Hyponatraemia- Principles, Investigation and Management

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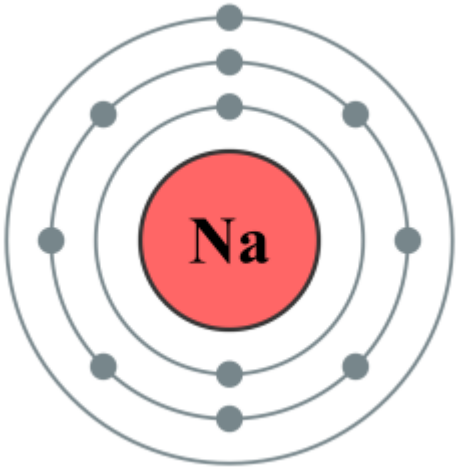
Biochemistry

Contents



- Background
- Investigation
- Classification
- Normal Osmolality
- General management and SIADH
- Cases

Background

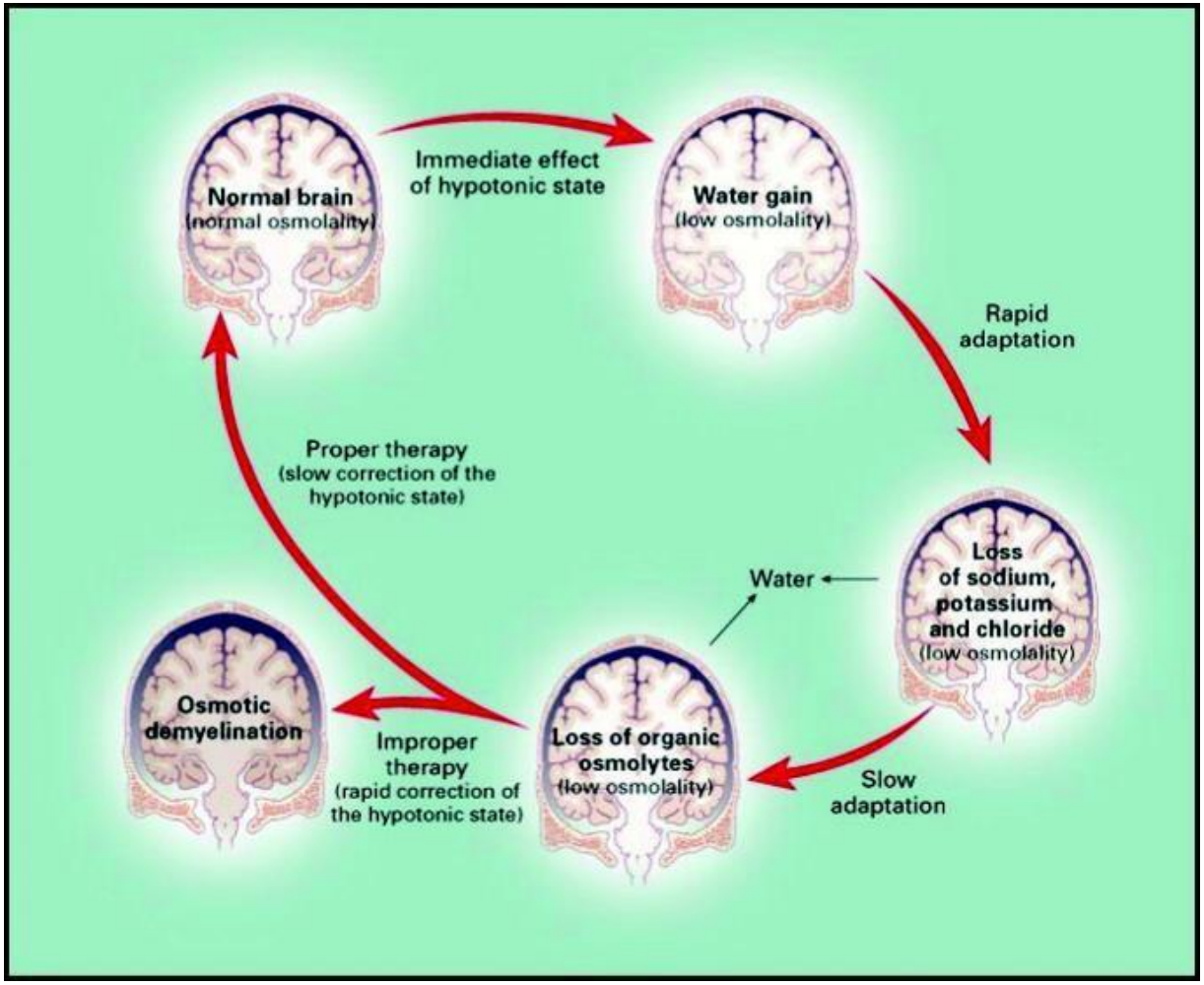
- Relatively common:
  - Estimated 15-30% prevalence in all acute admissions
  - Prevalence 2.48% in community with 0.97% incidence in 1985

