INTRAVENOUS FLUID THERAPY IN PAEDIATRICS

GORDON H. BUSH D.M., F.F.A.R.C.S.

Consultant Paediatric Anaesthetist, Alder Hey Children's Hospital, Liverpool

INTRAVENOUS FLUID THERAPY in paediatrics comprises the administration of—

(*a*) water and electrolytes,

(*b*) blood,

(*c*) substances which will provide calorific intake to maintain nutritional requirements.

It is not the purpose of this paper to discuss the problems concerned with blood transfusion or so called 'intravenous alimentation' since these mainly arise in specialized units and are hence of limited general interest, whilst the infusion of water and electrolytes is a common procedure and of great practical importance. Yet it apparently appears to be complicated and blurred. Indeed there is evidence that even those entrusted with the care of infants and children do not fully comprehend some of the basic essentials of fluid requirements. Pledger and Buchan[1] investigated deaths in children with acute appendicitis during the five-year period 1963–67 and found that in 54 out of 146 children of all ages who died from this disease, and in 25 out of 51 children under five years of age, there was evidence of inadequate fluid replacement: this was defined as a child with peritonitis who—

(*a*) received no intravenous therapy.

(*b*) was given less than his normal daily requirements to treat dehydration,

(*c*) was given no intravenous colloids in the presence of oligaemic shock,

(*d*) had abnormal serum urea or electrolytes.

It is clear that inadequate knowledge of fluid therapy was an important cause contributing to the mortality in these cases.

The main reason for this lack of understanding would seem to be—

(*a*) Inadequate interpretation of signs of dehydration.

(*b*) Confusion as to the best method of calculating fluid requirements; that is to say whether this should be based on body weight, body surface area, or metabolic rate. Indeed advocates for all these methods can be found in different treatises on the subject. One even has a choice of at

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least 37 formulae for deriving surface area. The most acceptable method is one that is simple to derive, to remember and to apply.

(*c*) Lack of precise data of the effects of fluid deprivation and administration in infants and children since most studies of fluid requirements are either incomplete because of the technical and ethical problems involved or performed under abnormal circumstances. Hence the many and varied regimes advocated in standard texts.

(*d*) Failure of appreciation of the difference that exists between the adult and the child, and thus simple extrapolation is completely inadequate and also that renal function in infants is well adapted to their particular needs.

Distribution and maintenance of fluids and electrolytes

Water. In the newborn there is—

(*a*) A larger amount of total body fluid compared with the adult.

(*b*) A difference in the distribution in that extracellular fluid is proportionately greater in the newborn and infant (Fig. 1). This is related to a smaller proportion of cellular material in infants and this accounts for the variation of sodium and potassium contents. At birth per Kg body weight, the sodium content is 50% higher and the potassium content 20% lower than in adults.

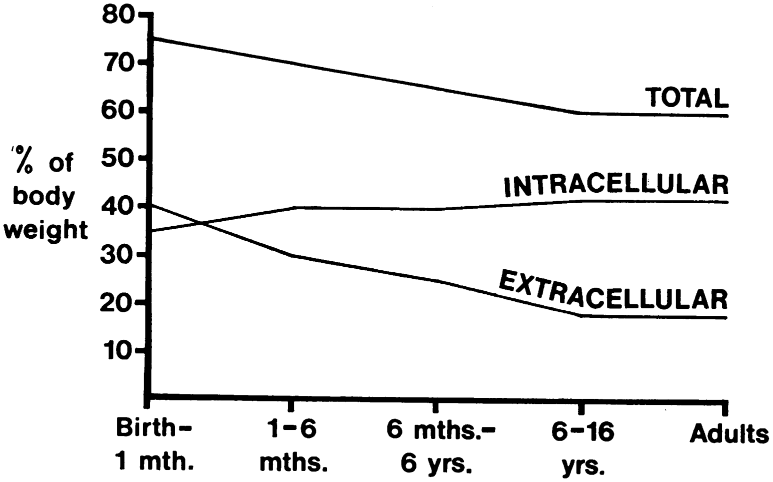

Fig. 1. Distribution of total, intracellular and extracellular water expressed as a percentage of body weight related to age.

Water balance

Extrarenal losses. 1. Insensible water loss is dependent upon—

(*a*) Energy consumption, since about 25% of total body heat production may be dissipated as insensible water loss, though increased activity and fever may increase the amount of fluid lost.

