Fluids and electrolytes

Fluids

Fluid intake is derived from two sources:

(1) Exogenous; and

(2) Endogenous.

Exogenous water is either drunk or ingested in solid food. The quantities vary within wide limits, but average 2—3 litres per 24 hours, of which nearly half is contained in solid food.

Taking into consideration their body weight, the water requirements of infants and children are relatively greater than those of adults because of: **(1)** the larger surface area per unit of body weight; **(2)** the greater metabolic activity due to growth; and **(3)** the comparatively poor concentrating ability of the immature kidney.

Endogenous water is released during the oxidation of ingested food; the amount is normally less than 500 ml/24 hours. However, during starvation, this amount is supplemented by water released from the breakdown of body tissues.

Fluid Compartments in the Body

The body's water is effectively compartmentalized into several major divisions.

- **Intracellular Fluid (ICF)** comprises **2/3** of the body's water.
  - If your body has 60% water, ICF is about 40% of your weight.
  - The ICF is primarily a solution of potassium and organic anions, proteins etc. (Cellular Soup!).
  - The cell membranes and cellular metabolism control the constituents of this ICF.
  - ICF is not homogeneous in your body. It represents a conglomeration التكتل of fluids from all the different cells.
- **Extracellular Fluid (ECF)** is the remaining **1/3** of your body's water.
  - ECF is about 20% of your weight.
  - The ECF is primarily a NaCl and NaHCO3 solution.
  - The ECF is further subdivided into three sub-compartments:
    - **Interstitial Fluid (ISF)** surrounds the cells, but does not circulate. It comprises about 3/4 of the ECF.

- **Plasma** circulates as the extracellular component of blood. It makes up about 1/4 of the ECF.
- **Transcellular fluid** is a set of fluids that are outside of the normal compartments. These 1-2 liters of fluid make up the CSF, Digestive Juices, Mucus, etc.

Special Notes:

- All the body's fluid compartments are in osmotic equilibrium (except for transient changes).
- The ions and small solutes that constitute the ECF are in equilibrium with similar concentrations in each subcompartment.
- **The ECF volume is proportional to the total Na content.**

Fluid output

Water is lost from the body by four routes.

• ***By the lungs:*** About 400 ml of water is lost in expired air each 24 hours. In a dry atmosphere, and when the respiratory rate is increased, the loss is correspondingly greater (this also applies to the patient who has their trachea intubated).

•***By the skin:*** When the body becomes overheated, there is visible perspiration, but throughout life invisible perspiration is always occurring. The cutaneous fluid loss varies within wide limits in accordance with the atmospheric temperature and humidity, muscular activity and body temperature. In a temperate climate the average loss is between 600 and 1000 m1/24 hours.

• ***Faeces:*** Between 60 and 150 ml of water are lost by this route daily. In diarrhoea this amount is greatly multiplied.

• ***Urine:*** The output of urine is under the control of multiple influences, such as blood volume, hormonal and nervous influences, among which the antidiuretic hormone plays a major role controlling tonicity of the body fluids, a function that it performs by stimulating the reabsorption of water from the renal tubules The normal urinary output is approximately 1500 ml/24 hours, and provided that the kidneys are healthy, the specific gravity of the urine bears a direct relationship to the volume. A minimum urinary output of approximately 400 m1/24 hours is required to excrete the end products of protein metabolism.

<u>Water depletion</u>

Pure water depletion is usually due to diminished intake. This may be due to lack of availability, difficulty or inability to swallow because of painful conditions of the mouth and pharynx, or obstruction in the oesophagus. Exhaustion and paresis of the pharyngeal muscles will produce a similar picture. Pure water depletion may also follow the increased loss from the lungs after tracheostomy. This loss may be as much as 500 ml in excess of

the normal insensible loss. After tracheostomy, humidification of the inspired air is an important preventive measure.