- Associated with gait disturbance, cognitive disturbance, osteoporosis and mortality
  - (Not causative)

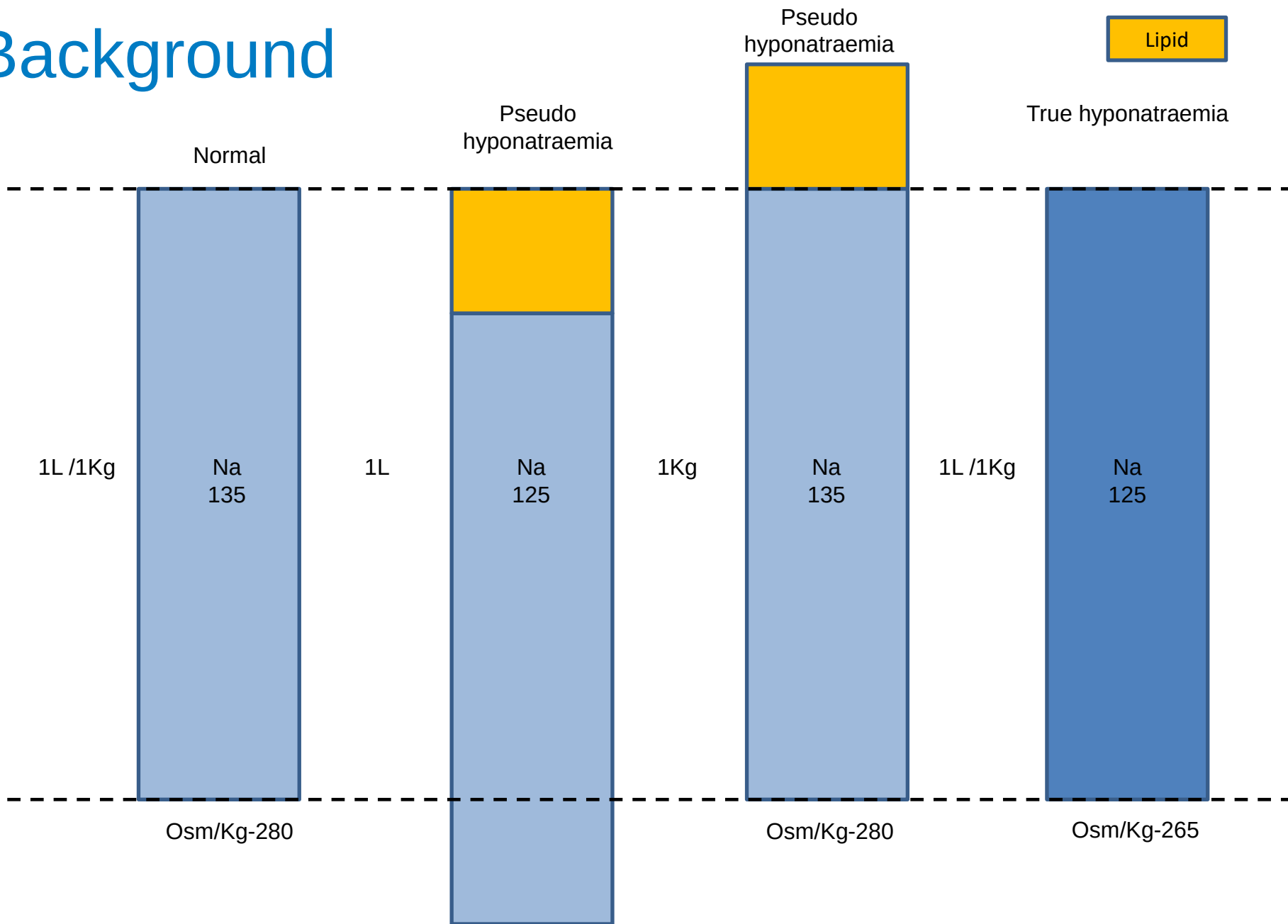
Background

- Hyponatraemia: Serum sodium of less than 135mmol/L
  - Mild: 130-135
  - Moderate:125-129
  - Severe: <125
- Acute is <48 hours, Chronic is >48 hours.
- Reflects serum osmolality
- Hyponatraemia causes cell overhydration

