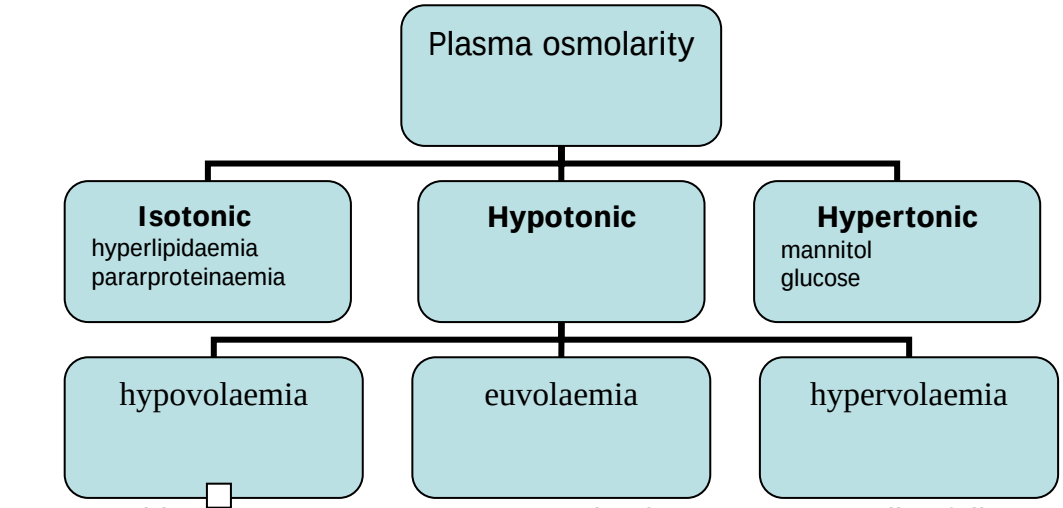
ELECTROLYTE ABNORMALITIES

SODIUM

Hyponatremia

Causes:



Extrarenal loss:
- urine Na<20mmol/l
- GI loss
- skin loss

Renal loss:
- urine Na >20mmol/l
- diuretics
- mineralocorticoid deficiency
- salt losing nephritis
- cerebral salt wasting

Water overload:
- water intoxication
- stress
- drugs
- renal insufficiency
- endocrine causes
- SIADH

- cardiac failure
- cirrhosis
- nephrotic syn
- renal failure

Clinical Features:
Nausea, vomiting, headache, confusion, seizures and coma

Treatment:
Depends on underlying cause. (e.g. fluid restriction in SIADH)
Rapid correction is only indicated in symptomatic patients (e.g. convulsions)
100mmol – 250mmol Na over 10min
after which the rate of correction must be slow

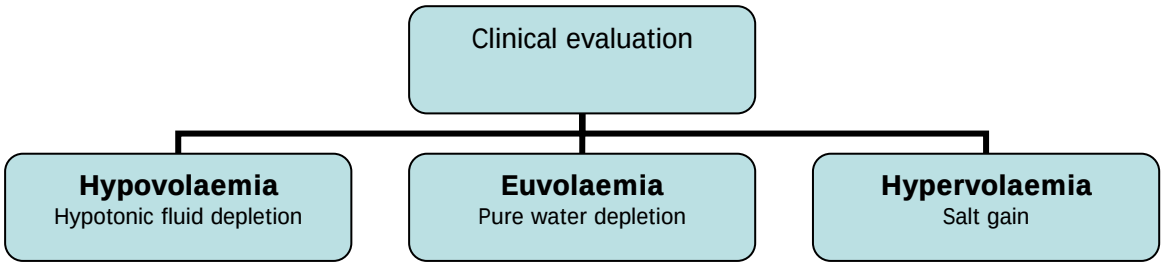
Acute (<3 days)
No greater than 2mmol/l/hr, do not raise Na >12mmol/24hours
Chronic (>3 days)
No greater than 0.5mmol/l/hr, do not raise Na >12mmol/24hours
Important:
1. In patients where you are not sure whether hyponatremia has developed acutely or chronically, assume it has developed chronically.
2. Correct Na slowly because of the risk of central pontine myelinolysis.
3. Plasma Na must be monitored closely.
4. If hypertonic saline is to be used, must discuss with ICU senior.

