



جامعة الجزيرة الخاصة
ALJAZEERA PRIVATE UNIVERSITY

3. Hyponatraemia and Hypernatraemia

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Hyponatraemia

Hyponatraemia is defined as a serum sodium concentration below the reference interval of **133–146 mmol/L**.

It is the electrolyte abnormality most frequently encountered in clinical biochemistry.



Development of hyponatraemia

The serum concentration of sodium is simply a ratio, of sodium (in millimoles) to water (in litres), and hyponatraemia can arise either because of loss of sodium ions or retention of water.

1. *Loss of sodium*

2. *Water retention*



Hyponatraemia

Fluid retention

Sodium deficit

Oedematous

Non-oedematous

↓↓ ECF volume

↑ **Water intake**
e.g. inappropriate
IV saline

↓ **Water excretion**
e.g. CCF,
nephrotic
syndrome

↑ **Water intake**
e.g. compulsive
water drinking

↓ **Water excretion**
e.g. SIAD,
renal
failure

↑ **Loss**
(e.g. from gut,
kidney
or skin)

↓ **Intake**
(extremely rare
cause of sodium
depletion)



1. Loss of sodium. .

Sodium is the main **extracellular cation** and plays a critical role in the **maintenance of blood volume and pressure**, by **osmotically regulating** the passive movement of water.

Thus when significant **sodium depletion** occurs, **water is lost** with it, giving rise to the characteristic clinical signs associated with **ECF compartment depletion**. Primary sodium depletion should *always be actively considered* if only to be excluded; failure to do so can have fatal consequences




1. Loss of sodium.

❖ Sodium depletion effectively occurs only when there is pathological sodium loss, either from the **gastrointestinal tract** or **in urine**.

❖ **Gastrointestinal losses** commonly include those from **vomiting** and **diarrhoea**; in patients with fistulae due to bowel disease, losses may be severe.

❖ **Urinary loss** may result from **mineralocorticoid deficiency** (especially aldosterone) or from drugs that antagonize aldosterone, e.g. spironolactone.



Clinical examination



A postural decrease in blood pressure



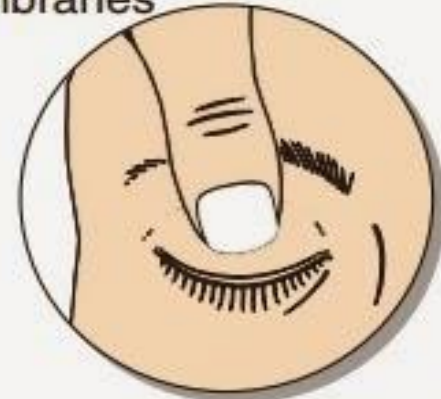
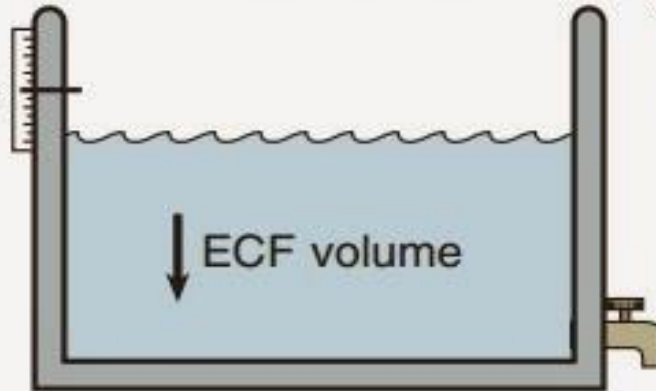
Increased pulse



Dry mucous membranes



Decreased urine output



Soft/sunken eyeballs



Decreased consciousness



Decreased skin turgor

Sodium depletion – a word of

caution-

Not all patients with sodium depletion

are hyponatraemic.

Patients with sodium loss due to an

osmotic diuresis may become

hypernatraemic if **more water than**

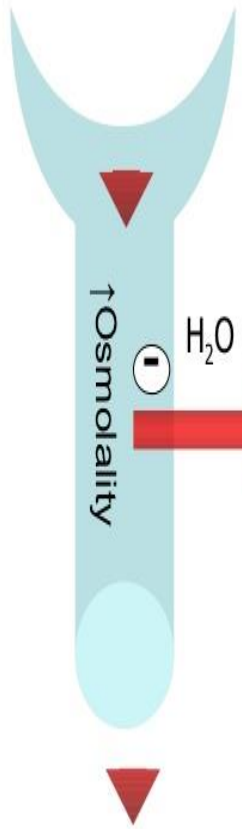
sodium is lost.