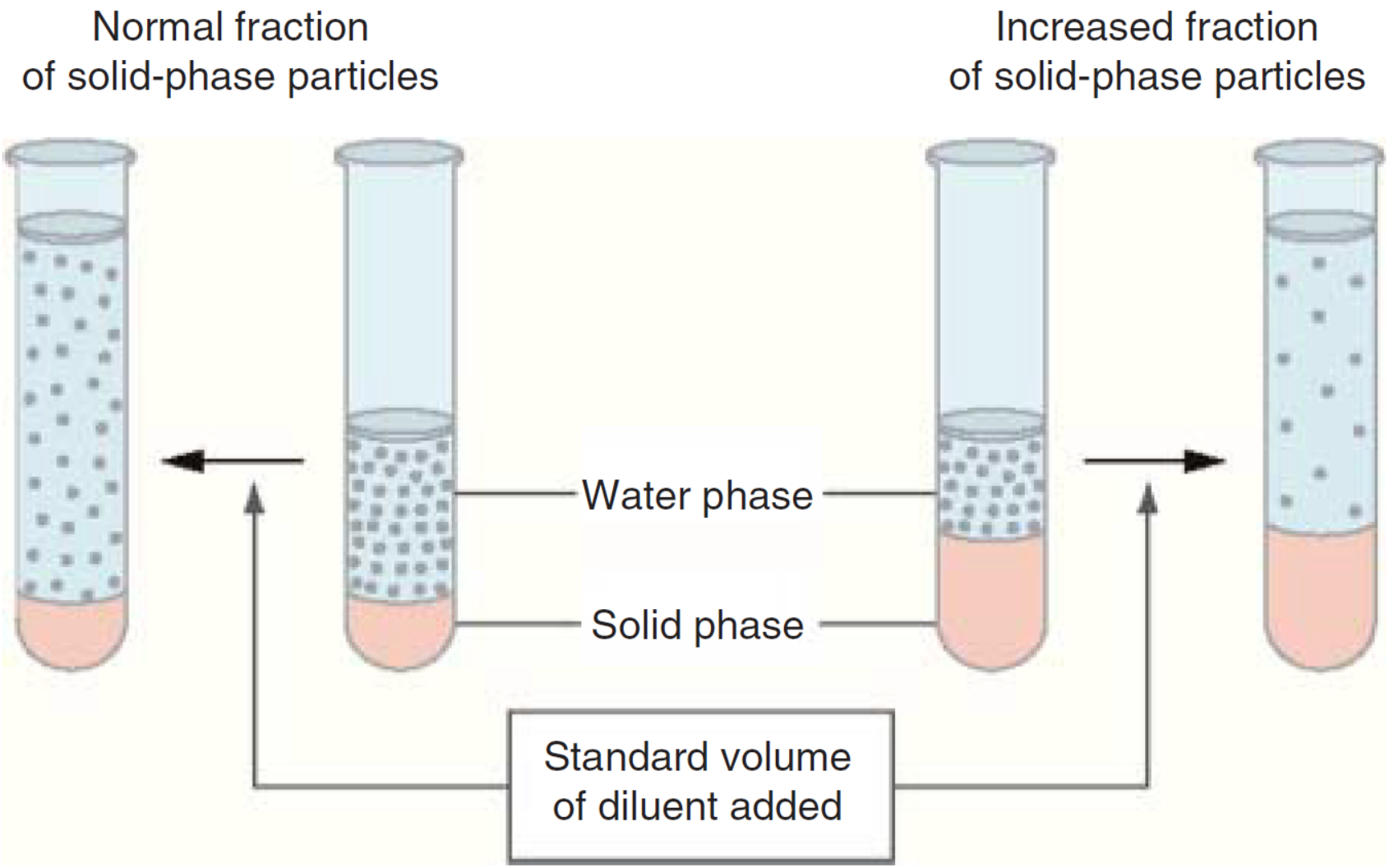
Background adaptation



Background


Pseudo hyponatraemia
Lipid
Normal
Pseudo hyponatraemia
True hyponatraemia
1L /1Kg
Na 135
1L
Na 125
1Kg
Na 135
1L /1Kg
Na 125
Osm/Kg-280
Osm/Kg-280
Osm/Kg-265

Background


Normal fraction
of solid-phase particles
Increased fraction
of solid-phase particles
Water phase
Solid phase
Standard volume
of diluent added

Background - Symptoms

- Mild:
  - Asymptomatic
  - Headache, anorexia, nausea, lethargy
- Moderate:
  - Confusion, muscle cramps, weakness, confusion, ataxia
