POCKET CONSULTANT

Fluid Balance

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Preface

The newly-qualified intern faces a number of problems in the field of fluid management, particularly on the surgical wards where the daily prescribing of fluids and electrolytes is largely his or her domain. Many of the practical aspects are learnt from more senior doctors but much of the time he is on his own.

This book covers the basic aspects of fluid management problems commonly encountered, including fluid therapy in the pre- and postoperative patient, and offers a practical approach to common electrolyte and acid-base disorders and enteral and parenteral nutrition. It also outlines practical aspects of peripheral and central venous access, and the approach to acute fluid resuscitation in the injured patient.

It will be of use as a practical guide to the newly-qualified house surgeon, as well as to the medical student in his or her clinical years and to nursing staff on surgical wards and in intensive therapy units. It is designed to supplement the many excellent larger texts available on the subject of fluid balance and deals with theoretical backgound only in so far as is necessary for a practical understanding by the man 'on the spot'.

I would like to thank the Pharmacists, Intravenous nursing team and Nutrition nursing team at the Royal Marsden Hospital, Fulham Road, London for their help. I also thank Drs Chris Dark, Rob Davidson and Mark Vincent for their constructive comments.

Tim Davidson
1987

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1.1 Body fluid compartments

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An understanding of body fluid compartments is of practical importance in fluid management. Biochemical changes measured on serum samples reflect only the composition of intravascular fluid and may not fully represent the disturbance to total body fluid or electrolyte status.

Total body water (TBW) constitutes about 60% of body weight in males and 52% in females (due to the higher fat content in females). In obese persons and in the elderly TBW is likewise lower. A healthy 70 kg man therefore has about 40 litre TBW, 2/3 of this being *intracellular* fluid (ICF). Of the *extracellular* fluid (ECF) about 25% is plasma volume and the rest interstitial fluid (Fig. 1.1). Adult *blood volume* is about 5 litres (70 ml/kg). Blood volume in the infant and child is relatively greater, 85 ml/kg in the first year of life, 80 ml/kg between 2 and 5 years, 75 ml/kg between 6 and 12 years.

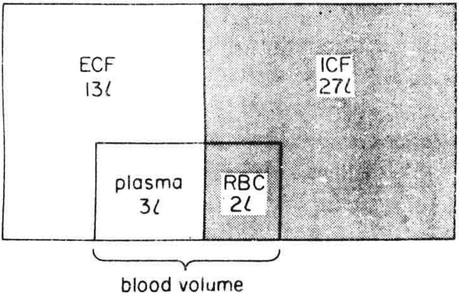

Fig. 1.1. Distribution of body water in a 70 kg adult. ECF (13 l) comprises ISF (10 l) and plasma (3 l). ICF (27 l) includes the red cell volume (2 l).

The composition of ICF and ECF differs because of cell membrane function with active transport of ions via the Na-K-ATPase pump and containment of high MW molecules. Electrical neutrality is maintained in each compartment (total anions = total cations when expressed in mEq/l, *see* Fig. 1.2) but an 'anion gap' in plasma is measurable due to the presence of negatively charged plasma proteins, particularly albumin, and to a lesser extent

