Maintenance fluid therapy

What routine intravenous maintenance fluids should be used?

N P Mann

An introduction to the debate

Intravenous maintenance fluid is widely used in general paediatric practice and more children who come into hospital receive intravenous fluid than in the past. The intravenous route is frequently used because enteral maintenance or rehydration treatment is more labour intensive and uses valuable staff time; furthermore modern pumps for delivery of fluids are safe. Nevertheless in developing countries the enteral route is still more widely used even for sick dehydrated children.

Are there are any dangers of intravenous fluids? Clearly there is a possibility of miscalculation of infusion rates and also the potential for mistakes in terms of dosing errors with additives. It has been widely recognised in recent years that there is a high incidence of hyponatraemia in children treated with intravenous maintenance fluids. Is this because of excessive water or too little salt?

Moritz and Ayus discussed the high frequency of hyponatraemia in these children in their paper in *Pediatrics* in February 2003.[1] They suggested the use of isotonic saline rather than use of hypotonic fluids for maintenance therapy. More than 20 years ago there were concerns about profound neonatal hyponatraemia causing neurological problems in infants as the result of either excessive or the wrong kind of fluid given to mothers during labour.[2]

It is therefore timely to revisit this problem. Two experts have been asked to give their views to encourage further debate (see accompanying articles[3 4]). Do write to *ADC* with your comments about how paediatric practice in this area can be improved.

Arch Dis Child 2004;**89**:411

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Maintenance fluid therapy

Pouring salt on troubled waters

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The case for isotonic parenteral maintenance solution

Intravenous fluid and electrolyte therapy for acutely ill children has been a cornerstone of medical practice for well over 50 years. The scientific methodology behind fluid regimens generated much debate in the early 1950s following the pioneering work of Darrow, Talbot, Gamble and others who recognised the important relation between caloric expenditure and requirements for water.[1–3]

Caloric expenditure was originally calculated according to body surface area, which at the bedside required either tables or nomograms.[1] In 1957 Holliday and Segar simplified this approach, relating energy expenditure to one of three weight based categories (<10 kg, 10–20 kg, >20 kg).[4] Electrolyte requirements were also calculated on a weight basis, producing an "ideal", hypotonic solution comprising 0.2% saline in 5% dextrose water (0.18% saline in 4% dextrose in the United Kingdom). This simple regime was subsequently adopted on a global scale and is recommended in current paediatric and medical textbooks.

Advances in our understanding of water and electrolyte handling in health and disease have called into question the validity of the Holliday and Segar approach. Specifically, many authors have reported how hypotonic maintenance fluid may result in iatrogenic hyponatraemia in hospitalised patients, often with devastating consequences.[5–10] In this article we re-evaluate each of the concepts on which this traditional regime is based (energy expenditure, and water and electrolytes requirements) and use this to make the case for an alternative, namely isotonic fluid.

PITFALLS OF THE WEIGHT BASED HOLLIDAY AND SEGAR APPROACH

Energy expenditure

Talbot originally estimated basal metabolic rate in children based on water loss.[11] Crawford extended this concept, by presenting *total* energy requirements (basal metabolic rate plus growth and activity) using this data in relation to body surface area (fig 1). Holliday and Segar further advanced this by indexing energy expenditure to body weight rather than surface area, assuming 1 ml of water loss was associated with the fixed consumption of 1 kilocalorie.[4] The typical fluid losses for children (table 1) thus equate with an energy requirement of 120 kcal/kg/day for a 10 kg child.[12]

There are two main flaws with this approach. First, it is now known that resting energy expenditure is closely related to *fat free mass* which includes muscle and the four major metabolic organs (heart, liver, kidneys, and brain).[13] Eighty per cent of the resting energy expenditure is accounted for by these four organs which comprise only 7% of total body mass. As a result, the use of weight alone to calculate energy expenditure may significantly overestimate caloric requirements. On average, the weight based method overestimates energy requirements in infants by 14% compared to the surface area method (fig 1).[4] Second, energy expenditure in healthy children, on whom historic models are based, is vastly different in acute disease or following surgery. Using calorimetric methods, energy expenditure in these patients is closer to the basal metabolic rate proposed by Talbot, averaging 50–60 kcal/kg/day.[14–16] This overestimate is multifactorial: ill

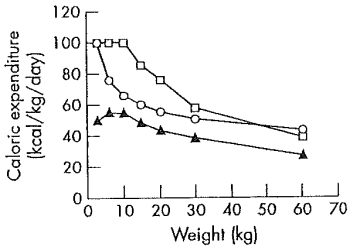

Figure 1 Daily caloric expenditure according to the weight based method of Holliday and Segar and by surface area method of Crawford, and basal metabolic rate. Comparison of two different methods for calculating caloric expenditure across weight ranges (open squares = Holliday and Segar's weight based method; open circles = Crawford's surface area method*; referenced against basal metabolic rate**).

patients are catabolic, often relatively inactive, and, in the intensive care environment may be pharmacologically sedated or muscle relaxed.[14–17] Almost half of the caloric intake suggested by Holliday and Segar is designated for growth, an unrealistic goal in acute disease.[16] Although fever and sepsis per se may increase metabolic rate this is usually limited to less than 1.5 times the Basal metabolic rate, burns being an exception.