(*b*) Temperature of environment; this is of particular importance in the tropics where increased losses may be anticipated, whilst in infants nursed at a neutral temperature metabolic demands are reduced.

(*c*) Humidity of ambient air which again in the case of infants nursed in a high ambient humidity will lead to less insensible loss.

(*d*) Surface area; in infants there is a large area in relation to body weight and hence a greater area for evaporation of fluids.

2. Loss of fluids in sweat relatively unimportant in temperate climates.
3. Faecal loss which is small except in those cases with diarrhoea.

Renal losses. These vary according to the age and are related to the osmotic load of solutes to be excreted.

Intake of water is derived from oral fluid, which is by far the greatest source of fluid, and only a small proportion is supplied by water of oxidation and from ingested solids.

Methods of calculating fluid maintenance requirements

Any method of calculating fluid maintenance requirements must be related to energy metabolism since insensible loss and urinary loss of fluid roughly parallel oxygen consumption. It has been calculated by Holiday and Segar[2] that insensible loss approximates 50 ml./100 cals./24 hr.

Urinary loss approximates	65 ml./100 cals./24 hr.
	115 ml./100 cals./24 hr.
Water of oxidation accounts for some	15 ml./100 cals./24 hr.
This leaves a total to be supplied of	100 ml./100 cals./24 hr.

Expenditure of energy may be related to body weight by assuming that in the weight range 0–10 Kg. the calorie expenditure is 100 cals./Kg./24 hr., but between 11 and 20 Kg. it falls to 1,000 cals. + 50 cals./Kg. over 11 Kg./24 hr. and for 20 Kg. upwards it is 1,500 cals. + 20 cals./Kg. over 20 Kg./24 hr. As it is computed that 100 ml. of fluid are required per 100 cals. extended for 24 hr., fluid requirements may be calculated on a body-weight basis. Thus between 0–10 Kg. the fluid requirements are 100 ml./Kg./24 hr., for infants between 11 and 20 Kg. 1,000 ml. + 50 ml./Kg. over 11 Kg./24 hr., and for all other children 1,500 ml. + 20 ml./Kg. over 20 Kg./24 hr.

This method of calculation of fluid requirements is simple, easy to remember and clinically acceptable. Proponents advocating surface area as a more satisfactory parameter for calculating drug dosage accept that particularly in dehydration body-weight measurements are more meaningful[3]. Indeed the principle of body surface area as a basis of parenteral fluid dosage has been criticized on the grounds of lack of validity of estimation, failure of correlation with physiological activity and inability to verify a constant relationship with metabolism[4].

A number of charts[5], [6] are available correlating fluid and electrolyte requirements to body weight, and whilst these may be valuable when they can be exposed for all to see, it is better to have an easily remembered formula.

Sodium

Depletion may be —

(*a*) Real—produced by loss of body fluids which are replaced by water. This may arise following the administration of N/5 saline to correct dehydration in the mistaken belief that the infant's kidney cannot concentrate sodium in the urine and lead to water intoxication, coma and convulsions.

(*b*) Apparent—where excessive water is administered without previous sodium loss.

Excess may be due to—

(*a*) Real—following excessive intravenous administration of sodium, e.g. when sodium bicarbonate has been given as a result of pH measurements performed on capillary blood obtained in conditions of circulatory shutdown.

(*b*) Apparent—where there is a disproportionate loss of water, e.g. in those conditions producing hypernatraemia, e.g. diarrhoea, fever.

Maintenance sodium requirements may be calculated according to the metabolic needs and these are 3 mEq./100 cals./24 hr. Thus in the range 1–10 Kg. the requirements are 3 mEq./Kg./24 hr., in the range 11–20 Kg., 30 + 1.5 mEq./Kg. over 10 Kg./24 hr. and in excess of 21 Kg. 45 + 0.6 mEq./Kg. over 20 Kg./24 hr. The requirements for chloride are similar.

Potassium

This ion mainly occurs intracellularly. It may be lost from the body in —

(*a*) Proportionate loss of potassium and nitrogen, e.g. starvation or injury producing cellular destruction.