- Severe:
  - Drowsiness, coma, vomiting, brainstem herniation, seizures
- Rapid correction:
  - Cerebral pontine demyelinolysis

Assessment

Examination

- Capillary refill
- Tissue turgor
- Radial pulse- ?tachycardia
- Mucous membranes
- JVP
- Heart sound- ?flow murmurs ?S3
- Breath sounds- ?crepitations
- Ascites
- Peripheral oedema

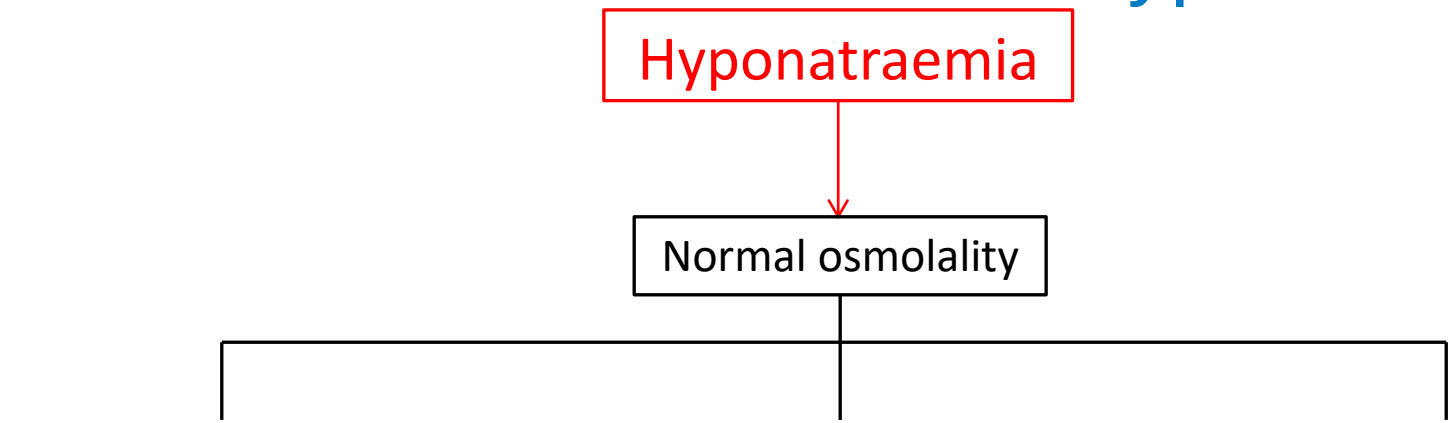
Investigations

- U+Es
- LFTs
- Osmolality (paired serum and urine)
- Urine Sodium and Potassium
- Thyroid function Tests
- 9am Cortisol
- CXR
- CT Head (?SIADH)