Clinical features

The main symptoms are weakness and intense thirst. The urinary output is diminished and its specific gravity increased. The increased serum osmotic pressure causes water to leave the cells (intracellular dehydration), and thus delays the onset of overt compensated hypovolaemia.

Water intoxication

This can occur when excessive amounts of water, low sodium or hypotonic solutions are taken or given by any route. The commonest cause on surgical wards is the overprescribing of intravenous 5% glucose solutions to postoperative patients. Colorectal washouts with plain water, instead of saline, have caused water intoxication during total bowel wash-through prior to colonic surgery. A major component of the TURP (transurethral resection of the prostate) syndrome is the water intoxication caused by excessive uptake of water (and glycine) from irrigation fluid.

Similarly, water intoxication can occur if the body retains water in excess to plasma solutes. This can be seen in the syndrome of inappropriate antidiuretic hormone (SIADH) secretion which is most commonly associated with lung conditions such as lobar pneumonia, empyema and oat-cell carcinoma of bronchus, as well as head injury.

Clinical features

These include drowsiness, weakness, sometimes convulsions and coma. Nausea and vomiting of clear fluid are common, and, with the notable exception of the SIADH, usually the patient passes a considerable amount of dilute urine. Laboratory investigations may show a falling haematocrit, serum sodium and other electrolyte concentrations.

Treatment

The intake of water having been stopped, the best course is water restriction. If the patient fails to improve, transfer to an intensive care or high dependency unit will be necessary for more invasive monitoring and controlled manipulation of fluids and electrolytes. The administration of diuretics or hypertonic saline should not be undertaken lightly as rapid changes in serum sodium concentration may result in neuronal demyelination and a fatal outcome.

Electrolyte balance

When inorganic salts are in solution, as in the extracellular or intracellular fluids of the body, they dissociate into ions. Ions are of two kinds: **(1) cations,** which are electropositive; and **(2) anions,** which are electronegative: collectively these are the electrolytes. The most accurate way of describing the chemical concentrations, reactivity and osmotic power of these ions is in **SI** units as millimoles per litre (mmol/litre). The cations include sodium,

potassium, calcium and magnesium; the anions include chloride, phosphate, bicarbonate and sulphate. The distribution of the salts within the fluid compartments of the body controls the passage of water through the cell walls and maintains acid—base equilibrium.

Sodium balance

Sodium is the principal cation content of the extracellular fluid. The total body sodium amounts to approximately 5000 mmol, of which 44 per cent is in the extracellular fluid, 9 per cent in the intracellular fluid and the remaining 47 per cent in bone. The sodium housed in bone merits special notice: a little more than half of it is osmotically inactive and requires acid for its solution; the remainder is water soluble and exchangeable. Thus, there is a large storehouse of sodium ready to compensate abnormal loss from the body. The daily intake of sodium is inconstant. On average it is 1 mmol/kg sodium chloride or 500 ml of isotonic 0.9 per cent saline solution. An equivalent amount is excreted daily, mainly in the urine and some in the faeces.

Control by adrenal corticoids

The output of sodium, governed by the renal tubules reabsorb sodium from the glomerular filtrate and the amount of sodium excreted by the sweat glands, is under the control of the adrenal corticoids, the most powerful conservator of sodium being aldosterone. When the adrenal glands have been destroyed by disease or extirpated, there is an open loss of sodium in the urine.

The sodium excretion shut down of trauma

Following trauma/surgery there is a variable period of reduced excretion of sodium. For this reason it may be inadvisable to administer large quantities of isotonic (0.9 per cent) saline solution after an operation. The period of sodium excretion shut down can last for up to 48 hours and is due to increased adrenocortical activity.

Sodium depletion (hyponatraemia)

Causes

***** obstruction of the small intestine, with its rapid loss of gastric, biliary, pancreatic and intestinal secretions by antiperistalsis and ejection, whether by vomiting or aspiration.

*Duodenal, total biliary, pancreatic and high intestinal external fistulae also are all bringing about early and profound hyponatraemia.