Water requirements

Historically water requirements have been based on crude estimates of both insensible (skin, respiratory tract) and sensible (urine and stool) water losses.

Insensible water loss

This was generously estimated at 930 ml/m^2/day (27 ml/kg/day).[18] Recent data suggest the true figure may be only half of this, with basal insensible losses from the skin being 250 ml/m^2/day (7 ml/kg/day) and via the respiratory tract 170 ml/m^2/day (5 ml/kg/day).[19] Additionally many other risk factors may reduce insensible water loss such as use of humidifiers in ventilated patients (80% reduction in respiratory water loss) or a thermo neutral environment.[17] Bluemle *et al* have shown insensible water losses of as little as 330 ml/m^2/day (10 ml/kg/day) in catabolic acute renal failure patients.[20]

*Crawford calculated caloric expenditure based on the calories utilised per surface area of the body. The calculated caloric expenditure at each body surface area increment can be converted to weight by cross-referencing surface area to weight using standard growth charts. The ratio of weight to surface area rapidly declines from birth to 10 kg. The Holliday and Segar method does not take this into account.

**From the data of Talbot.

Table 1 Typical water losses per 100 kilocalories (kcal) of energy expended for a healthy 10 kg child

Source of water loss	Estimated water loss (ml per 100 kcal/day)
Insensible	
Skin	30
Respiratory	15
Sensible	
Stool	10
Minimal sweating	10
Urine	50
Total	115

Data calculated according to Pickering and Winters.[12]

Urinary loss of water

According to Holliday and Segar, urinary water losses for healthy children amount to 50–60 ml/kg/day[4] based on the work of Pickering and Winters (table 1).[12] The basis of this fluid regime was the observation that 15/28 infants and 20/25 children (unspecified diagnoses) who were given intravenous dextrose produced urine with an "acceptable" urine osmolarity between 150 and 600 mosm/l H_2O.[4] They presumed patients with dilute urine received too much water and conversely those with concentrated urine too little water.

Today we recognise this does not take into account the overriding influence of antidiuretic hormone (ADH) on urine flow rate.[21] When ADH is present, the renal solute load is effectively excreted in a smaller urine volume producing concentrated urine. Under these conditions urine output is often less than half the values observed in healthy children (approximately 25 ml/kg/day).[22] An increase in ADH is common during many childhood diseases, in response to stress (pain, fever, surgery) or secondary to use of opiates and non-steroidal anti-inflammatory drugs.[23–25] Under these conditions the administration of free water frequently leads to hyponatraemia because the kidneys are unable to excrete the water load.[5 6 26] Interestingly, the type of fluid administered may influence ADH levels. Judd *et al* showed that 0.9% saline but not 5% dextrose reduced ADH concentrations postoperatively.[21]

Thus the total fluid loss (sensible plus insensible) during acutely illness or following surgery may amount to approximately half that suggested by Holliday and Segar (50–60 ml/kg/day).[5 6] Also, the often overlooked production of endogenous water from tissue catabolism (water of oxidation) may be increased in acute disease.[20] In healthy children, this has been estimated to be 15 ml/100 kcal burnt.[4] Thus, all these factors need consideration when assessing overall water balance.

Electrolyte requirements

In healthy breast fed infants Holliday and Segar computed a dietary sodium intake of 1 mEq/100 calories per day.[4] Darrow recommended 3 mEq of sodium per 100 calories of energy expended per day.[4] This is based on urinary excretion rates of sodium in healthy, milk fed infants. However, daily electrolyte requirements in disease may differ considerably from this. For example, large urinary losses of sodium and potassium may occur through the phenomenon of desalination.[27 28] Furthermore, Al-Dahhan *et al* showed a beneficial effect on neurodevelopmental outcome from doubling the daily sodium intake (4 to 5 mmol/kg) in neonates.[26] This refutes the assumption that the neonatal kidney is incapable of "handling" a high sodium load. The recent discovery of the most potent natriuretic hormones, urodilatin and gut-related natriuretic peptide has also shed new light on sodium regulation.