If required:

- Lipid profile
- Protein electrophoresis
- Glucose

Normal Osmolalities - Pseudohyponatremia



Hyperlipidaemia/
Hyperproteinaemia

- High levels of lipid or protein
- Does not contribute to osmolality

Hyperglycaemia

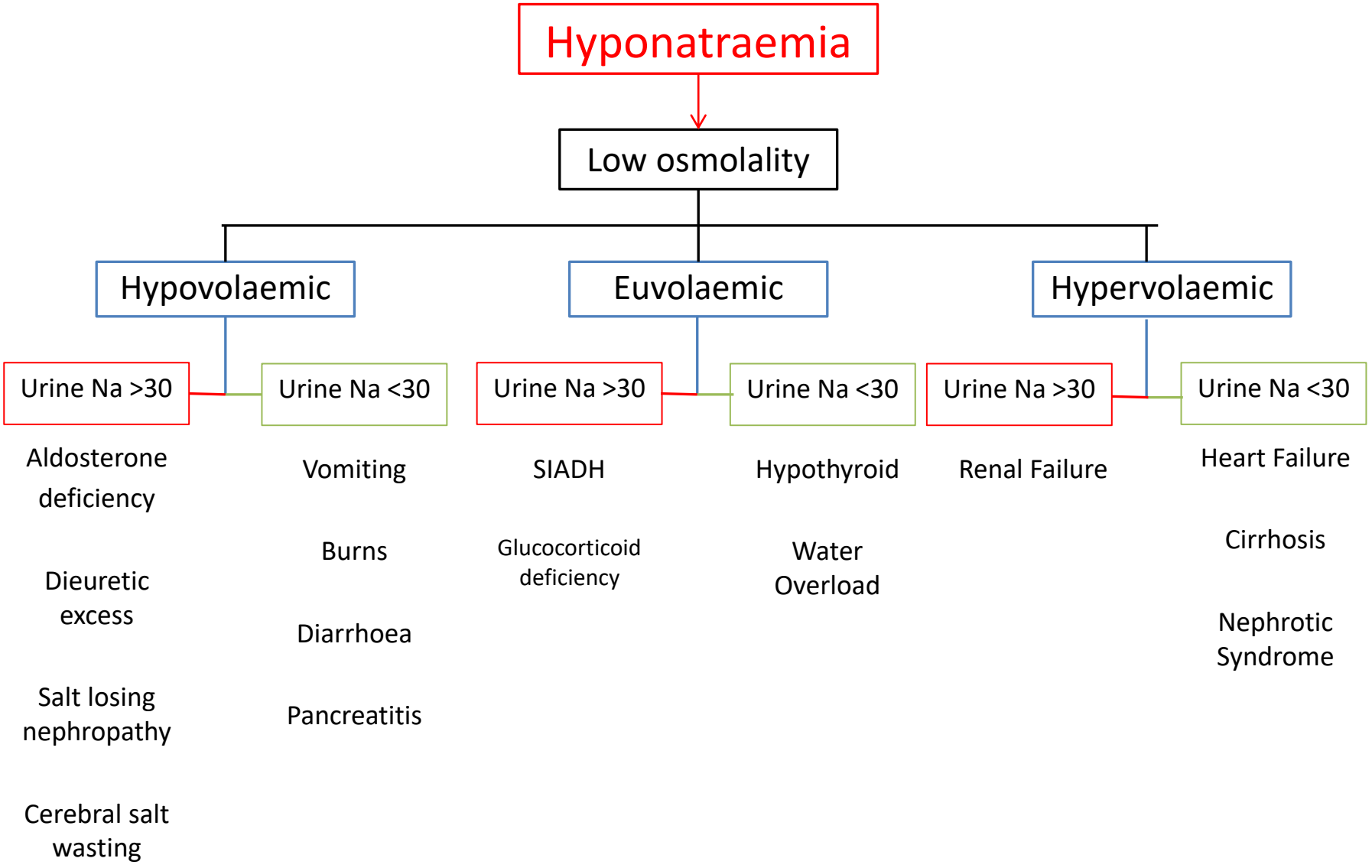
- Compensated osmolality
- Possibly raised osmolar gap depending on cause