Life-threatening sodium depletion can

also be present with a normal serum

sodium concentration.

↑ glucose filtration



Osmotic diuresis

Increased blood glucose



↑ Glomerular filtration of glucose



↑ Osmotic pressure of renal tubular fluid



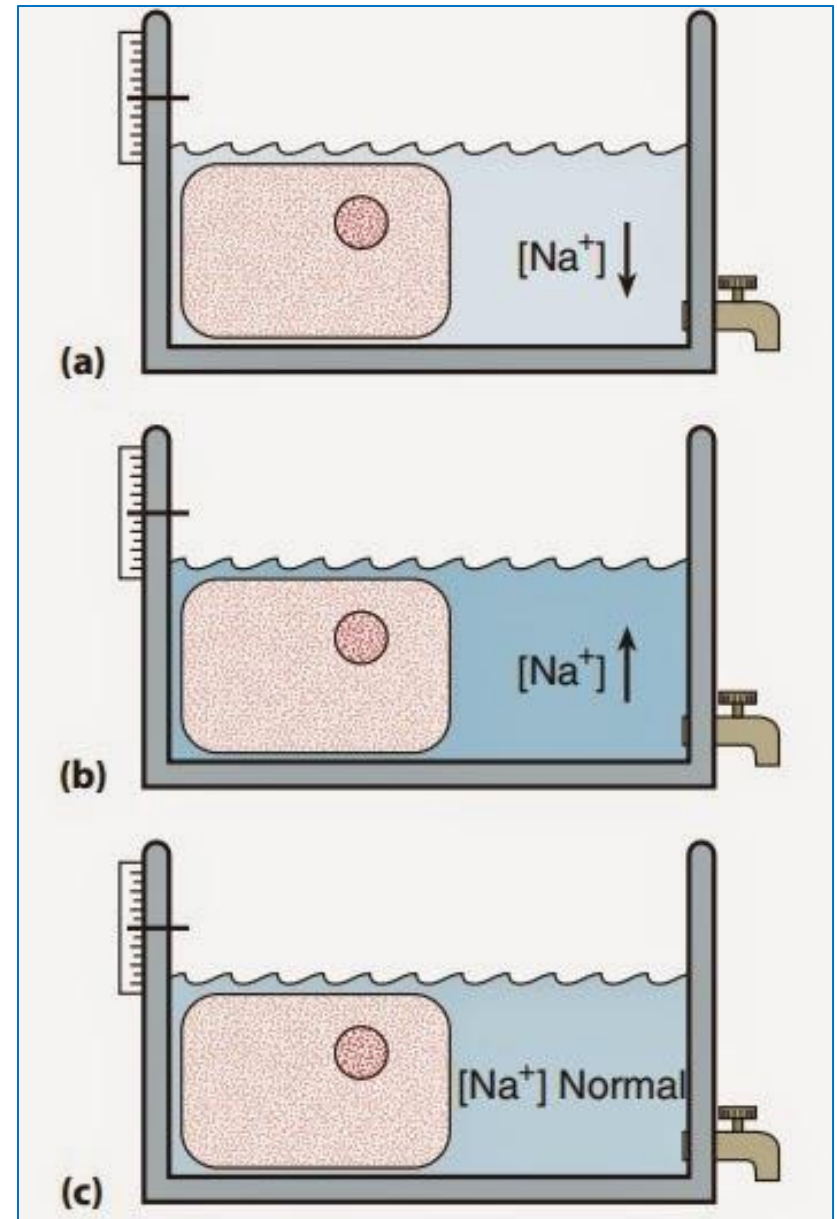
↓ Water reabsorption



Osmotic diuresis

Notice

In short, the **serum sodium** concentration does not of itself provide any information about the presence or severity of **sodium depletion**. The history and clinical examination are much more useful in this regard.