The rationale behind the traditional approach is to balance sodium intake to match sodium loss. However, this fails to appreciate the *single* most important role of sodium in acute illness, namely maintenance of plasma tonicity.[25 29] There is a strong inverse relation between plasma sodium concentration and intracellular volume.[30] Cell membranes are permeable to water but not electrolytes. As sodium is the major extracellular cation (and hence osmole), it regulates the movement of water across cells along an osmotic concentration gradient, thus explaining cellular swelling in the presence of hyponatraemia.

It is also important to recognise the role of potassium in the regulation of tonicity balance. Potassium is a major intracellular osmole, and may directly influence extracellular sodium concentration by altering the distribution of water between fluid compartments.[25] Potassium loss, both urinary and stool, may be significant in disease; yet its direct influence on serum sodium concentration is often not considered.[25 28]

Tonicity of intravenous fluids

It is crucial that clinicians appreciate the difference between osmolarity and tonicity. The osmolarity of a solution is the number of osmoles of solute per litre of solution. The tonicity of a solution refers to the total concentration of solutes that exert an osmotic force across a membrane in vivo. For example, 5% dextrose has the same osmolarity as plasma (286 mosm/l H_2O) but is rapidly metabolised in blood to water. Thus its *in vivo tonicity*

Table 2 Approximate sodium concentration, in vitro osmolarity, in vivo tonicity, and theoretical volume of electrolyte free water (EFW) provided by commonly used intravenous solutions

Intravenous solution	Sodium* (mmol/l)	In vitro osmolarity† (mOsm/l H_2O)	In vivo tonicity‡ (mOsm/l H_2O)	Volume of EFW§ per litre infused
5% dextrose	0	286	0	1000
0.18% saline in 4% dextrose	30	300	60	824
0.45% saline	75	150	154	500
0.45% saline in 5% dextrose	75	432	150	500
0.9% saline	154	308	308	0
0.9% saline in 5% dextrose	154	586	308	0

*The apparent discrepancy between the in vitro sodium concentration (0.9% saline) of 154 mmol/l and the in vivo plasma sodium of 144 mmol/l is due to the phenomenon of pseudohyponatraemia. In human plasma, approximately 7% of the plasma volume is occupied by albumin and lipid, falsely lowering the true sodium concentration plasma by 10 mmol/l (7% of 155).
†In vitro osmolarity refers to the number of osmoles of solute per litre of solution.
‡In vivo tonicity refers to the total concentration of solutes which exert an osmotic force across a membrane in vivo (excludes the osmotic effect of dextrose because it is rapidly metabolised in blood).
§Calculated on the basis that electrolyte free water distributes to the intracellular and extracellular space in a ratio of 2:1.

is equal to that of electrolyte free water, as it contains no salt or other active osmole (zero tonicity). Every litre of 5% dextrose infused results in the expansion of the intracellular and extracellular fluid space by one litre (two thirds of this distributes to the intracellular space and one third to the extracellular space). Similarly, for every litre of 0.18% saline in 4% dextrose water infused, only 1/5th (200 ml) is isotonic to plasma (table 2). The remaining 800 ml is electrolyte free water, which will expand the intracellular fluid compartment. This is particularly relevant if excretion of water is limited by ADH.[5–7 21 28 31] This fluid shift may even occur in the absence of hyponatraemia.[32] Small increases in tissue water through the use of hypotonic fluids may be harmful in conditions such as cerebral oedema where minor increases in cerebral water may lead to disproportionately large increases in intracranial pressure.

The incidence and neurological complications of acute hyponatraemia

Hyponatraemia is a common biochemical finding in hospitalised children and is most commonly due to excess water intake rather than salt loss.[6 7 22 23] Shann and Germer showed an incidence of hyponatraemia (Na <134 mmol/l) as high as 45% in hospitalised children with pneumonia and 50% in bacterial meningitis.[8] Hanna *et al* recently reported a 30% incidence of admission hyponatraemia in infants with bronchiolitis requiring intensive care admission in the United Kingdom, 13% of which had seizures.[9] Halberthal *et al* was able to show a direct link between hyponatraemia and the use of hypotonic maintenance fluid.[7] The neurological complications of acute hyponatraemia include encephalopathy with seizures, irreversible brain damage, or brain death from cerebral herniation.[5–10] Children are also among the most susceptible to hyponatraemic brain injury.[5 6] Fatal hyponatraemia can occur within hours of hypotonic fluid administration, particularly if standard fluid maintenance rates are used (100–120 ml/kg/day).[10]

THE RATIONALE FOR ISOTONIC MAINTENANCE FLUID

The paramount consideration in the choice of intravenous fluid is the requirement to maintain serum sodium at a normal level. The use of isotonic solutions such as 0.9% saline is more appropriate in acutely sick children as they do not theoretically expand the intracellular fluid space. Isotonic solutions preserve intracellular function and integrity, by minimising changes in plasma sodium concentration and tonicity.