Note the sodium content in the various Na supplement:

NaCl tablet 600g	10mmol
NaCl 0.9%	0.15mmol/ml
NaCl 3%	0.5mmol/ml
NaCl 23.4%	4mmol/ml
$NaHCO_3$ 8.4%	1mmol/ml

Estimated Na deficit = 0.6 x BW x (desired – current Na)

Hypernatraemia
Causes:



check urine/plasma osmolarity
>1 extrarenal loss
= 1 osmotic diuresis
<1 diabetes insipidus

iatrogenic
mineralocorticoid xs

Note:
Hypotonic fluid depletion = isotonic fluid loss and pure fluid loss
Isotonic fluid loss from extracellular component =>minimal fluid shift
- ⇨ small increase in sodium and osmolarity
- ⇨ but vascular collapse

Pure water depletion

- ⇨ water loss shared between intra and extracellular components
- ⇨ minimum circulatory effects
- ⇨ but large increase in sodium and osmolarity

Clinical features:
Thirst, lethargy, seizures and coma

Treatment:
Depends on underlying cause/ correct underlying problems
(e.g. treat DI with ddAVP)
Correct systemic haemodynamics (isotonic component)
With 0.9% saline
Correct water deficit (pure water component)
Water deficit = 0.5 x BW X (plasma Na/140 – 1)
Replace with water orally, ½ saline, D5 solution
Dialysis may be considered

Important:

1. Correct Na slowly because of possible cerebral oedema
   Max rate = 0.5mmol/l/hour
2. Plasma Na must be monitored closely.

POTASSIUM

Hypokalaemia

Causes: (list not exhaustive)
Drip arm effect
Decrease intake
Increase loss: GI – vomiting, huge gastric aspirate, diarrhea, fistula
Renal – primary and secondary hyperaldosteronism
excess mineralocorticoid
drugs – diuretics, amphotericin, gentamicin
chronic pyelonephritis, polycystic kidney
diuretic phase of ATN
renal tubular acidosis
magnesium deficiency
Compartmental shift: hypothermia
alkalosis
insulin
refeeding syndrome
treatment of severe megaloblastic anaemia
hypokalaemic periodic paralysis
β2 agonists

Clinical features:
CVS – ECG changes (flattening of T waves, ST depression, U waves, prolong QT)
Arrhythmia – SVT, VT, torsades
GI - ileus, constipation
CNS – cramps/ parasthesia/ weakness/ tetany/ rhabdomyolysis
Others – glucose intolerance/ renal polyuria/ metabolic alkalosis

Treatment:
Treat underlying cause (e.g. replace Mg/correct alkalosis)
Oral route: syrup KCL – 1G = 13.4mmol
slow K - 600mg = 8mmol
intravenous: 10 – 20 mmol in 100 ml NS/D5 over 1 hour
This is a very concentrated K supplement and must be administered via central line

Hyperkalaemia

Causes: (list not exhaustive)

Pseudohyperkalaemia

Excessive intake: exogenous - K supplement/ massive transfusion
endogenous – burns, trauma, rhabdomyolysis/ tumor lysis

Decrease renal excretion:
Addison's disease
hypoaldosteronism
drugs – K sparing diuretics, indomethacin etc.
renal failure

Compartmental shift: acidosis
Insulin deficiency
Drugs – suxamethonium
Hyperkalaemic periodic paralysis

Clinical Features:

CVS – ECG changes (tall T waves, flat P waves, prolonged PR interval, prolonged QRS, sine waves)
Arrhythmia – VF

Muscle weakness

Treatment:

Treat underlying cause

Urgent situations – can cause cardiac arrest (follow ACLS guidelines)
50 -100ml 8.4% $NaHCO_3$ IV
10ml 10% $CaCl_2$ IV

enhance K shift to cells:
10 units actrapid + 50ml 50% D50 over 20 min
(close monitor K and H'stix)

enhance K excretion:
resonium A (sodium polystyrene sulphonate)
resonium C (calcium polystyrene sulphonate)
oral – 15g Q6H
rectal – 30g Q12H
consider dialysis

CALCIUM

Hypocalcaemia

Ionized Ca is more physiological important than total calcium
Adjusted Ca = (40 – albumin)/40 + measured Ca

Causes: (list not exhaustive/ many types of classification)
Factitious
Reduction in ionized Ca with normal total plasma calcium
- Respiratory alkalosis
- Citrate toxicity

Decreased PTH activity
- Hypoparathyroidism
- Pseudohypoparathyroidism
- Hypomagnesaemia

Vitamin D deficiency
Others
- Critical illness (sepsis, burns)
- Pancreatitis
- Rhabdomyolyis
- Hyperphosphataemia

Clinical Features:
CVS – hypotension
bradycardia
insensitivity to catecholamine and digoxin
ECG changes – QT/ST prolongation
Neuromuscular – anxiety, psychosis, confusion, seizures
Tetany, cramps, parasthesia
Laryngospasm, bronchospasm

Treatment:
Emergency – bolus 2.5-5mmol over 10min
Intravenoous - Ca gluconate 10ml = 2.3mmol
Ca chloride 10ml = 6.8mmol
Oral - Ca carbonate (Oscal 500mg/tablet, caltrate 600mg/tablet)
Usual requirement 1000mg/day
Measure ionized Ca levels
Most experts do not routinely recommend treating ionized Ca levels unless <0.8mmol/L or if symptoms develop.