Hyponatraemia

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2. Water retention

Water retention usually results from:

- ❖ and rarely from **increased intake** (compulsive water drinking).
- ❖ **impaired water excretion.**

Most patients who are hyponatraemic due to water retention have the so-called **syndrome of inappropriate antidiuresis (SIAD)**.



Syndrome Of Inappropriate Antidiuresis

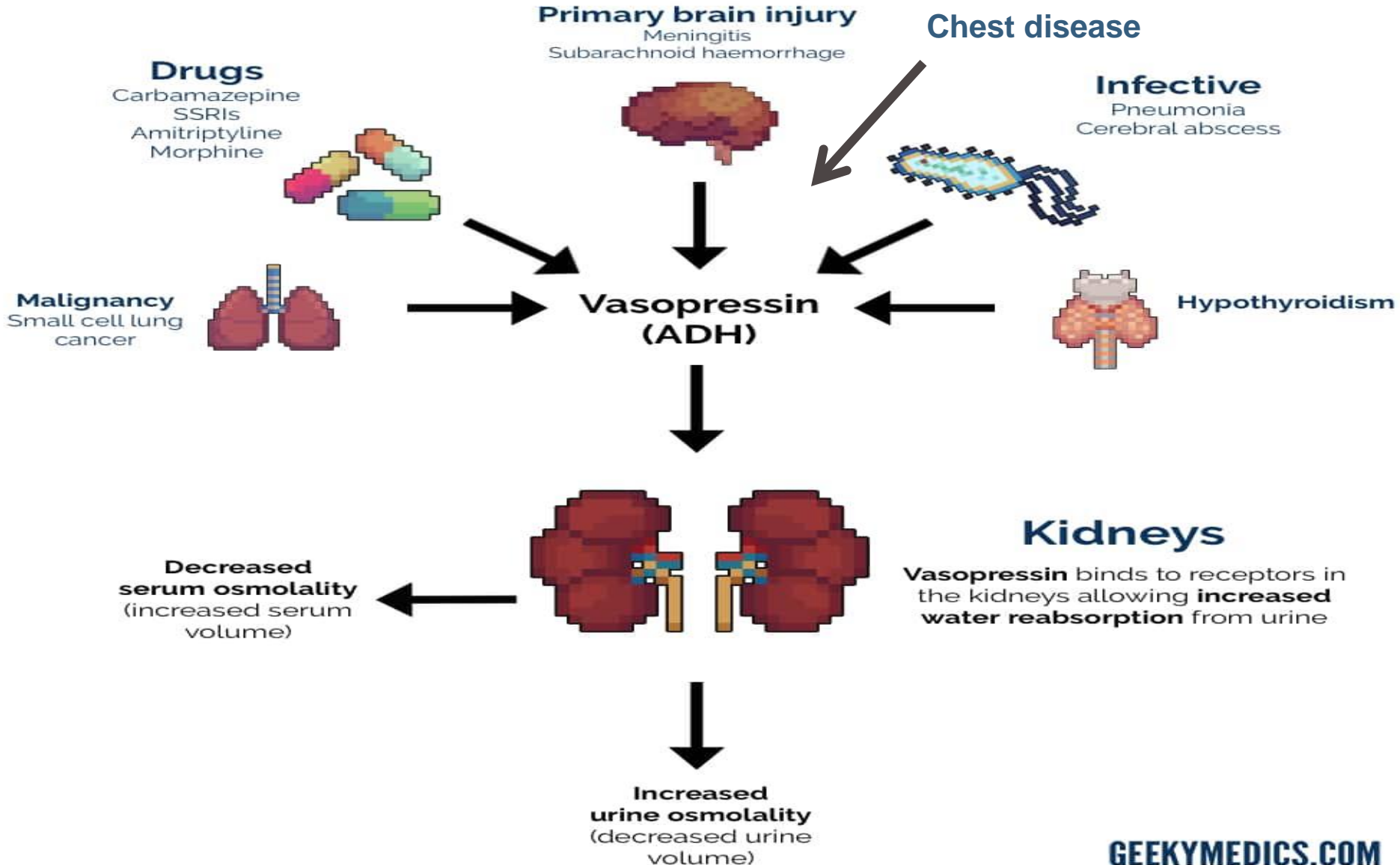
SIAD

- Is a condition in which the body makes too **much antidiuretic hormone (ADH)**.
- This hormone helps the kidneys control the amount of water your body loses through the urine.
- SIADH causes the body to retain too much water.
- Whereas in health the AVP concentration fluctuates between 0 and 5 pmol/L due to changes in osmolality, in SIAD huge (non-osmotic) increases (**up to 500 pmol/L**) can be seen.



