HYPONATRAEMIA GUIDELINES

The degree of biochemical hyponatraemia is classified into three groups:

- Mild: Na+ 130-133 mmol/L
- Moderate: Na+ 125 – 129 mmol/L
- Profound: Na+ < 125 mmol/L

Severity of presentation does not match the degree of biochemical hyponatraemia. Profound hyponatraemia may be symptom free, while some patients with moderate biochemical hyponatraemia may be symptomatic, particularly if it has developed acutely

- **Severe symptoms:** persistent vomiting, cardiorespiratory arrest, seizures, reduced consciousness. *Please follow guidelines on page 2*
- **Moderately severe symptoms:** confusion, headache. *Please follow guidelines of page 2*
- **Mild or absent symptoms.** *Please follow guidelines on page 3*

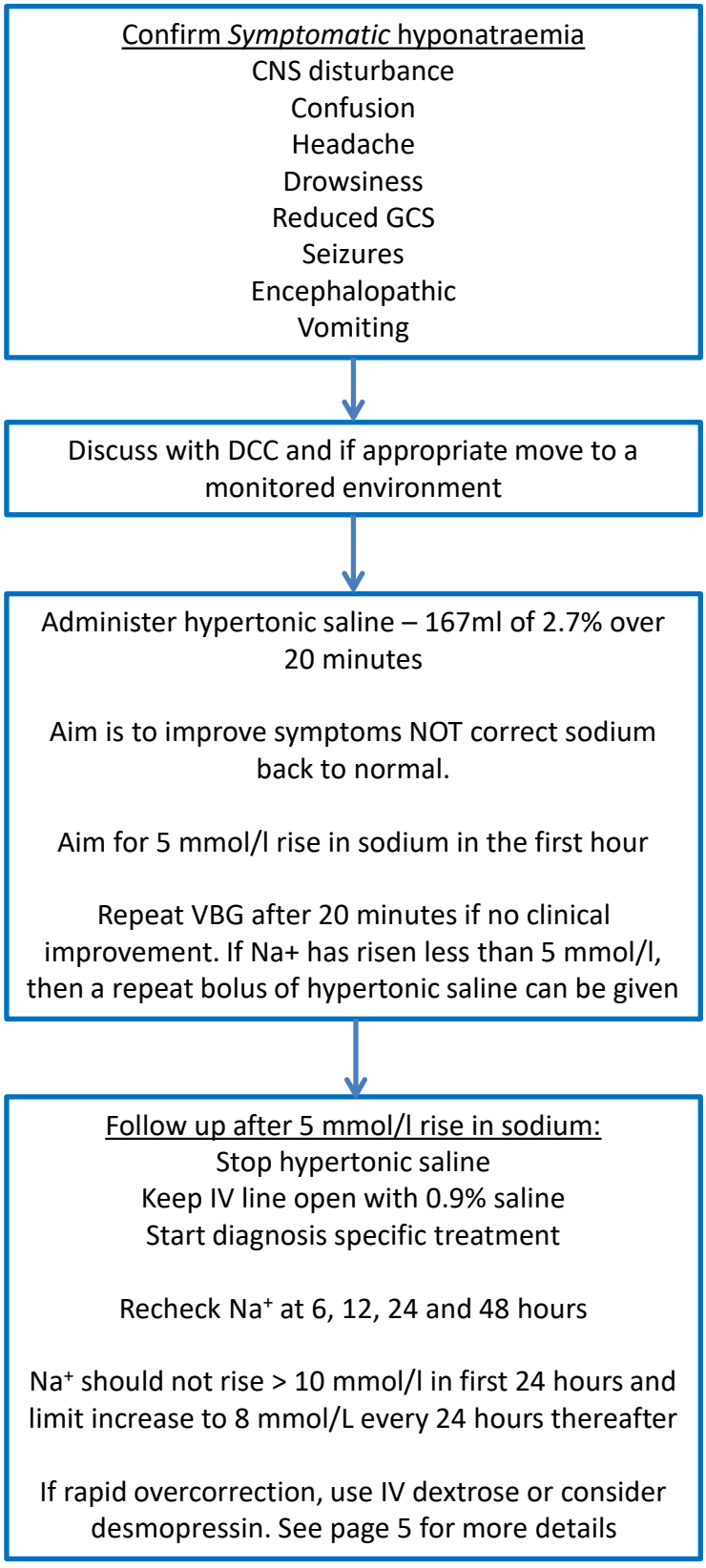
For all patients:

Note that if blood sample is reported to be lipaemic – consider pseudohyponatraemia (page 5 for more details)