1 Body fluid composition and daily requirements

1.1 Body fluid compartments

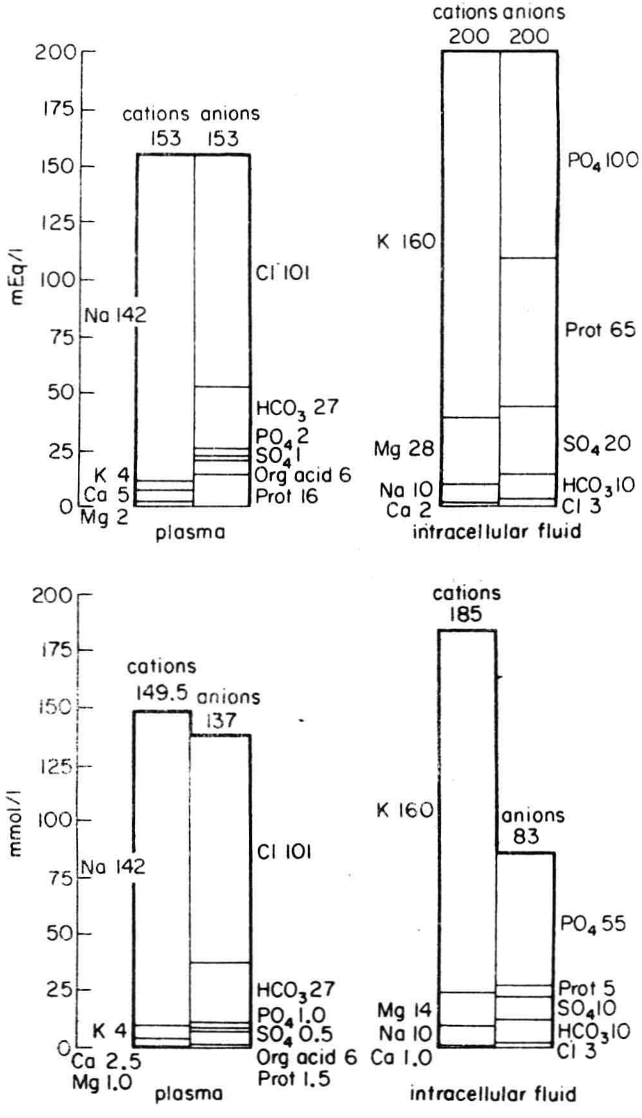

Fig. 1.2. Composition of plasma and ICF.

phosphate, lactate and organic acids. Anion gap may be calculated by:

$$(Na + K) - (Cl + HCO_3) \text{ (expressed as mmol/l)}$$

giving a value with a normal plasma sample of 12–18 mmol/l (or, if K is excluded from the equation, 5–12 mmol/l). Anion gap is most commonly increased in metabolic acidosis (uraemia, ketoacidosis, lactic acidosis); also with exogenous anions (ethanol, salicylates, penicillin). It is of value in detecting an abnormality before more specific investigations can be performed (*see* 8.6). Less commonly, anion gap is decreased (hypoalbuminaemia, dilutional states, hypercalcaemia). The ionic composition of plasma and *interstitial fluid* (ISF) differs slightly because plasma proteins are confined largely to the intravascular space.

1.2 Osmolality

The total molar concentration of particles per kg of solvent is termed *osmolality*. (The molar concentration of particles per litre of solution is termed *osmolarity* and for many biological solutions is numerically similar to osmolality because 1 litre of solution contains close to 1 kg water.)

Most of the osmotically active particles in plasma are the electrolytes and small molecules. Na (and Cl) concentration is the main determinant of ECF osmolality, whereas K (and PO_4) concentration is the main determinant of ICF osmolality. Less than 1% of plasma osmolality is attributable to colloid because large protein molecules represent a relatively tiny fraction of osmotically active particles in solution. Normal plasma colloid osmotic pressure (22–25 mmHg) is in fact partly attributable to the slightly higher concentration of Na ions in plasma than in ISF as a result of plasma proteins (Donnan effect).

Plasma osmolality may be rapidly approximated from the formula

$$2(Na + K) + \text{glucose} + \text{urea (each expressed in mmol/l).}$$

Normal plasma osmolality is 280–295 mosm/kg. An isotonic or normal (N) solution has osmolality similar to plasma. Hypotonic solutions (for example 0.45% saline) have osmolality less than, and hypertonic solutions (for example, 20% dextrose) osmolality greater than that of plasma.

1.3 Water balance

In a healthy subject water intake and output vary considerably but average daily water requirement in an adult is about 1500 ml/m^2, or 2600 ml/day (Table 1.1). Water intake is via oral fluids, the water content of food, and from metabolism (water of oxidation). Oxidation of food to carbon dioxide and water provides 120 ml/1000 Kcal. In health the proportion of metabolic water is small, but it may need consideration when large amounts of carbohydrate are administered with parenteral nutrition (*see* 6.6).

Insensible water loss from body surface and lungs is about 1000 ml/day. Water loss via the skin varies greatly depending on body temperature, muscular activity, and environmental temperature and humidity. In temperate climates it is about 600 ml/day but can be increased substantially in tropical climates and with pyrexia. Insensible perspiration is low in electrolytes; moderate sweating on the other hand contains Na and Cl 50–100 mmol/l and 5 mmol/l K. In children and infants insensible water loss is proportionately higher relative to body weight.

Saturation of expired air with water vapour results in about 400 ml loss per day via the lungs. This loss can increase several fold with tachypnoea and tracheostomy, particularly if the inspired air is unhumidified.

Gastrointestinal fluid loss in health is small but may be greatly increased with diarrhoea. Large volumes of fluid are secreted into and reabsorbed from the GI tract each day (*see* Fig. 4.1) and losses by vomiting, NG aspiration or fistula may result in rapid dehydration unless adequately replaced.