(*b*) Disproportionate loss of potassium greater than that of nitrogen which occurs in intracellular dehydration, with loss of potassium and water producing symptoms of thirst and oliguria. Loss of potassium from the cells is replaced by sodium and hydrogen which prevents loss of water from the cells but causes a reduction in extracellular volume. Presence of $H+$ causes an intracellular acidosis and an extracellular alkalosis. Hence persistent extracellular alkalosis requires adequate potassium infusions. The maintenance potassium requirements may be calculated from the formula of 2 mEq./100 cals./24 hr. Thus in the range 1–10 Kg. the requirements are 2 mEq./Kg./24 hr., in the range 11–20 Kg. 20 + 1 mEq./Kg. over 10 Kg./24 hr. and over 20 Kg. 30 + 0.4 mEq./Kg. over 20 Kg./24 hr.

Alterations in disease

Dehydration may be either—

Primary affecting intracellular constituents, i.e. potassium and water.

Secondary affecting extracellular, i.e. sodium and water.

Circulatory disturbances are a marked feature of secondary dehydration. The commonest causes of dehydration are vomiting and diarrhoea. Both cause losses in proportion to the constituents of the intestinal secretions and it must always be remembered that fluids and electrolytes may be lost into the bowel lumen and thus lost to the body compartments and yet their loss not calculated in the total fluid requirements. Since the K+ concentration is higher than that in serum, a disproportionate loss of K+ will occur.

The diagnosis of dehydration depends on the type and severity (Table I). Of particular value are the observed loss of weight which may be substantiated from previous weight records, blood pressure, pulse, state of peripheral circulation, urine output, moisteners of mucous membranes, skin turgidity, serum electrolytes and blood urea estimations. Haematocrit estimates are unreliable due to the large variation in haemoglobin that occurs in infants and young children.

TABLE I

CLINICAL FEATURES OF DEHYDRATION

Mild	Irritability. Thirst. Dry mouth. Flushed warm dry skin. Fontanelle soft. Oliguria.
Moderate	Restlessness. Anxious expression. Very dry tongue and mouth. Eyes sunken. Fontanelle depressed. Marked oliguria. Raised temperature. Skin elasticity reduced.
Severe	Apathetic unresponsive. Hypotonia. Extreme dryness of mouth and tongue. Eyes staring and sunken. Fontanelle markedly depressed. Rapid feeble pulse. Cyanosis of extremities. Marked loss of skin elasticity. Pale cold skin. Fever.

The volume of fluid required to correct dehydration is dependent upon the extent of dehydration (Table II). Maintenance fluids must be added in calculating the total fluid requirements. In the treatment of primary dehydration there is need for water and potassium and this may be given in the form of N/5 saline with added potassium, whilst in the treatment of secondary dehydration the low plasma volume must be first corrected by the administration of plasma followed by saline and once the urine flow is established by a high potassium containing solution such as Darrow's solution. The importance of giving adequate potassium cannot be stressed too highly as deficiency of this ion is difficult to measure and invariably greater than suspected.

Repeated estimation will ensure that treatment is proceeding along the correct line and blood urea and electrolytes are of great value in assessing what further solutions should be required. Urine output is a good guide to the adequacy of the administered fluid.

Metabolic response to surgery

In adults there is a well defined metabolic response to trauma including surgery. This is shown by—

(*a*) cellular breakdown resulting in increase in urinary nitrogen and potassium,
(*b*) mobilization of energy stores,
(*c*) release of adrenal cortical hormones,
(*d*) release of antidiuretic hormone,
(*e*) change in renal function.

In severe trauma in infants and children a similar pattern is seen and interestingly enough is also seen in infants with marked respiratory distress. In infants and children undergoing mild to moderate surgery these responses are absent.

Cellular changes. Nitrogen balance studies carried out in infants and children undergoing surgical procedures[7] showed no evidence of increased urinary nitrogen following starvation, general anaesthesia or a moderate degree of surgical trauma. If anything, urinary nitrogen tended to diminish in the postoperative period. Only following major surgical procedures did urinary nitrogen increase. In these instances intravenous protein hydrolysate and carbohydrate in the form of 5% glucose or fructose prevented a negative nitrogen balance.

TABLE II

CLINICAL ASSESSMENT OF DEHYDRATION

	Mild	*Moderate*	*Severe*
Fluid loss expressed as percentage of total body water	6	9	12
Reduction in weight loss per cent resulting from fluid loss	5	7.5	10
Fluid deficit expressed as percentage of extracellular volume	15	22.5	30
Volume of fluid per kilogram body weight required to correct dehydration	50	75	100
Amount of sodium and chloride expressed as mEq./Kg. required to correct pre-existing loss	5	7.5	10
Amount of potassium expressed as mEq./Kg. required to correct pre-existing loss	3	4.5	6

Mobilization of energy stores In the postoperative period, glycogen stores only provide a small contribution to energy requirements and decreased nitrogen excretion such as already mentioned suggests that there may be sparing of protein breakdown. The only other source of energy is storage triglycerides, which can be metabolized to glycerol phosphate and free fatty acid. Dibbin and Kiesewetter[8] found that there was an increase in free fatty acid in infants undergoing herniorraphy without significant elevation of catecholamines. This was interpreted as the mobilization of triglycerides to provide a preferential source of energy in the absence of stress as shown by the absence of elevation of catecholamines. There is, however, some doubt whether catecholamines can be correlated with the degree of stress present.