- ❑ Clinical assessment
- ❑ Blood tests to include:
  - ❑ Serum osmolality
  - ❑ Repeat sodium level to confirm low level
  - ❑ Glucose (Sodium can be corrected for high glucose levels using calculator here: https://www.mdcalc.com/calc/50/sodium-correction-hyperglycemia)
  - ❑ 9am cortisol (unless on steroid treatment)
  - ❑ Thyroid function tests
  - ❑ Liver function tests
- ❑ Urine osmolality and urine Na+ *(must be sent in a white top specimen bottle)*
- ❑ Review drug charts and stop any contributing medications *(see table below)*

Potential causes of drug induced hyponatraemia (not an exhaustive list)	
Anticancer agents	vinca alkaloids (e.g. vincristine), platinum compounds (e.g. cisplatin), alkylating agents (e.g. cyclophosphamide)
Anti-depressants	tricycylic antidepressants, SSRIs, MAOI
Anti-epileptic medications	carbamazepine, sodium valproate
Anti-hypertensives	ACEi, ARB, amlodipine
Anti-psychotic medications	phenothiazines, butyrophenones
Diuretics	thiazides, indapamide, amiloride, loop diuretics
Proton pump inhibitors	omeprazole

MANAGEMENT OF SEVERE SYMPTOMATIC HYPONATRAEMIA



Important to compare blood testing with same method each time e.g. VBG with a VBG

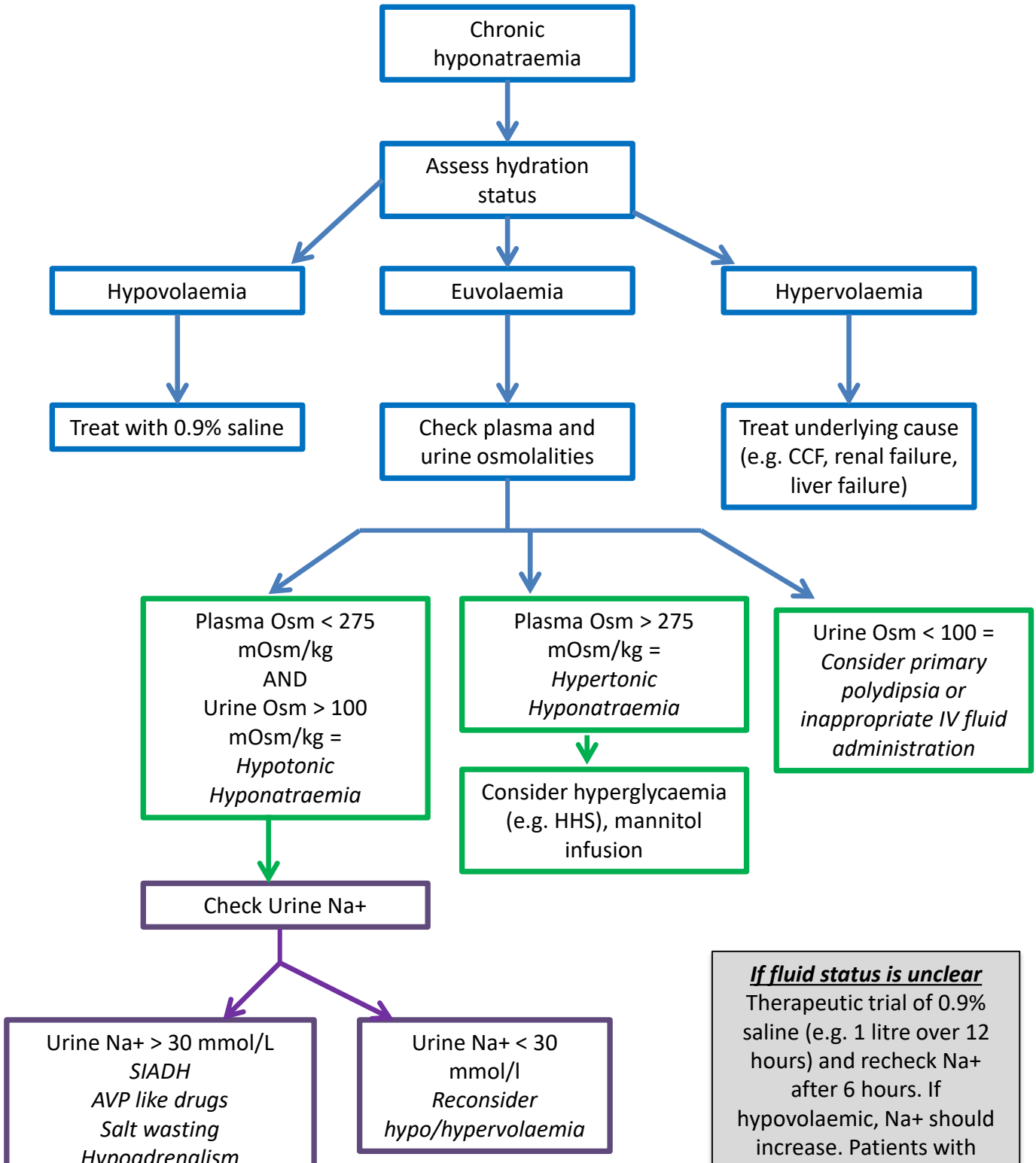
For hypertonic saline use:

167ml of 2.7% or 250ml of 1.8%

Should only be used following discussion with a consultant (registrar overnight)

Order hypertonic saline from pharmacy, or out of hours, obtain stock from the pharmacy emergency cupboard in CGH or GRH

CHRONIC HYPONATRAEMIA GUIDELINES



If fluid status is unclear

Therapeutic trial of 0.9% saline (e.g. 1 litre over 12 hours) and recheck Na+ after 6 hours. If hypovolaemic, Na+ should increase. Patients with SIADH don't improve or may worsen – discontinue fluids if so.

SIADH GUIDELINES

Confirm that:

❑ Clinically euvolaemic
❑ Excluded renal failure
❑ Excluded adrenal insufficiency
❑ Excluded severe hypothyroidism

❑ Urine Na+ > 30 mmol/l
❑ Urine Osmolality > 100 mOsm/kg
❑ Serum Osmolality < 275 mOsm/kg

❑ Consider underlying cause e.g. malignancy, infection, drug induced

❑ Stop any drugs that can cause hyponatraemia.

- If thought to be drug induced this may be all that is required. Monitor Na+ levels after stopping medications but there is no need to do fluid restriction unless Na+ not improving
- If hyponatraemia persists after stopping medications, repeat urine Na+ off treatment for a more accurate result

SIADH management

❑ Commence fluid restriction *500 -1000mls per day depending on severity & overall clinical situation*
❑ Ensure accurate fluid balance
❑ Treat underlying cause where possible
❑ Refer to endocrinology team via EPR if sodium not improving after 48 hours of fluid restriction

Common causes and likely duration of SIADH

Cause	Likely duration
ADH secreting tumour	Duration of the underlying disease
Medication induced, with continuation of the drug	Duration of treatment
Idiopathic SIADH of the elderly	Indefinite
Stroke or SAH	1 – 4 weeks
Cerebral tumours	Duration of the underlying disease
Respiratory failure	Dependent on response to therapy
HIV infection	Dependent on response to therapy
Head trauma	From 2-7 days to indefinite
Inflammatory cerebral lesions	Dependent on response to therapy
Nausea, pain, prolonged exercise	Variable, depends on cause
Post-op hyponatraemia	2-3 days
Pneumomia	2-5 days

NOTES

Rates of correction

Safe limit – 10 mmol/L in first 24 hours, 8 mmol/L in subsequent 24 hours
Groups at more risk of osmotic dymelination are elderly patients, children < 16, malnourished, alcoholics, CNS disease and post operative patients. May need to consider lowering limits for correction in these groups of patients.

Management of over-correction of serum Na+

- Monitor urine output
- Stop hypertonic fluid
- Consider using hypotonic (5% dextrose) fluid to slow rate of progression
- DDAVP (desmopressin) can be considered but only after discussion with endocrinology team

Tolvaptan advice

If using Tolvaptan (ADH antagonist) the following is advised:

- Discuss with endocrinology team before administration. Prescription can only be authorised by an endocrinology consultant
- Remove any fluid restriction
- Allow patient to drink to thirst response
- Dose will recommended by endocrine team, this then needs prescribing as a STAT dose on EPR
- Repeat Na^+ 6 hours after administration of Tolvaptan
- Repeat dose if no improvement after 24 hours (and if no improvement after second dose – reconsider diagnosis)
- May only need one or two doses to correct sodium levels back to normal so do not prescribe as a regular dose
- If sodium corrects too quickly, follow advice above to use 5% dextrose fluid

Pseudohyponatraemia

- Laboratory artefact caused by the presence of severe hyperlipidaemia or hyperproteinaemia. True sodium levels are not low, the result is due to interference with analysis of the test
- Measured serum osmolality will be normal in pseudohyponatraemia
- Measurement of sodium using the direct ISE in blood gas analyser will yield the true sodium concentration
- Need to treat underlying cause of raised lipids or protein and no specific treatment or investigation is needed for sodium levels in this situation

References

Society for Endocrinology endocrine emergency guidance: emergency management of severe hyponatraemia in adult patients. 2016. Ball et al

Table of SIADH causes adapted from:
I. Runkle et al / Med Clin (Barc). 2013; 141 (11): 507.e1-507.e10