1.3 Water balance

Table 1.1. Adult daily water intake and output.

intake (ml)		output (ml)	
fluid	1300	urine	1500
solids	1000	skin	600
metabolism	300	lungs	400
		faeces	100
	2600		2600

In health the kidneys excrete about 1500 ml/day, excreting unwanted solutes by active transport. The minimum volume of urine in which the solute load can be adequately excreted is about 500 ml/day. Depending on the ratio of solute/water, urine osmolality can vary widely, from 30 mosm/kg to 1400 mosm/kg. Measurement of urine specific gravity (SG) is less accurate than osmolality but can be measured at the bedside with a hygrometer or reagent strips (Multistix SG). In the absence of glucose, protein or mannitol in the urine, SG correlates with urine osmolality thus:

urine SG	*osmolality (mosm/kg)*
1.010	350
1.020	700
1.030	1050
1.040	1400

The neuroendocrine control of fluid and electrolyte balance is complex. Water depletion or an increased osmolar load will stimulate the thirst centre in the hypothalamus and secretion of *antidiuretic hormone* (*ADH*) from the posterior pituitary causing water retention by the distal renal tubules. Nonosmotic control mechanisms are also important. Hypovolaemia is detected by carotid sinus and aortic baroreceptors, and stimulation of the *renin–angiotensin–aldosterone* system results in Na and water retention by the kidney. *Atrial natriuretic peptide* (ANP) has a potent diuretic action causing increased Na, water, Ca and Mg excretion in the urine and ANP levels are raised in response to heart failure, hyperaldosteronism and increased Na intake. Other hormones including catecholamines, glucocorticoids and prostaglandins affect ADH and renal water excretion.

The normal kidney can maintain fluid and osmotic homeostasis over a wide range of fluid intakes, which is why differing fluid regimens in common hospital practice prove adequate for most patients (*see* 2.2). The metabolic response following trauma or surgery however reduces the kidney's ability to excrete large quantities of water or sodium (*see* Fig. 4.2). For the kidney to excrete excess water and render the urine hypotonic ('free water' excretion) ADH must be suppressed, whereas ADH production is increased as part of the metabolic response to stress.

1.4 Fluid requirements in childhood

Fluid requirements appear relatively higher than in adults because rate of fluid metabolism corresponds to surface area rather than body weight. Fluid requirements in infants and adults are identical when expressed per surface area. Use the following guidelines in children:

first 10 kg body wt : 100 ml/kg/day
second 10 kg body wt : 50 ml/kg/day
wt in excess of 20 kg : 25 ml/kg/day

For example, a 14 kg toddler's daily basal requirements are 1000 + 200 = 1200 ml or a 24 kg child's daily requirements are 1000 + 500 + 100 = 1600 ml.

Electrolyte requirements: Na 3 mmol/100 ml water
K 2 mmol/100 ml water

Fluid balance in sick neonates and infants requires expert supervision. The concentrating power of the kidney is reduced in infants and in dehydrated and ill children. Daily fluid requirements in the first few days of life are reduced:

Age	*Daily maintenance requirements*
1 day	30–60 ml/kg
2 days	60 ml/kg
3 days	90 ml/kg
4 days & over	100–110 ml/kg

1.5 Sodium

A 70 kg man contains 4000 mmol Na, 50% in ECF, 40% in the skeleton (most of which is nonexchangeable) and 10% in ICF. Normal adult Na requirement is 80 mmol/day. Because Na occupies largely the ECF compartment, if overinfusion with saline occurs the expansion is primarily in ECF volume (and plasma Na may be normal or raised) whereas in a patient overloaded with 5% dextrose the extra water is distributed throughout the TBW (*see* Fig. 8.1). Na output is almost entirely in the urine (unless there is excessive sweating). Abnormal Na loss can occur as follows:

- from the GI tract (vomiting, ileus, fistula);
- from the kidney (diuretics, polyuric renal failure, postobstructive diuresis, adrenal insufficiency);
- from the skin (sweating, burns);
- severe trauma, peritonitis.

Hyponatraemia

Pure Na depletion is rare (Na cannot be lost without accompanying water) and usually there has been loss of Na and water with inadequate replacement of Na (for example, excessive sweating replaced with drinking water only, GI losses replaced with 5% dextrose or dextrose–saline).

Normal plasma Na is 135–148 mmol/l. With any lowering of ECF Na concentration there is a fall in ECF osmotic pressure with fluid shift into the cells and fall in ECF volume. Renal conservation of sodium is normally highly efficient, therefore plasma Na concentration falls late. Symptoms are rare until plasma Na is less than 120 mmol/l. Stupor, muscular weakness, cramps and nausea occur; the patient is not necessarily thirsty. Eventually circulating volume falls with hypotension and peripheral circulatory failure. As plasma Na falls below 110 mmol/l, drowsiness progresses to coma with convulsions.