Causes of SIADH

And trauma (including surgery)



Oedema

- ❖ **Oedema** is an accumulation of fluid in the interstitial compartment.
- ❖ It is readily elicited by looking for **pitting in the lower extremities** of ambulant patients or in the **sacral area** of recumbent patients.
- ❖ It arises from a **reduced effective circulating blood volume**.
- ❖ Due either to **heart failure** or

Hypoalbuminaemia.

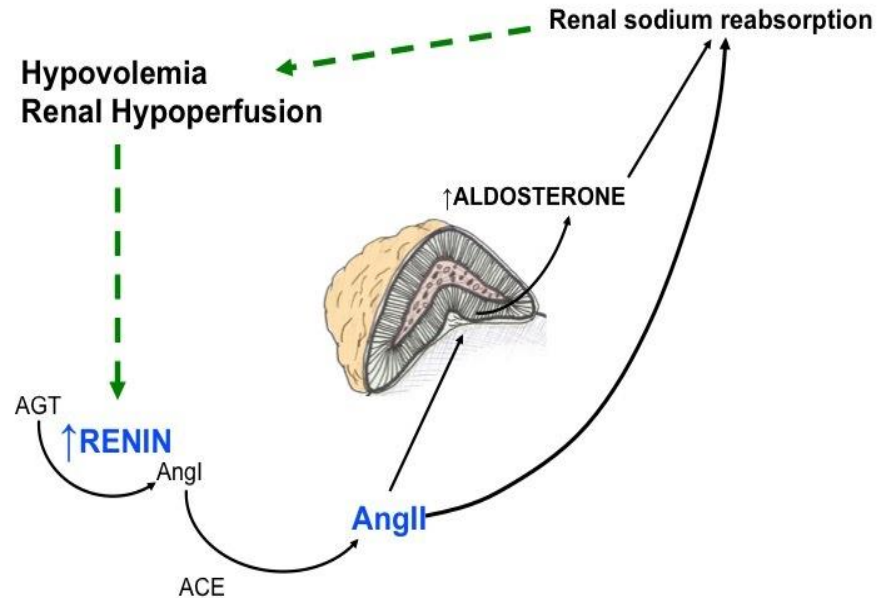


Oedema

❖ The response to this is
secondary hyperaldosteronism.

Aldosterone causes sodium (and
water) retention, thus
expanding the ECF volume.