Hypercalcaemia

Causes: (common causes)
Hyperparathyroidism
Malignancy
Granulomatous disease
Immobilization
Thyrotoxicosis
Excess vitamin D intake

Clinical features:
CVS – hypertension
arrhythmia
ECG changes – QT shortening
GI - anorexia, constipation
peptic ulcer, pancreatitis
CNS – depression, retardation, coma, seizure
Weakness, areflexia, atrophy
Others skeletal problems
Renal problems e.g. nephrocalcinosis, tubular dysfunction, DI

Treatment:

1. general measures
   - remove offending cause. Treat underlying cause.
   - dietary restriction
   - hydration (dilution effect)
2. increase Ca excretion
   - saline 2-3L over 3-6 hours
     maintain urine output 200ml/hour
     frusemide 10-40mg Q4H
   - consider dialysis

3. decrease bone resorption (should only be given after specialist advice)
   calcitonin – onset 6-10hours
   glucocorticoids
   biphosphonate, mithramycin

MAGNESIUM

Hypomagnesaemia

Causes: (list not exhaustive)

GI loss
- Reduced intake
- Reduced absorption
- Vomiting, prolonged diarrhea, refeeding syndrome

Renal loss

Drug induced loss – e.g. diuretics, amphotericin, aminoglycoside

Others – burns, sepsis, cardiopulmonary bypass, Mg free dialysate

Clinical Features:

CVS – arrhythmia, coronary artery spasm, CHF, prolong PR and QT

GI - anorexia, dysphagia, cramps

CNS - muscle weakness, lethargy, seizure, confusion, irritability

Associated with hypokalaemia and hypocalcaemia

Treatment:

Severe symptomatic hypomagnesaemia – 10mmol $MgSO_4$ over 5 min

Asymptomatic - 10mmol $MgSO_4$ over 2 hours

Preparation $MgSO_4$ 1g= 98mg Mg = 4mmol

Cautious replacement in patients with renal failure

Hypermagnesaemia

Causes: usually iatrogenic

Clinical Features:

Plasma concentration (mmol/L)	Effects
0.7 – 1.0	normal range
2.0 – 3.0	therapeutic range
3.0 – 3.5	ECG changes
4.0 – 6.0	areflexia
6.0 – 7.0	respiratory arrest
10.0 – 12.5	cardiac arrest

Treatment:

Stop Mg supplement

NM effects antagonized by 10ml 10% Ca gluconate

Normal renal function - frusemide

Renal failure - dialysis may be necessary

PHOSPHATE

Hypophosphataemia

Causes:

GI – decrease oral intake
malabsorption
fistula
diarrhoea

Renal – increase loss
diuretics, steroids
haemodialysis

Others – hyperparathyroidism, vitamin D deficiency,
alcoholism, treatment of DKA, refeeding syndrome,
burns, alkalosis

Clinical Features:

CVS – myocardial depression, hypotension and heart failure
Resp – respiratory failure
CNS – confusion, delirium, seizures
GI - anorexia, vomiting
Renal - renal tubular acidosis, hypermagnesuria, hypercalciuria
Haemat - haemolysis, platelet and leucocyte dysfunction
* Skeletal – muscle weakness, rhabdomyolysis, difficult to wean

Treatment:

Severe hypophosphataemia/symptomatic hypophosphataemia
KH_2PO_4/K_2HPO_4 10ml = 14.5mmol PO_4, 18.5mmolH and 25mmol K
Replace 5 – 10ml in maintenance fluid or normal saline over 6 hours
(check K and PO_4 and Ca after replacement)
Consider sodium phosphate if hyperkalaemia

Oral supplement: mist PO_4 10ml Q8H
(0.4mmol/ml)

Note: $[Ca][PO_4] > 6$
Risk of Calcium deposition in soft tissue

Hyperphosphataemia

Causes:

Factitious – haemolysis, sample separation delay
Redistribution – trauma,
rhabdomyolysis,
acidosis (keto and lactic)

tumor lysis
diphosphonate therapy

Positive phosphate balance – acute phosphate administration
phosphate enema
excess IV administration
vitamin D toxicity

Renal retention – renal failure
hypoparathyroidism
pseudohypoparathyroidism
acromegaly

Clinical Features:
(Not well documented. Symptoms are usually caused by the accompanying hypocalcemia and not the hyperphosphataemia *per se*)
Others - nephrocalcinosis, nephrolithiasis, band keratopathy

Treatment:
Usually aims at correcting the underlying hypocalcemia.
2 approaches to correct hyperphosphataemia:

1. Promote PO_4 binding in the upper GI tract (ie. GI dialysis)
   - lower the serum PO4 even in the absence of any oral intake phosphate
   - sucralfate or aluminium-containing antacids
   - calcium acetate tablets if significant hypocalcemia
     (each calcium acetate tablet = 667 mg contains 8.45 mmol calcium.
     Recommended dose 2 tab tid)
2. Enhance PO_4 clearance
   - hemodialysis. Reserved for patients with renal failure