- Na/K ATP-ase dysfunction
- Associated with hypoxia and sepsis
- Normal compensated osmolality

Corrected plasma Sodium:

$$\text{Measured Sodium conc.} + \frac{\text{Plasma glucose}}{4}$$

Classification

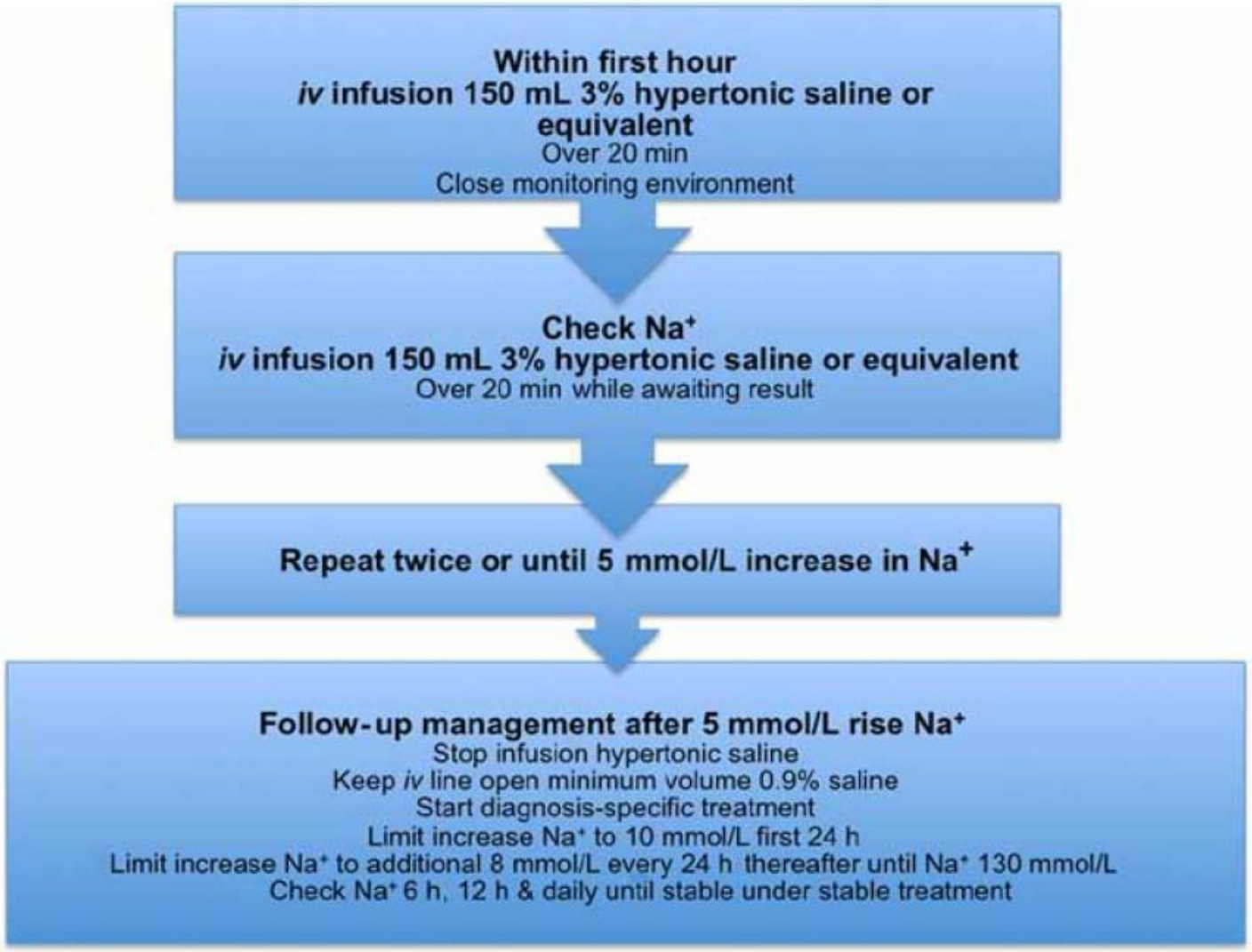


General Management- Emergencies

- If evidence of brain herniation:
  - Aim to raise Na by 5mmol/L in first hour, and then 1mmol/hr until resolution of neurological symptoms
  - If no resolution: aim to raise sodium to approximately 130mmol/L or by max 10mmol/L
  - Involve ITU
  - Sodium measurements 6 hourly
- Use Hypertonic saline
  - 3% saline in emergencies

