)	Cl^- (mmol/L)	Other (mmol/L)
5% dextrose	50 g	200	0	0	0
Normal (0.9%) saline	0	0	154	154	0
Hartmann's solution	0	0	131	111	K^+ 5 Ca^{2+} 2 $Lactate^-$ 29

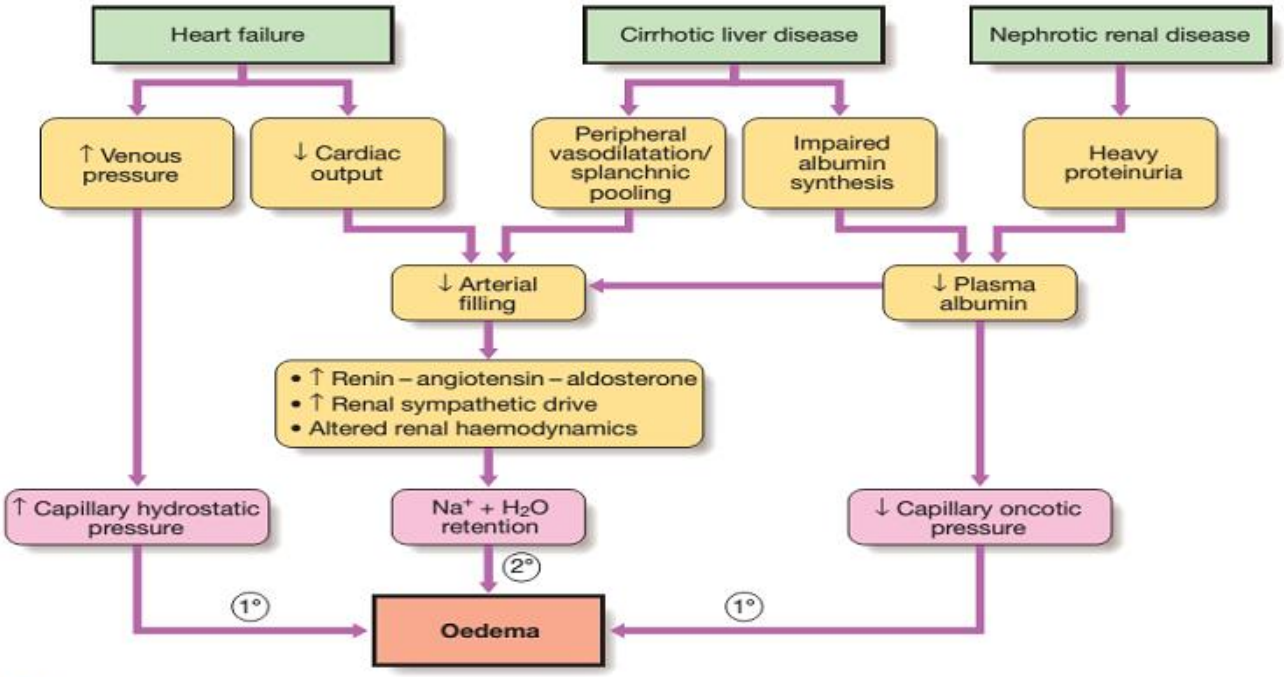

Fig. 16.5 Secondary mechanisms causing sodium excess and oedema in cardiac failure, cirrhosis and nephrotic syndrome. Primary renal retention of Na and water may also contribute to oedema formation when GFR is significantly reduced (see Box 16.10 and p. 478).

Hyponatraemia:

Aetiology and clinical assessment:

Hyponatraemia (plasma Na <135 mmol/L) is a common electrolyte abnormality, which is often asymptomatic but which can also be associated with profound disturbances of cerebral function, manifesting as anorexia, nausea, vomiting, confusion, lethargy, seizures and coma.

Notes...

The likelihood of symptoms occurring is related more to the speed at which electrolyte abnormalities develop rather than their severity. When plasma osmolality falls rapidly, water flows into cerebral cells, which become swollen and ischaemic. However, when hyponatraemia develops gradually, cerebral neurons have time to respond by reducing intracellular osmolality, through excreting potassium and reducing synthesis of intracellular organic osmolytes .

The osmotic gradient favouring water movement into the cells is thus reduced and symptoms are avoided.

16.12 Causes of hyponatraemia

Volume status	Examples
Hypovolaemic (sodium deficit with a relatively smaller water deficit)	Renal sodium losses Diuretic therapy (especially thiazides) Adrenocortical failure Gastrointestinal sodium losses Vomiting Diarrhoea Skin sodium losses Burns
Euvolaemic (water retention alone)	Primary polydipsia Excessive electrolyte-free water infusion SIADH Hypothyroidism
Hypervolaemic (sodium retention with relatively greater water retention)	Congestive cardiac failure Cirrhosis Nephrotic syndrome Chronic renal failure (during free water intake)

(SIADH = syndrome of inappropriate antidiuretic hormone secretion; see Box 16.13).

Investigations:

Plasma and urine electrolytes and osmolality are usually the only tests required to classify the hyponatraemia

Urine Na and osmolality in the differential diagnosis of hyponatraemia*

Urine Na (mmol/L)	Urine osmolality (mmol/kg)	Possible diagnoses
Low (< 30)	Low (< 100)	Primary polydipsia Malnutrition Beer excess
Low	High (> 150)	Salt depletion Hypovolaemia
High (> 40)	Low	Diuretic action (acute phase)
High	High	SIADH Cerebral salt-wasting Adrenal insufficiency

*Urine analysis may give results of indeterminate significance, and in this case the diagnosis depends on a comprehensive clinical assessment.