*Severe diarrhoea due to dysentery, cholera, ulcerative colitis or pseudomembranous colitis will cause hyponatraemia with acidosis.

*The finding of hyponatraemia with elevated potassium would suggest adrenocortical insufficiency.

*Hyponatraemia is also seen in SIADH.

* Gastric aspiration combined with allowing the patient to drink as he or she pleases and promptly aspirating the fluid swallowed. The act of drinking excites the flow of gastric juice, and this is also aspirated. During this form of therapy, should the patient be receiving intravenous dextrose solution to maintain fluid balance, he or she will soon become a victim of hyponatraemia.

Clinical features

Clinical features of hyponatraemia with salt and water depletion are due to extracellular dehydration. In established cases the eyes are sunken and the face is drawn. In infants the anterior fontanelle is depressed. The tongue is coated and dry; in advanced cases it is brown in colour. Unlike the dehydration produced by loss of water only, in water and salt depletion thirst is not particularly in evidence. The skin is dry and often wrinkled, making the patient look older than his or her years. The subcutaneous tissue feels lax. Peripheral veins are contracted and contain dark blood. The arterial blood pressure is likely to be below normal. The urine is scanty, dark in colour, of a high specific gravity and, except in cases of salt-losing nephritis, contains little or no chloride.

Presuming that the haemoglobin level before the dehydration commenced was normal, the haematocrit reading (PCV) provides an index of the degree of haemo concentration. However, haemoconcentrations can be masked by pre-existing anaemia. Laboratory investigations would show normal or slightly reduced serum sodium with low urinary output and low urinary sodium.

Postoperative hyponatraemia

Hyponatraemia with a normal or increased extracellular fluid volume arises as a result of too prolonged administration of a sodium-free solution.

Sodium excess (hypernatraemia)

This is likely to arise if a patient is given an excessive amount of 0.9 per cent saline solution intravenously during the early postoperative period when, as has been described, some degree of sodium retention is to be expected. The result is an overloading of the circulation with salt and its accompanying water.

Clinical feature

Slight puffiness of the face is the only early sign. The patient makes no complaint. Pitting oedema should be sought, especially in the sacral region, but for pitting oedema to be present at least 4.5litres of excess fluid must have accumulated in the tissue spaces. The patient’s weight increases pan. Signs of overhydration in infancy (infants are very susceptible) are increased tension in the anterior fontanelle, increased weight, an increase in the number of urinations and oedema.

Potassium balance

Potassium is almost entirely intracellular. No less than 98 per cent is intracellular, and only 2 per cent is present in the extracellular fluid. Three quarters of the total body potassium (approximately 3500 mmol) is found in skeletal muscles. When the body needs endogenous protein as a source of energy, potassium, as well as nitrogen, is mobilised. Each day a normal adult ingests approximately 1.0 mmol/kg of potassium in food; fruit, milk and honey are rich in this cation. Except for a very small quantity in formed faeces, and a still smaller quantity in sweat.

The augmented potassium excretion of trauma

Following trauma, including operation trauma, there is a spellنوبة, varying directly with the degree of tissue damage, of increased excretion of potassium by the kidneys. This loss is greatest during the first 24 hours and lasts, for example in the case of partial gastrectomy, for about 3 or 4 days. So great are the body's reserves of potassium that, unless the patient was severely depleted at the time of the operation, hypokalaemia may not reveal itself for 48 hours. However, potassium is such a key intracellular cation that carefully monitored replacement should start early in the postoperative period in all patients, with the exception of those that have evidence of renal dysfunction.

Hypokalaemia

Hypokalaemia can occur suddenly or gradually.

Sudden hypokalaemia is unlikely to be encountered in surgical practice. It occurs most frequently in diabetic coma treated by insulin and prolonged infusion of saline solution.

Gradual hypokalaemia is the type encountered in surgical practice.

*****It is most commonly seen in patients who present for surgery with chronic hypokalaemia as a result of potassium-losing medications such as diuretics.