- Reduces volume of fluid given

General Management- Emergencies



General Management

Sodium change:

$$\frac{\text{Na (infusate)- Na(serum)}}{\text{TBW+1}}$$

TBW = Body weight x %Water

%Water: Elderly = 50%
Adults = 55%
Children = 60%

$$\frac{513 \text{ mmol/L- } 105\text{mmol/L}}{80 \text{ X } 0.5 + 1}$$

= 10mmol/L change (if given 1L of 3%)

SIADH

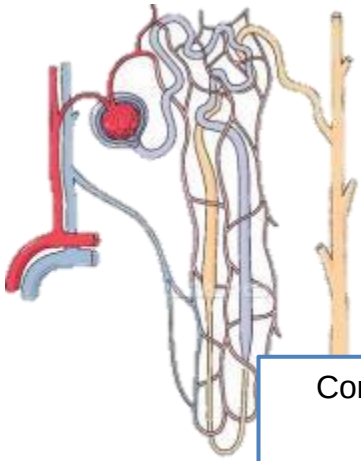
- Inappropriate secretion of ADH from posterior Pituitary or ectopic source
- Hyponatraemia with hypo-osmolality (<275mOsm/kg)
- Euvolaemia with raised urinary sodium (>20mOsm/kg) and inappropriately concentrated urine (>100mOsm/kg)
- Normal body response inhibited
  - Free water should be excreted
  - Normally urine osm should strictly be less than serum osm