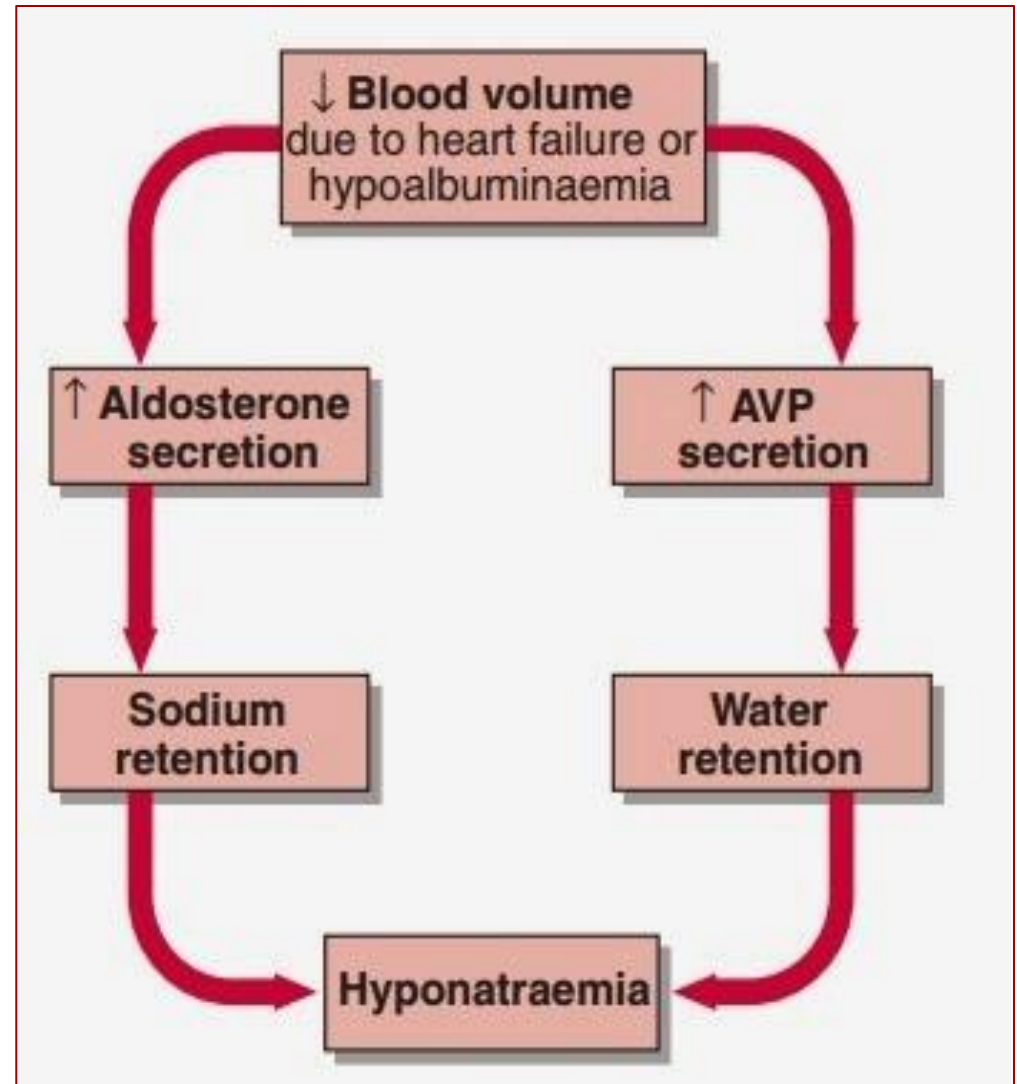
Figure 1



Secondary hyperaldosteronism



Patients with **oedema** become **hyponatraemic** despite sodium retention because the effective hypovolaemia also stimulates AVP secretion, resulting in additional water retention



Severity

In assessing the risk of serious morbidity or mortality in the patient with hyponatraemia, several pieces of information should be used:

1. The presence of **signs or symptoms** attributable to hyponatraemia
2. **Evidence of sodium depletion**
3. the **serum sodium concentration**
4. **How quickly the sodium concentration has fallen** from

Normal to its current level.



Treatment

- ❖ Hypovolaemic patients are **sodium depleted and should be given sodium.**
- ❖ Normovolaemic patients are likely to be retaining water and should be **fluid restricted.**
- ❖ Oedematous patients have an excess of both total body sodium and water; they should be given a **diuretic** to induce natriuresis, and be fluid restricted.
- ❖ More aggressive treatment (usually requiring **hypertonic saline**) may be indicated if symptoms attributable to hyponatraemia are present, or the sodium concentration is **less than 110 mmol/L.**

Hypernatraemia

Hypernatraemia is an increase in the serum sodium concentration above the reference interval of **133–146 mmol/L**.

Hypernatraemia develops either because of:

➤ **water loss**

➤ **or sodium gain.**



Water loss

Pure water loss may arise from :

- 1. decreased intake*
2. or **excessive loss.**



Water loss

1. *decreased intake*

- ❖ Severe hypernatraemia due to *Pure water loss* may arise from *decreased* intake or excessive loss is most often seen in **elderly patients**.
- ❖ Less commonly there is failure of AVP secretion or action.