*The diarrhoea from ulcerative colitis, villous tumours of the rectum and the loss from external fistulae of the alimentary tract are also common causes (e.g. duodenal fistula, ileostomy); the potassium content of the discharge from some of these fistulae is twice that of the plasma potassium concentration.

*****Another frequent cause of hypokalaemia is prolonged gastroduodenal aspiration with fluid replacement by intravenous isotonic saline solution.

*****It is also prone to occur in the postoperative period following extensive resections for carcinoma of the alimentary tract, because often the operation has to be undertaken after months of weight loss and potassium depletion.

Clinical features

Most patients are asymptomatic, but at risk of the sequelae of hypokalaemia such as cardiac arrythmias. Such consequences are more likely during surgery and anaesthesia, especially in the presence of pre-existing myocardial disease. Symptoms of severe hypokalaemia include listlessness and slurred speach, muscular hypotonia, depressed reflexes and abdominal distension as a result of a paralytic ileus. Weakness of the respiratory muscles may result in rapid, shallow, gasping respirations; these are conducive to postoperative pulmonary complications. The diagnosis is supported by electrocardiography (ECG), which may. show a prolonged QT interval, depression of the ST segment and flattening or inversion of the T-wave.

Treatment

Oral potassium: Potassium can be given in the form of milk, meat extracts, fruit juices and honey. However, in hospital practice, effervescent tablets of potassium chloride 2 g can be given by mouth 6-hourly.

Intravenous potassium: Rapid intravenous supplementation (especially when renal function is impaired) carries the risk of dysrhythmias and cardiac arrest if the serum concen-tration rises to a dangerous level. Administration should be properly controlled; the level of potassium should be checked daily; the urine output must be adequate. The potassium deficit can be restored by adding 40 mmol potassium chloride to each litre of 5per cent glucose, glucose—saline or 0.9 per cent saline solution, which is given 6—8-hourly. Severe hypokalaemia should be treated in a high dependency or intensive care environment.

Estimation of electrolyte balance

Sodium

The serum sodium value is normally between 137 and 147 mmol /litre. Whenever possible, the serum chloride and bicarbonateshould be estimated simultaneously because variations in the one may be accompanied by oppo-site changes in the other. The normal level of chloride is 95—105 mmol/litre, and of bicarbonate 25—3 0 mmol /litre; the sum of the two remains roughly constant at 120—135 mmol/litre.

Potassium

Potassium deficiency is present if the serum potassium value is less than 3.5 mmol/litre. The normal range is 3.5—5.0 mmol/litre. It must be remembered that intracellular potassium deficiency may be present although the plasma concentration is normal, and that deficiency is to be expected if oral feeding has been withheld for more than 4 days.

Calcium

Calcium is an extracellular cation with a plasma concentration of 2.2—2.5 mmol/litre. It exists in three forms: bound to protein, free nonionised and free ionised — the last form being the component necessary for blood coagulation and affecting neuromuscular excitability. The ionised proportion falls with increasing PH; thus in respiratory alkalosis due to hyperventilation there may be tetany — with an apparently normal total serum calcium level. In the urine, the ionisation and the solubility of calcium are similarly depressed if the pH is elevated, thus promoting stone formation. The serum level of calcium is likely to be modified by any factor promoting or inhibiting its absorption from the bowel, its storage in bone or its elimination by the kidneys: such factors include vitamin D and , parathormone and calcitonin , and the state of renal and small-bowel function.

The management of abnormal calcium blood levels depends, where possible, on removal of the cause, for example removal of a parathyroid tumour , or in the coagulation disorder due to massive transfusion of blood containing acid citrate dextrose , 10 ml of 10 per cent calcium gluconate may be injected slowly intravenously. If oral administration is possible, calcium tablet is useful. On a long-term basis, the diet should be adjusted to provide high calcium and a low phosphate intake.