SIADH – Normal physiology

Osmoreceptors detect high osmolality

ADH secreted

Aquaporin upregulated



Increased water reabsorption

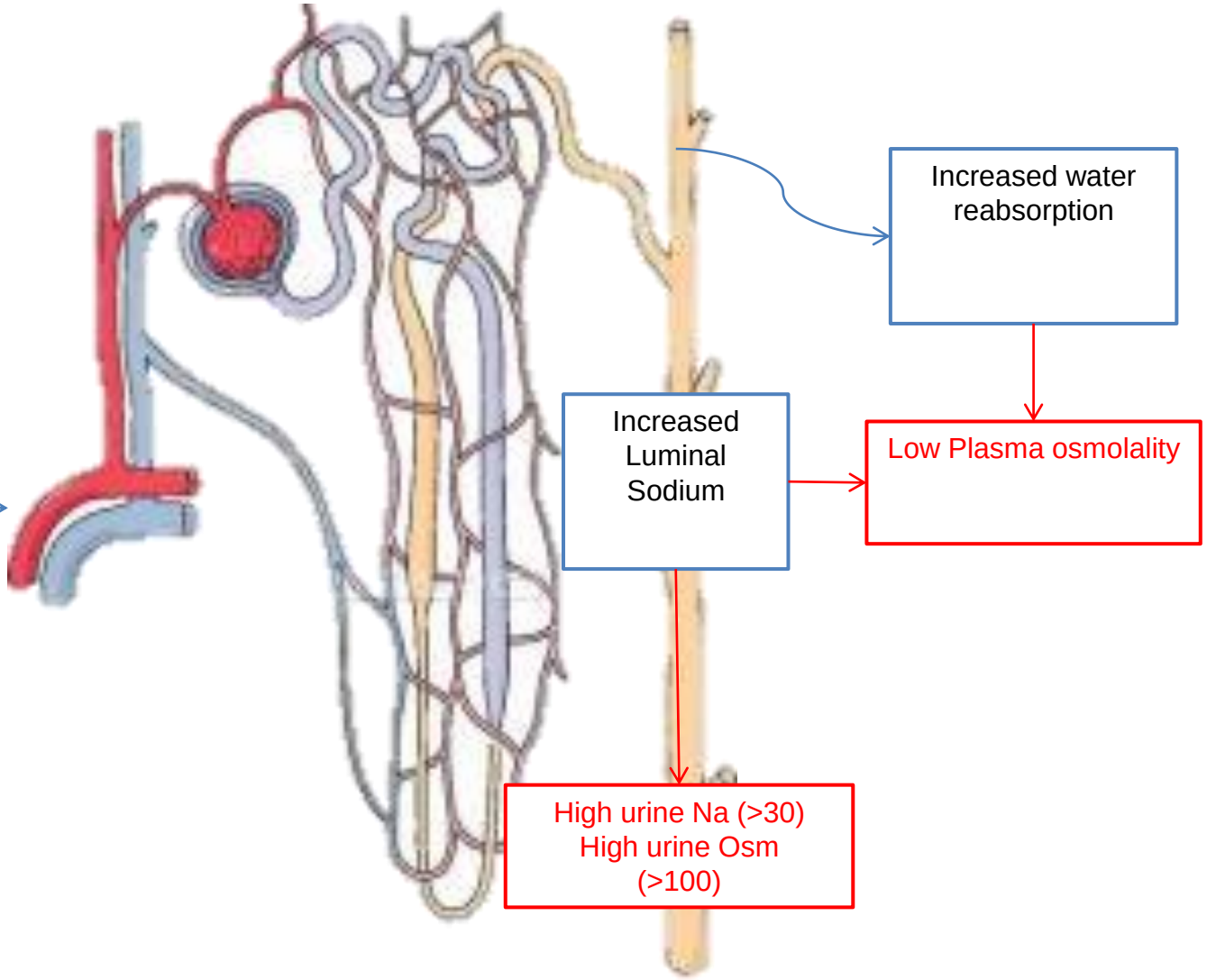
Concentrated urine

Increased sodium reabsorption (Aldosterone)

Normalising Osmolality

SIAH - Pathophysiology

Inappropriate ADH secretion



Increased water reabsorption

Increased Luminal Sodium

Low Plasma osmolality

High urine Na (>30)
High urine Osm (>100)

SIADH - Causes

- Intracranial pathology:
  - Trauma, Tumour, Infection, Thrombosis
- Pulmonary:
  - Small cell lung Ca, Mesothelioma, Abscesses, TB
- Malignancy:
  - GI (pancreas and stomach), Lymphoma, Leukaemia
- Drugs:
  - TCAs, SSRIs, AEDs, Vincristine, cyclophosphamide, Lithium, ecstasy
- Idiopathic

SIADH - Management

- If severe with symptoms
  - Treat with 3% saline until resolution of symptoms or Na- 130mmol or increase of 10mmol/L
- Fluid restrict- 1st line (unlikely to cause adverse effects)
- NaCl tablets + low dose furosemide – 2nd line
- Oral urea (0.25-0.5g/Kg) –alternative 2nd line
- Demeclocycline, then Vaptans in resistant cases???
- Patients should not be given 0.9% saline

Hyponatraemia – When to refer/admit

- There are no unified UK guidelines
  - Only local guidelines and European guidelines

- NICE CKS indicate admission if patients:
  - Have acute onset or severe hyponatraemia (serum sodium concentration of less than 125 mmol/L)
  - Are symptomatic
  - Have signs of hypovolaemia

- Discussion with endocrinology if patient:
  - Has asymptomatic, moderate hyponatraemia (serum sodium concentration of 125–129 mmol/L).
  - If Addison's disease is suspected, admission or urgent referral may be required.

Hyponatraemia – When to refer/admit

- Refer to an endocrinologist, the urgency depending on clinical judgement:
  - If the cause of hyponatraemia is not clear.
  - If SIAD or another endocrine cause is suspected.
  - If reset osmostat syndrome or cerebral salt wasting is suspected.

- Refer to an appropriate specialist:
  - If the person has hyponatraemia thought to be caused by heart failure, kidney disease, or liver disease.

Summary

- Treatment of hyponatraemia depends on classification
- Ensure that hyponatraemia is a true hyponatraemia
- Treat the underlying cause where possible
- If euvolaemic, fluid restrict and complete sodium screen