When *volume* replacement has also been inadequate, hyponatraemia is accompanied by dehydration with raised haematocrit, urea and plasma protein levels. Urinary osmolality, SG and urea are increased with urinary Na less than 10 mmol/l (unless renal tubular damage is present). Management is:

- replace electrolyte and fluid losses as closely as possible, for example plasma for burns, 0.9% saline for GI losses;
- replace losses slowly as rapid equilibrium across the blood–brain barrier may cause further CNS deterioration;
- avoid the use of hypertonic saline;
- steroids if adrenal insufficiency suspected.

Hyponatraemia with *normal* ECF volume can occur with excess ADH secretion (Ca bronchus, pneumonia, artificial ventilation, head injury, meningoencephalitis), psychogenic water intoxication or excessive consumption of beer. Management is that of the underlying condition, with water restriction.

When hyponatraemia occurs with *expansion* of ECF volume, total body Na may be considerably increased with water retention proportionately greater. Causes include cardiac failure, acute or chronic renal failure, nephrotic syndrome, liver failure with hypoproteinaemia. Oedema usually is present. Management is that of the underlying condition and includes salt and water restriction.

Sick cell syndrome. In very ill patients hyponatraemia may result from widespread increase in cell membrane permeability (from hypoxia, ATP depletion and endotoxins) with leakage of Na into the cells (and K out). There is an accompanying shift of water from ECF to ICF with nonpitting oedema of the limbs and trunk. Management includes:

- Administration of glucose and insulin (with added K unless plasma K is high).
- Steroids.
- Plasma expanders may be necessary to correct hypovolaemia and salt-poor albumin may be preferred to HAS (*see* 2.5). Administration of Na is dangerous and may further increase K loss from the cell.
- Recovery is associated with a shift of water back to the ECF and its excretion as a diuresis.

Hypernatraemia

Hypernatraemia usually reflects disproportionate water loss rather than Na gain. High oral intake of Na is unlikely to produce hypernatraemia because of intense thirst. Causes are:

- osmotic diuresis (diabetes, iatrogenic – mannitol, hyperglycaemia);
- in children with diarrhoea and a high solute intake (for example, unmodified cow's milk) hypernatraemic dehydration can result;
- diabetes insipidus (central/nephrogenic);
- iatrogenic Na overload (IV infusion, enteral feeding, TPN).

Management of hypernatraemia is by:

- treatment of the underlying cause;
- oral (or NG) rehydration with water; or, if not possible;
- *slow* IV 5% dextrose (or dextrose–saline or half-strength Darrows solution).

1.6 Potassium

A 70 kg adult contains approximately 3400 mmol K, with 90% in ICF and its distribution therefore quite different from that of Na. The concentration gradient of K determines electrical activity of the cell membrane and deviation from the normal range can affect nerve and muscle function.

Low plasma K may result from either:

- external losses, for example GI tract (diarrhoea, vomiting), renal (acidosis, diuretics, steroids), or

- transfer to ICF compartment *eg* with insulin-glucose, rise in pH, previous ICF K depletion.

Plasma K concentration can be misleading in assessing K deficiency and may be within normal range despite 40% deficiency of total body K. This is important in K deficient patients in whom surgery is planned. With uncorrected K deficiency the patient is at risk postoperatively as regards cardiac function and paralytic ileus.

Almost all K is excreted by the kidneys and K elimination is closely related to reabsorption of Na and H ions. *Potassium replacement* (*see* 8.4) can be monitored with urinary K levels. When these start to rise, this implies that administered K is not being retained and that the deficiency has been corrected.

1.7 Calcium and magnesium

Ca and Mg have electrochemical functions similar to those of Na and K; Ca is mainly an ECF and Mg an ICF cation. Although neither Ca nor Mg need be considered in short-term replacement of fluid and electrolytes, both become important in the context of IV feeding.

Calcium. The average adult contains 1000 g Ca of which 99% is stored in the skeleton. Adult intake is 10 mmol/day. Three major hormones regulate Ca: parathormone, 1,25-dihydroxyvitamin D and calcitonin. These hormones act on bone, kidney and gut to control Ca content of both plasma and the skeleton. The small exchangeable fraction plays a vital role in maintenance of neuromuscular function, especially cardiac muscle.

- Normal plasma Ca is 2.1 – 2.6 mmol/l. About half the plasma Ca exists as free ions (the physiologically active fraction), the rest bound mainly to albumin.
- Ionization is pH-dependent, increasing with drop in pH.
- In hypoalbuminaemic states, calculate 'corrected' Ca by adding 0.02 mmol/l for every g/l by which albumin is lower than 40 g/l (for example albumin 26 g/l, uncorrected Ca 1.92, corrected Ca 2.20 mmol/l).