SydPath
ST VINCENT'S PATHOLOGY

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SODIUM INFORMATION SHEET

INTRODUCTION

The finding of abnormal sodium concentrations in the blood is a common clinical finding and correct management relies on making the correct diagnosis. The concentration of sodium in the blood is a affected by both the amount of sodium and the amount of water in the vascular space. The clinical effects of abnormal sodium concentrations are due to the movement of water in or out of cells, particularly in the brain, and are related to the speed of the changes as well as the absolute value of the concentration of sodium in the blood. See table 1 for a list of the likely effects of varying serum sodium concentrations.

The determination of the cause of abnormal sodium concentrations depends on knowledge of a number of factors:

- Clinical history
- Drug treatment (see table 2)
- State of hydration
- Urine sodium and osmolality

Additional blood tests which may also be of assistance include:

- Osmolality, as a test for true hyponatraemia
- Urea and creatinine, as markers of renal failure or dehydration
- Albumin and other LFTs

Table 1: Clinical Effects of serum sodium

> 160 mmol/L	Seizures, coma *
> 150 mmol/L	weakness, lethargy *
137 - 146 mmol/L	normal
125 - 136 mmol/L	no clinical changes expected
< 125 mmol/L	nausea, drowsiness *
< 120 mmol/L	vomiting, confusion *
< 110 mmol/L	convulsions, coma *

* Clinical effects may not be seen if changes have occurred gradually

HYPONATRAEMIA

Significant hyponatraemia is an important finding which requires investigation and treatment. The first steps in investigation are to exclude drugs, most commonly diuretics, as a cause and then to demonstrate the presence of a true (hypo-osmolar) hyponatraemia. The algorithm attached indicates a process for diagnosing the cause of hyponatraemia in most patients. Note that mild hyponatraemia in a bed-bound patient is an expected finding and not a cause for intensive investigation.

The syndrome of inappropriate secretion of anti-diuretic hormone (SIADH) is an important cause of hyponatraemia which requires the exclusion of cardiac, renal or endocrine disease, as well as a response to fluid restriction, in addition to the factors shown in the algorithm.

Table 2: Drugs and Hyponatraemia	
Diuretics	Thiazides, frusemide, spironolactone, amiloride, triamterene
Increase ADH secretion	Barbiturates, morphine, chlorpropamide, tolbutamide, vincristine, cyclophosphamide, carbemazepine, clofibrate, SSRIs
Potentiate ADH action	Chlorpropamide, NSAIDs, paracetamol

Syndrome of Inappropriate Anti-Diuretic Hormone (SIADH)

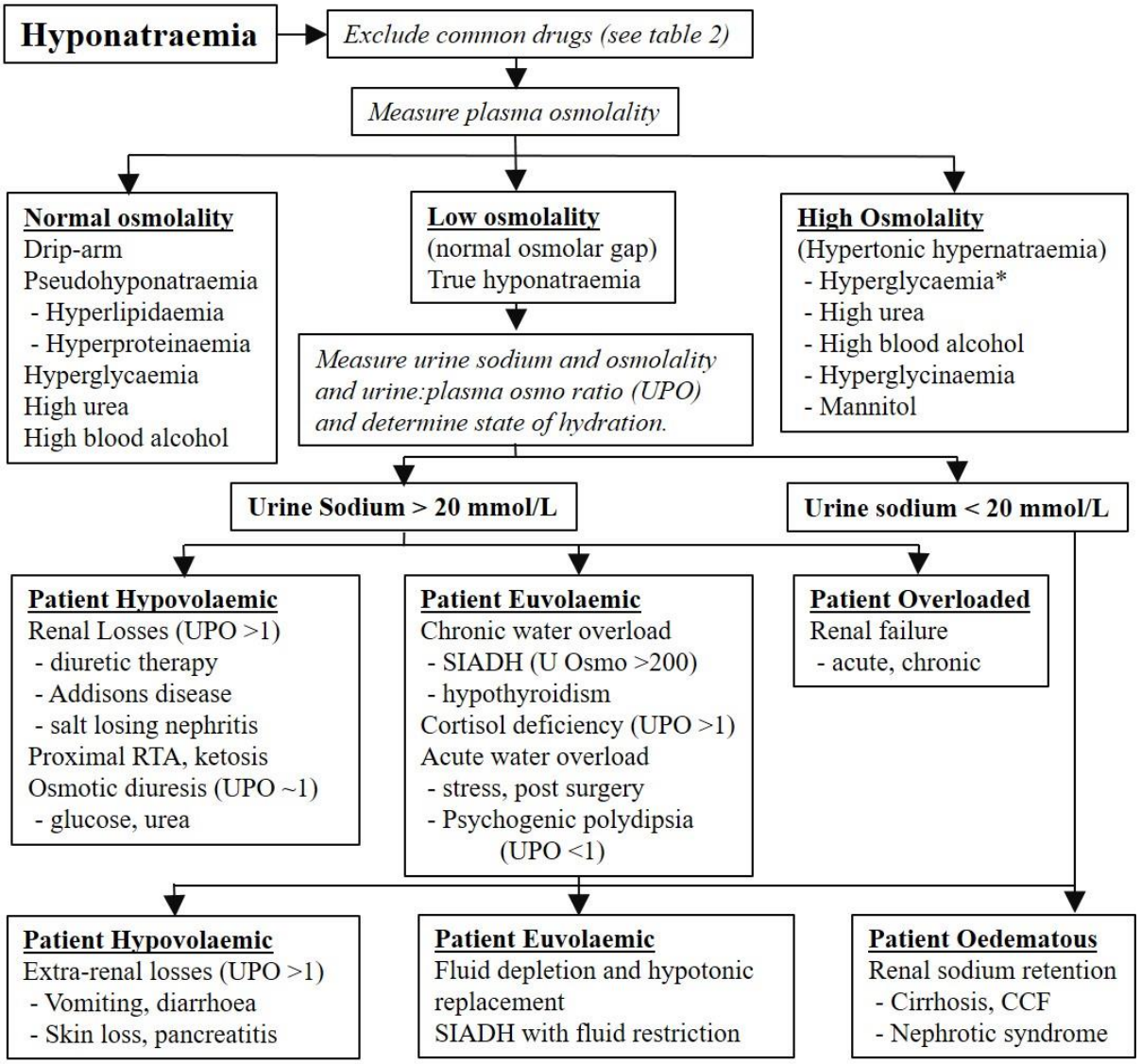
Diagnosis:

- Hyponatraemia (plasma sodium usually less than 130 mmol/L)
- Low plasma osmolality (normal osmolar gap)
- Urine osmolality > 200 mosm/kg
- Urine sodium > 20 mmol/L
- PLUS: Patient NOT dehydrated; NO cardiac, renal, pituitary or thyroid dysfunction; No drug cause for hyponatraemia; AND responds to reduced fluid intake

Causes:

- Tumours: carcinoma of bronchus, prostate, pancreas, brain
- Brain pathology: tumours, infections
- Lung Pathology: Tumours, infection, pneumothorax, hydrothorax
- Miscellaneous: Guillain-Barre syndrome, acute ethanol withdrawal.

HYPONATRAEMIA ALGORITHM

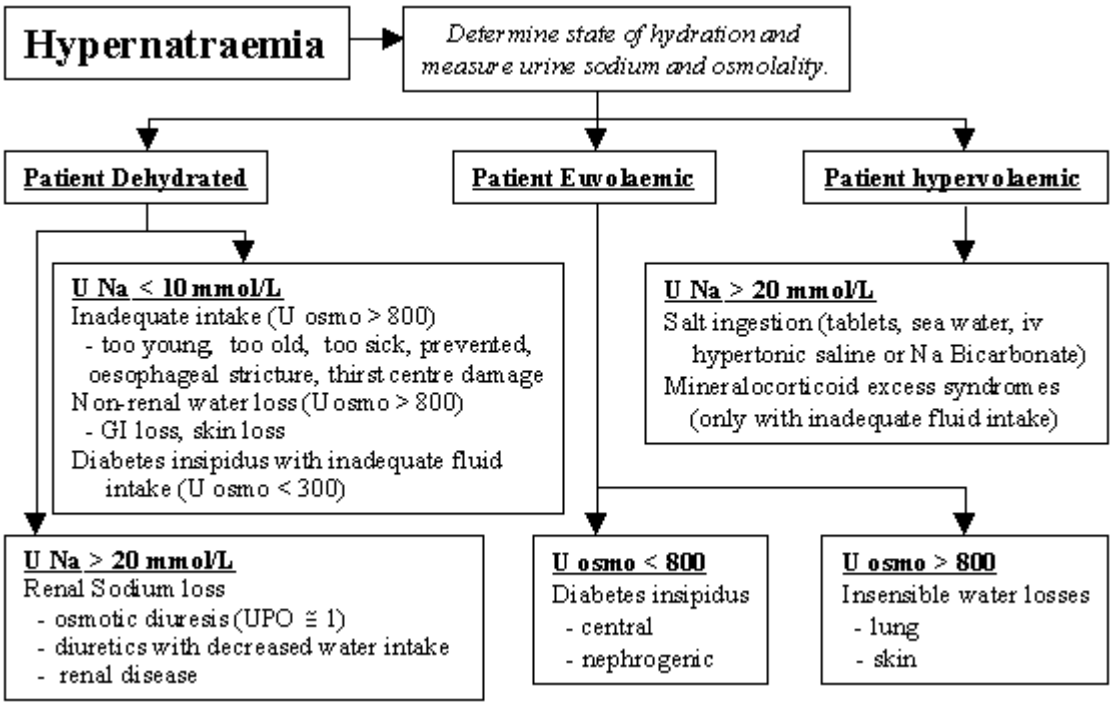


* Hyperglycaemia: Predicted sodium concentration after treatment of raised glucose = measured sodium + glucose/4 (all in mmol/L)

HYPERNATRAEMIA

Hypernatraemia is nearly always an indication of dehydration. The causes of hypernatraemia can be summarised as "too much salt or not enough water" and water loss is much more common than salt gain. An outline for determining the cause of hypernatraemia is shown overleaf.

HYPERNATRAEMIA ALGORITHM



CALCULATIONS INVOLVING SODIUM

Sodium Corrected for Hyperglycaemia
Anion Gap
Osmolar Gap (serum)
Fractional Excretion of Sodium (FENA)