This is called **diabetes insipidus**



Water loss

- ❖ Less commonly there is **failure of AVP** secretion or action, resulting in water loss and hypernatraemia. This is called **diabetes insipidus**; it is described as central if it results from failure of AVP secretion,
- ❖ *Water and sodium loss can result in hypernatraemia* if the water loss exceeds the sodium loss. This can happen in **osmotic diuresis**, as seen in the patient with poorly controlled **diabetes mellitus**, or due to **excessive sweating** or **adiarrhoe**, especially in children. However, loss of body fluids because of vomiting or diarrhoea usually results in *hyponatraemia*



Sodium gain

- ❖ Hyponatraemia due to sodium gain (often referred to generically as 'salt poisoning') is much less common than water loss. It can occur in several clinical contexts, each very different.
- ❖ The pathophysiological parallel to the administration of sodium is the rare condition of **primary hyperaldosteronism** (**Conn's syndrome**).
- ❖ Similar findings may be made in the patient with **Cushing's syndrome**, where there is excess cortisol production. Cortisol has weak mineralocorticoid activity. However, in both these conditions the serum sodium concentration rarely rises above 150 mmol/L.

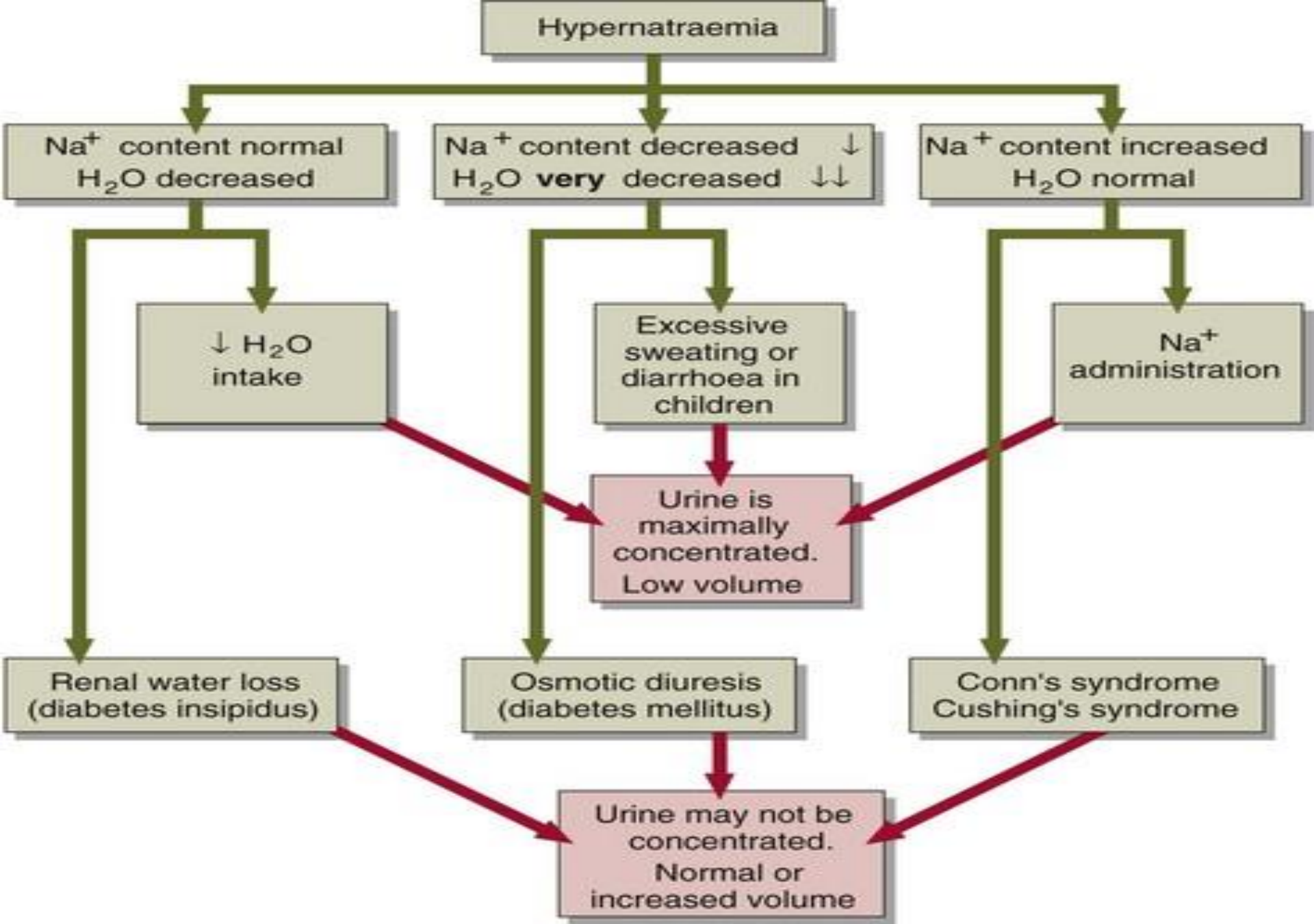


Causes of Sodium gain

'Salt poisoning

- ❖ Firstly, sodium bicarbonate is sometimes given to correct life threatening acidosis.
- ❖ Secondly, near-drowning in salt water may result in the ingestion of significant amounts of brine.
- ❖ Thirdly, infants are susceptible to hypernatraemia if given high-sodium feeds either accidentally or on purpose.

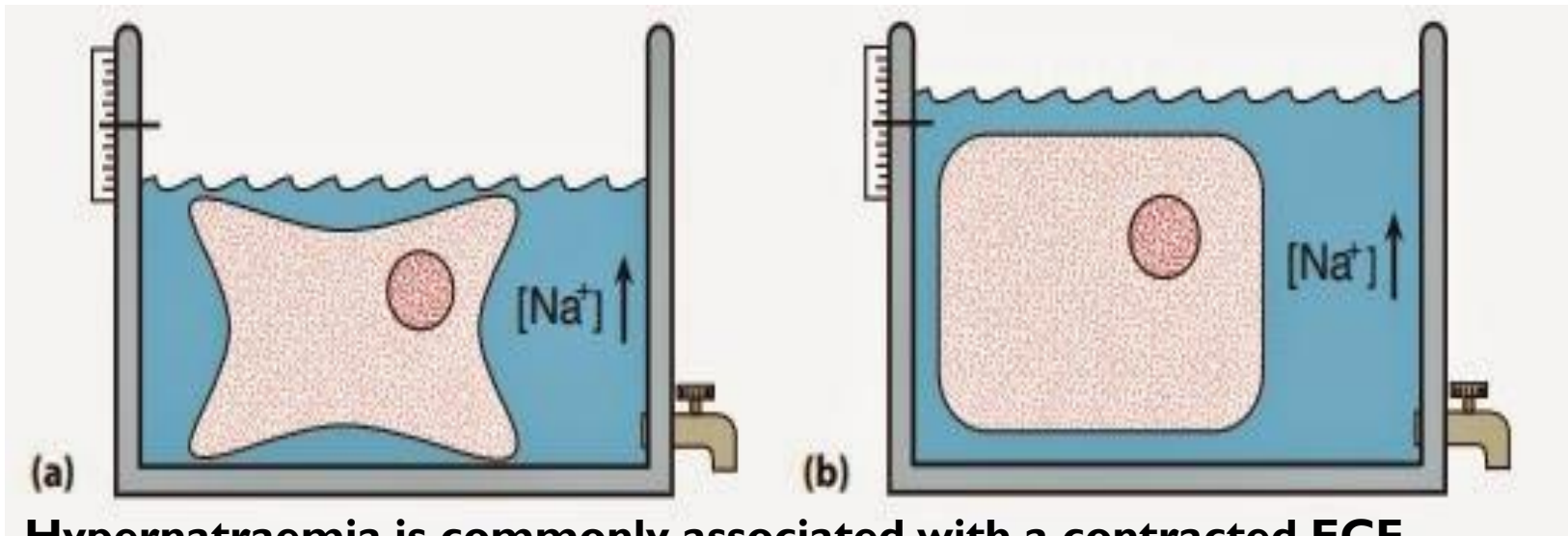




▶ The causes of hypernatraemia

Clinical features

1. Hyponatraemia may be associated with **a decreased, normal or expanded ECF volume**



Hyponatraemia is commonly associated with a contracted ECF volume, and less commonly with an expanded compartment.