Use of 0.9% saline as maintenance fluid, if combined with appropriate fluid restriction, will result in a two to threefold increase in daily sodium intake compared to the traditional regime. However, the concern that this may cause severe hypernatraemia is without foundation because the sodium concentration and tonicity of 0.9% saline is similar to plasma. Andersen *et al* showed a rise in plasma sodium only after intravenous administration of hypertonic 3% saline but not 0.9% saline, despite a temporary positive sodium balance.[33] Heer *et al* showed chronic sodium loading in volunteers does not produce an increase in plasma sodium, body water, or weight as previously suggested.[34] Many of the historical assumptions concerning sodium handling are based on salt depleted subjects. Indeed massive sodium loads from large volume resuscitation of infants and children with sepsis (80–180 ml/kg/day) using 0.9% saline did not produce hypernatraemia.[35] Additionally an epidemic of hypernatraemia has not been documented in hospitalised adults where isotonic maintenance fluids are routine. When present, the aetiology of hypernatraemia in this scenario is frequently due to well recognised factors such diabetis insipidus or over-use of loop diuretics.[36]

The debate as to the optimal isotonic fluid is ongoing. For example, Hartman's solution has a more physiological concentration of chloride than 0.9% saline and hence does not cause hyperchloraemia. The benefit of Hartman's solution versus 0.9% saline is not currently known. It is important to stress that dextrose may be added to these isotonic solutions (commonly in concentration of 5–10%), when clinically indicated to avoid hypoglycaemia without changing the solution's in vivo *tonicity* (table 2). Recent evidence suggests that a 1% dextrose solution following uncomplicated paediatric surgery may be adequate.[37] A suitable solution for neonates and infants is 0.9% saline in 5% dextrose water, which is commercially available. We advocate 0.9% saline (with or without added dextrose) as a safe maintenance solution, both perioperatively and in the acute phase of most childhood illnesses requiring hospitalisation (for example, pneumonia, bronchiolitis, and meningitis). Here, the water retaining effect of antidiuretic hormone may necessitate a moderate degree of fluid restriction (50–60%) to prevent fluid overload. The concept of fluid maintenance should not be confused with replacement therapy where abnormal or excessive quantities of water and electrolytes may be lost. In this instance the biochemical composition and tonicity of the replacement solution should approximate that which is lost.

CONCLUSION

We have shown a number of pitfalls in the Holliday and Segar approach to parenteral therapy, namely that it focuses on fluid and electrolyte requirements for healthy children. In acute disease or following surgery, caloric expenditure, insensible water losses, and urine output are frequently much less than in health (often 50–60% of the reference values). Furthermore, this approach fails to recognise the importance of tonicity with its central role in the distribution of water between fluid

compartments (intracellular and extracellular space).

We therefore agree with Moritz and Ayus who advocate isotonic solutions such as 0.9% saline for routine fluid maintenance in children.[38] Hypotonic solutions, such as 0.18% or even 0.45% saline, are potentially dangerous when renal water excretion is limited by ADH. This raises a significant ethical barrier to conducting a randomised control study as most acutely ill or postoperative patients have increased ADH levels. There are few occasions in medicine where mortality could be reduced by a task as simple as changing from a hypotonic maintenance solution to an isotonic one.

ACKNOWLEDGEMENTS

We would like to thank Dr Shane Tibby for his assistance in preparation of this manuscript.

Arch Dis Child 2004;**89**:411–414.
doi: 10.1136/adc.2003.045302

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Maintenance fluid therapy

Rubbing salt in the wound

M Hatherill

The case against isotonic parenteral maintenance solution

In a recent review, Moritz and Ayus have suggested that isotonic parenteral maintenance solution (PMS) should be used to prevent hospital acquired hyponatraemia in children.[1] Hospital acquired hyponatraemia may be exacerbated by non-osmotic production of antidiuretic hormone (ADH) associated with conditions such as bronchiolitis (33%), pneumonia (31% and 45%), bacterial meningitis (50%), and postoperative pain or nausea.[2–7] Although it has been termed a syndrome of inappropriate antidiuretic hormone secretion (SIADH), it may be more accurate to refer to non-osmotic ADH production, since haemodynamic baroreceptor stimuli, such as hypovolaemia, may be physiologically appropriate despite the adverse effect on sodium.[1 6 7]

The reported morbidity and mortality associated with hospital acquired hyponatraemia have given momentum to calls for increasing the tonicity of PMS.[1–3 8–11] Implicit in such proposals are the assumptions that hyponatraemia results from a net sodium deficit, exacerbated by hypotonic PMS, and that this sodium deficit may be avoided by using an isotonic solution.[1–3 8–11] Therefore, if we contemplate a change in practice, we must consider whether

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