Hormonal changes. Changes in protein metabolism suggests that cortisol production is less in infants and children undergoing mild to moderate stress, but this requires verification.

Aldosterone levels are normal in infants greater than seven days and a rise in response to sodium deprivation indicates that autoregulation is intact at this age. That such a mechanism exists can, of course, be inferred from normal stress responses of infants and children undergoing major surgical procedures.

A.D.H. is usually regarded as being under the control of osmoreceptors, but it is now apparent that volume reduction of body fluids and surgical procedures also play a part in release of A.D.H. It has been noted that A.D.H. levels prior to the induction of anaesthesia in adults are elevated but are rapidly reduced to normal following the infusion of fluid and the restoration of body fluid volumes.

Renal function. In adults free water clearance is depressed, urinary sodium concentration diminished due to greater tubular resorption, and this results in low urine volume with low sodium concentrations. It is suggested that these changes are aimed at the maintenance of normal internal fluid volumes.

In older children with severe trauma similar effects are seen, but the infant and young child seems to respond to trauma without these effects. It has been suggested that the state of hydration at the time of surgery influences the urine output postoperatively. Calcagno, Rubin and Singh[9] found that in non-surgical patients a water load of 30 ml./Kg. of 2.5% dextrose water in 10 minutes resulted in greater excretion in the well hydrated than in the dehydrated patient (i.e. those who had been given nothing for 15 hours). In surgical patients, that is infants less than one year old, the water load was not excreted in patients not given fluid during operation, but in patients who were given intravenous fluids during operation 84–98% was excreted in 2 hours following the water loading.

Rubin, Calcagno, Mukherji and Singh[10] studied the effect of sodium loading in infants up to 14 months of age. A sodium load of approximately 15 mEq./Kg. was given as 3% sodium chloride over a 30-minute period and the sodium excretion over the next 4 hours was measured. Surgical cases were studied between 12 and 24 hours postoperatively. Non-surgical cases showed that there was a mean excretion of 6.7 mEq./Kg./4 hr. whilst in surgical patients this amounted to 5.6 mEq./Kg./24 hr. There was thus no serious degree of sodium retention in the immediate postoperative period in these surgical patients.

Intra-operative requirements

Evidence concerning the sequestration of fluid in the traumatized area following surgery has come from a number of sources.

In children there have been very few studies that give any clear conclusion as to the extent that intra-operative fluids should be administered. Viguera[11] administered Ringer lactate in sufficient quantity during surgery to maintain the urine volume and concentration as determined by specific gravity at levels which were deemed normal for each child. In some instances this volume infused amounted to 20 ml./Kg./24 hr. of surgery. No adverse effects were seen and the procedure was deemed to be entirely beneficial.

Bennett, Dougherty and Jenkins[12] administered 8 ml./Kg./hr. of Ringer lactate solution during surgery in neonates undergoing operation, and during the postoperative period administered N/5 saline at the rate of 100 ml./Kg./hr. except when there was evidence of hyponatraemia when increased amounts of sodium were given. The urine volumes were maintained and, in spite of a large amount of sodium, hyponatraemia was not an uncommon finding.

Considerable difficulty arises as to the extent of sequestration of fluid and one also has to bear in mind the dehydration inevitably incurred as a result of limiting fluid intake before surgery. Thus a 6-hour deprivation of fluid will immediately produce a deficit of 25 ml./Kg. in a 5 Kg. infant, which is equivalent to a 16-hour period of fluid deprivation in the adult. In view of this evidence and the fact that, except in major trauma, there is little metabolic response to surgery, it would appear that there should be a definitive policy towards intra-operative fluid administration. Taking all these considerations into account it would seem logical and necessary to administer up to 10 ml./Kg./hr. of N/2 saline in 5% dextrose during surgery. This amount, except in operations of marathon length, would never exceed the expected normal daily requirements, and hence could not lead to overloading with water and electrolytes.