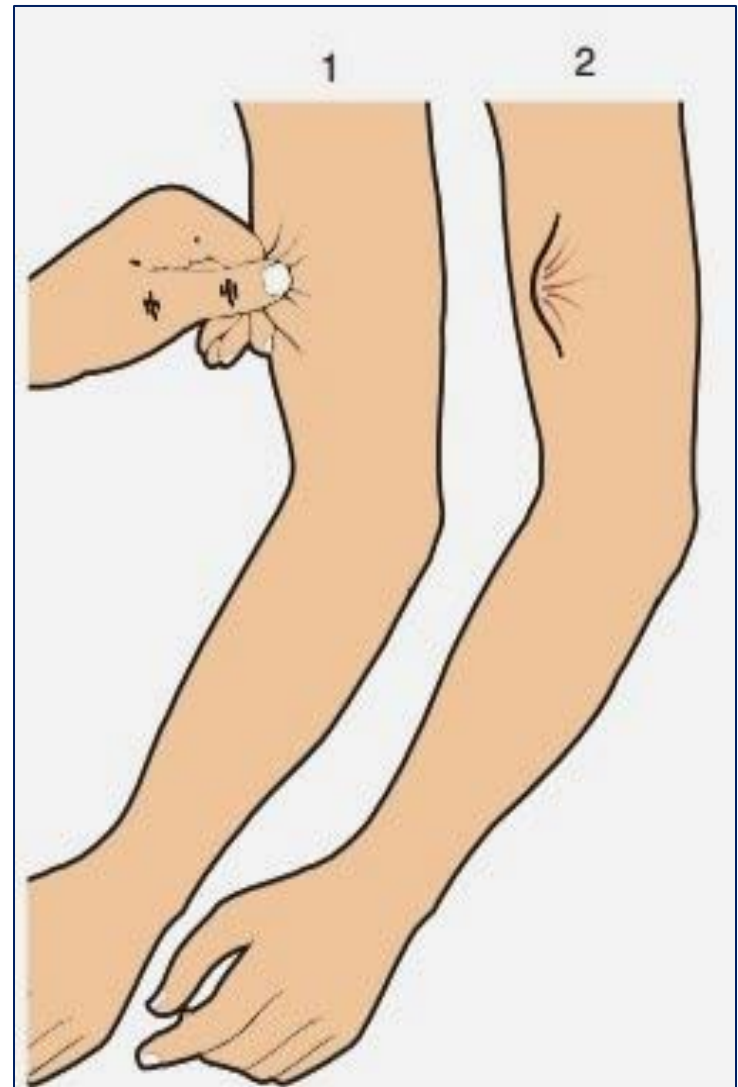
(a) Volumes of ECF and ICF are reduced.

(b) ECF volume is shown here to be slightly expanded; ICF volume is normal.

2. With mild hypernatraemia (**sodium** $<150\text{mmol/L}$), if the patient has obvious clinical features of **dehydration**.

it is likely that the **ECF volume is reduced** and that one is

dealing with **loss of both water and sodium.**



Decreased skin turgor.

This sign is frequently unreliable in the elderly, who have reduced skin elasticity. In the young it is a sign of severe dehydration with fluid loss from the ECF.

3. With more **severe hypernatraemia** (**sodium 150 to 170 mmol/L**), pure water loss is likely if the clinical signs of **dehydration are mild in relation to the severity** of the hypernatraemia.

This is because **pure water** loss is distributed evenly throughout all body compartments (ECF and ICF). (The sodium content of the ECF is unchanged in pure water loss.).



4. With gross hypernatraemia (**sodium >180 mmol/L**), one should suspect **salt poisoning** if there is little or no clinical evidence of dehydration;. **Salt gain** may present with clinical evidence of overload, such as **raised jugular venous pressure** or **pulmonary oedema**.



Treatment

- ❖ Patients with hypernatraemia due to pure water loss should be given **water**; this may be given orally, or intravenously as 5% dextrose.
- ❖ If there is clinical evidence of dehydration indicating probable loss of sodium as well, **sodium should also be administered**. Salt poisoning is a difficult clinical problem to manage.
- ❖ The sodium overload can be treated with **diuretics** and the natriuresis replaced with water.
- ❖ Caution must be exercised with the use of **intravenous dextrose** in salt-poisoned patients – they are volume-expanded already and susceptible to pulmonary oedema.