Notes...

Syndrome of inappropriate antidiuretic hormone secretion (SIADH): causes and diagnosis

Causes

- Tumours
- CNS disorders: stroke, trauma, infection, psychosis, porphyria
- Pulmonary disorders: pneumonia, tuberculosis, obstructive lung disease
- Drugs: anticonvulsants, psychotropics, antidepressants, cytotoxics, oral hypoglycaemic agents, opiates
- Idiopathic

Diagnosis

- Low plasma sodium concentration (typically < 130 mmol/L)
- Low plasma osmolality (< 270 mmol/kg)
- Urine osmolality not minimally low (typically > 150 mmol/kg)
- Urine sodium concentration not minimally low (> 30 mmol/L)
- Low-normal plasma urea, creatinine, uric acid
- Exclusion of other causes of hyponatraemia
- Appropriate clinical context (above)

Management

The treatment of hyponatraemia is critically dependent on its rate of development, severity and underlying cause. If hyponatraemia has developed rapidly (over hours to days), and there are signs of cerebral oedema such as obtundation or convulsions, sodium levels should be restored to normal rapidly by infusion of hypertonic (3%) sodium chloride. A common approach is to give an initial bolus of 100 mL, which may be repeated once or twice over the initial hours of observation, depending on the neurological response and rise in plasma sodium.

On the other hand, rapid correction of hyponatraemia that has developed slowly (over weeks to months) can be hazardous, since brain cells adapt to slowly developing hypo osmolality by reducing the intracellular osmolality, thus maintaining normal cell volume .

Under these conditions, an abrupt increase in extracellular osmolality can lead to water shifting out of neurons, abruptly reducing their volume and causing them to detach from their myelin sheaths.

The resulting '**myelinolysis'** can produce permanent structural and functional damage to mid-brain structures, and is generally fatal.

The rate of correction of the plasma Na concentration in chronic asymptomatic hyponatraemia should not exceed 10 mmol/L/day, and an even slower rate is generally safer.

Notes...

The underlying cause should be treated. For hypovolaemic patients, this involves controlling the source of sodium loss, and administering intravenous saline if clinically warranted. Patients with dilutional hyponatraemia generally respond to fluid restriction in the range of 600–1000 mL/day, accompanied where possible by withdrawal of the precipitating stimulus (such as drugs causing SIADH).

If the response of plasma sodium is inadequate, treatment with demeclocycline (600–900 mg/day) may be of value by enhancing water excretion, through its inhibitory effect on responsiveness to ADH in the collecting duct. An effective alternative for subjects with persistent hyponatraemia due to prolonged SIADH is oral urea therapy (30–45 g/day), which provides a solute load to promote water excretion. Where available, oral vasopressin receptor antagonists such as tolvaptan may be used to block the ADH-mediated component of water retention in a range of hyponatraemic conditions.

Hypervolaemic patients with hyponatraemia need treatment of the underlying condition, accompanied by cautious use of diuretics in conjunction with strict fluid restriction. Potassium-sparing diuretics may be particularly useful in this context where there is significant secondary hyperaldosteronism.

Hypernatraemia:

Aetiology and clinical assessment:

Just as hyponatraemia represents a failure of the mechanisms for diluting the urine during free access to water, so hypernatraemia (plasma Na >148 mmol/L) reflects inadequate concentration of the urine in the face of restricted water intake.

Patients with hypernatraemia generally have reduced cerebral function, either as a primary problem or as a consequence of the hypernatraemia itself, which results in dehydration of neurons and brain shrinkage. In the presence of an intact thirst mechanism and preserved capacity to obtain and ingest water, hypernatraemia may not progress very far. If adequate water is not obtained, dizziness, confusion, weakness and ultimately coma and death can result.

Notes...

16.15 Causes of hypernatraemia

Volume status	Examples
Hypovolaemic (sodium deficit with a relatively greater water deficit)	Renal sodium losses Diuretic therapy (especially osmotic diuretic, or loop diuretic during water restriction) Glycosuria (HONK, p. 814) Gastrointestinal Na losses Colonic diarrhoea Skin sodium losses Excessive sweating
Euvolaemic (water deficit alone)	Diabetes insipidus (central or nephrogenic) (p. 794)
Hypervolaemic (sodium retention with relatively less water retention)	Enteral or parenteral feeding IV or oral salt administration Chronic renal failure (during water restriction)

Management:

Treatment of hypernatraemia depends on both the rate of development and the underlying cause.

If there is reason to think that the condition has developed rapidly, neuronal shrinkage may be acute and relatively rapid correction may be attempted. This can be achieved by infusing an appropriate volume of intravenous fluid (isotonic 5% dextrose or hypotonic 0.45% saline) at an initial rate of 50–70 mL/hour. However, in older, institutionalised patients it is more likely that the disorder has developed slowly, and extreme caution should be exercised in lowering plasma sodium to avoid the risk of cerebral oedema.

Where possible, the underlying cause should also be addressed.

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