Magnesium

Magnesium is an intracellular cation which shares some of the properties of potassium and some of calcium. The normal magnesium concentration is 0.7—0.9 mmol /litre. The average daily intake is approximately 10 mmol. Magnesium deficiency may occur *when there is prolonged loss of gastrointestinal secretions due to fistulae or ulcerative colitis,

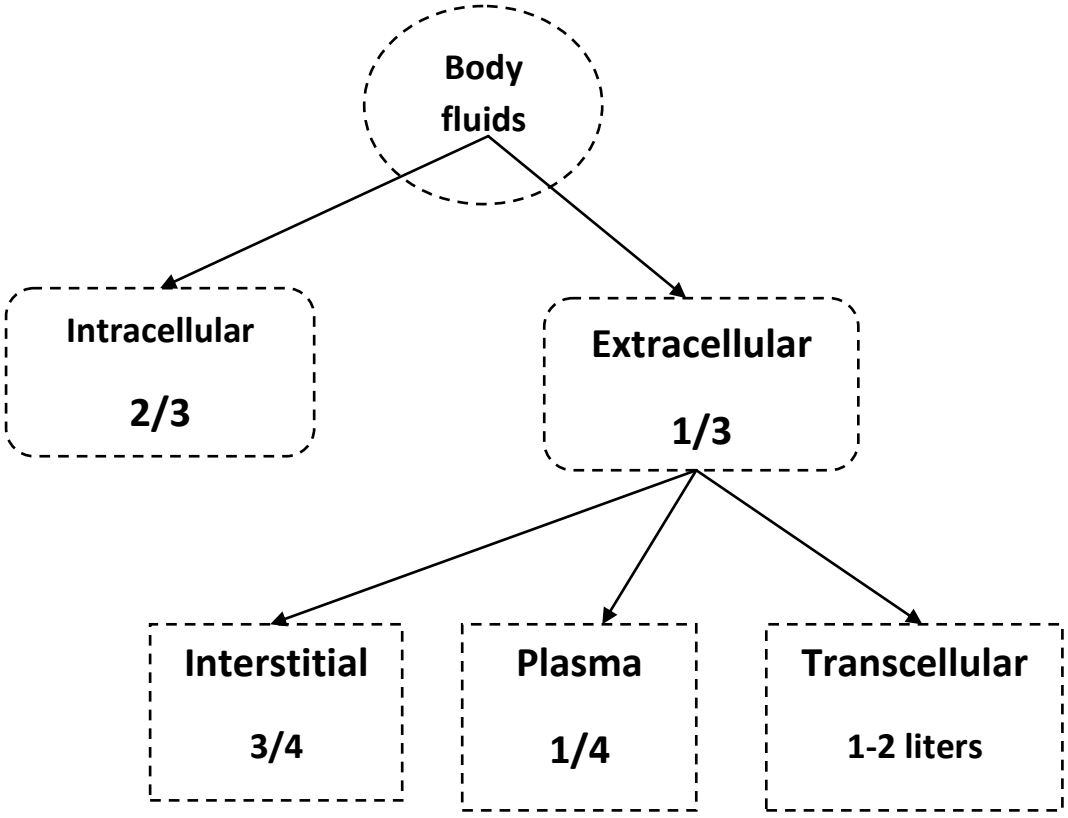
*very prolonged administration of intravenous fluids without magnesium supplements, *following massive small bowel resections,

*some cases of cirrhosis of the liver or disease of the parathyroids.

The clinical picture of magnesium deficiency is marked by central nervous system irritability, ECG changes, lowered blood pressure and lowered protein synthesis. Postoperative cardiac arrythmias (e.g. de novo atrial fibrillation) are commonly associated with both hypokalaemia and hypomagnesaemia.

Treatment

For the treatment of mild hypomagnesaemia 20 mmol as magnesium sulphate can be added to 5percent-dextrose or normal saline over a 24-hour period. Magnesium supplements are essential in hyperalimentation.


Body fluids
Intracellular
2/3
Extracellular
1/3
Interstitial
3/4
Plasma
1/4
Transcellular
1-2 liters

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