Neonatal fluid requirements

Renal function in the neonate has often been regarded as highly immature and on this basis intravenous fluid administration has been deemed to be extremely hazardous. The greatest difficulty lies in determining the extent of renal inefficiency in the newborn. Thus when compared on a body surface area basis, the clearance of inulin and para-aminohippurate are only about one-tenth that of adult values in infants shortly after birth and reach half adult levels at three months of age. It is extremely unlikely that the normal newborn could have renal function impaired to such a degree and it has been suggested that a better method of standard of comparison would be the relationship of clearance value to volume of body water. Recalculation of the data on this basis shows that the kidney function approximates adult levels within a few weeks of birth. When considered in terms of the normal solute load that is to be excreted in the newborn and young infant the renal function appears remarkably efficient.

Due to the large daily water turnover in the infant, which may amount to as much as one-half of the extracellular volume compared with one-seventh in the adult, slight disturbances of fluid intake can rapidly cause severe internal changes of fluid. The inability to excrete acid radicals and a highly concentrated urine results in a rise in blood urea and metabolic acidosis.

Normally in the first few days of life urine volume is 25 ml./day and this rises to 120 ml./day by the end of the first week. The osmolality falls from 400–500 mOsm./l. to about 100 mOsm./l. These values of course apply to the normal infant with a progressive intake of fluid and it would be wrong to apply these figures to a sick infant undergoing surgery. Bennett, Dougherty and Jenkins[12] found that administering fluid at a rate of 100 ml./Kg./24 hr. resulted in a urine output in excess of 100 ml. per day, without evidence of extracellular overhydration. Others have suggested that, in view of the difficulty in excreting a water load in a given time, fluid administration should be less than in older infants, and a figure of 50 ml./Kg./24 hr. would seem to be reasonable for the first few days of life.

The quantity of electrolytes to be administered must depend to a large extent on the degree of losses prior to operation, and the continued losses postoperatively. Basically N/5 saline will provide adequate requirements, though with extensive gastric losses normal saline should be given. Repeated electrolyte determinations will govern the type of fluid to be given. In view of the tendency of the premature infant to hypoglycaemia, blood sugar levels should be checked repeatedly and, if necessary, 10% dextrose administered. Calcium levels should also be measured and, if low, intravenous 10% calcium gluconate (1–2 ml.) should be given. Hypomagnesaemia may also occur and lead to prolonged epileptiform seizures. It is obvious that when dealing with the newborn, meticulous attention to detail is essential.

Acid base changes

These frequently accompany severe disturbances of water and electrolyte balance and require the appropriate treatment. It should be remembered that persistent alkalosis is frequently an expression of extensive intracellular potassium loss.

The aim of fluid therapy should be to place at the disposal of the regulating systems a quantity of fluid and electrolytes based on a thorough clinical and biochemical evaluation, and to leave the final adjustment of these quantities to the normal mechanisms of the body.

Guide lines of treatment can only be based on average requirements, and repeated reappraisals and, if necessary, alterations in treatment are essential for the correct management of fluid therapy in children.

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Courses and Symposia (Faculty of Anaesthetists)

Readers are asked to note that the Faculty will be holding a Course on Anaesthetics from 4th to 22nd October 1971. Further details may be obtained from the Secretary of the Faculty at the College.

A two-day symposium on The Anaesthetist and the Lung will be held in the Royal College of Surgeons of England, Lincoln's Inn Fields, London, WC2A 3PN, on 4th and 5th November 1971.

Those participating include Professor H. H. Loeschcke, Dr. J. W. Severinghaus, Dr. J. G. Whitwam, Dr. J. M. B. Hughes, Dr. A. B. Baker, Dr. J. B. Forrest, Dr. G. H. Hulands, Professor M. B. Laver, Dr. C. Prys-Roberts, Professor M. K. Sykes, Dr. C. Michell, Professor L. B. Strang, Professor B. E. Marshall.

Further details of this Symposium are obtainable from the Secretary, Faculty of Anaesthetists, Royal College of Surgeons of England, Lincoln's Inn Fields, London WC2A 3PN.

Other Courses and Symposia

Plastics in Medicine and Surgery. A Symposium is to be held in the Physics Building, University of Newcastle-upon-Tyne, 15th and 16th September 1971. Details from A. J. Harrow, The Medical School, The University, Newcastle-upon-Tyne, NE1 7RU.