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.

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Maintenance fluid therapy

Pouring salt on troubled waters

D Taylor, A Durward

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

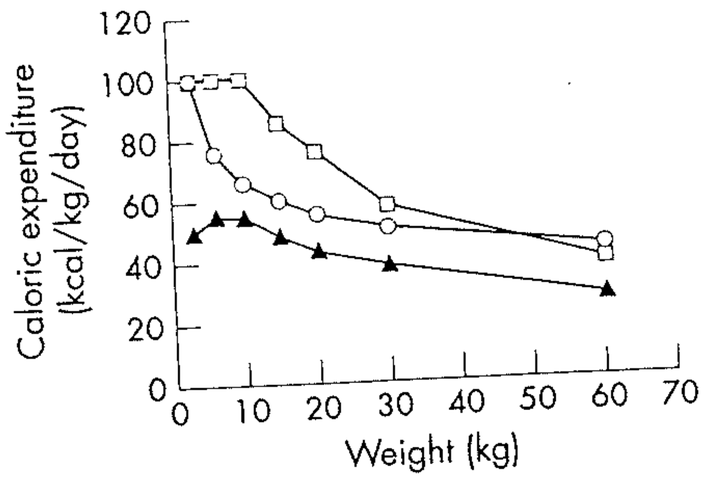

Figure 1 Daily caloric expenditure according to the weight based method of Holliday and Segar and by surface area method of Crawford, and basal metabolic rate. Comparison of two different methods for calculating caloric expenditure across weight ranges (open squares = Holliday and Segar's weight based 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 (280 mosm/l H_2O) but is rapidly metabolised in blood to water. Thus its *in vivo* tonicit

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 tracranial 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.[17] 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.

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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 b more accurate to refer to non-osmot ADH production, since haemodynam baroreceptor stimuli, such as hypov laemia, may be physiologically appr priate despite the adverse effect sodium.[1 6 7]

The reported morbidity and mortali associated with hospital acquired hyp natraemia have given momentum calls for increasing the tonicity PMS.[1–3 8–11] Implicit in such propos are the assumptions that hyponatraem results from a net sodium defic exacerbated by hypotonic PMS, and th this sodium deficit may be avoided using an isotonic solution.[1–3] Therefore, if we contemplate a chan in practice, we must consider whetl

Theoretical effects of variation in the volume of PMS

Both examples apply to a 10 kg child with non-osmotic ADH production, TBW = 6 l, estimated isotonic (Na + K = 154 mmol/l) urine output of 1 ml/kg/h, estimated IWL of 35 ml/kg/day, and initial sodium = 140 mmol/l.

Example 1
100 ml/kg/day hypotonic solution (0.2% sodium chloride equivalent, Na + K = 34 mmol/l).[1]

Water		**Sodium/potassium**	
Input 100 ml/kg	= 1 l	Input (Na+K)	= 34 mmol
Urine output 25 ml/kg/day	= 0.25 l	Urine output (Na+K)	= 39 mmol
IWL output 35 ml/kg/day	= 0.35 l		
Water balance	**= +0.4 l**	**(Na+K) balance**	**= −5 mmol**

New Na = [(Na × TBW) + balance (Na+K)]/[TBW + water balance][19]
New Na = [(140 × 6) −5]/[6 + 0.4]
= 130 mmol/l

Example 2
60 ml/kg/day hypotonic solution (0.2% sodium chloride equivalent, Na + K = 34 mmol/l)

Water		**Sodium/potassium**	
Input 60 ml/kg	= 0.6 l	Input (Na+K)	= 20 mmol
Urine output 25 ml/kg/day	= 0.25 l	Urine output (Na+K)	= 39 mmol
IWL output 35 ml/kg/day	= 0.35 l		
Water balance	**= 0 l**	**(Na+K) balance**	**= −19 mmol**
New Na	= [(140 × 6) −19]/[6 + 0]		
	= 137 mmol/l		

hyponatraemia is indeed caused by a deficit of sodium, or by an excess of water, and whether the logical response should be a change to the electrolyte content, or the prescribed volume of PMS.

This article will examine the flaws in the argument for increasing the tonicity of PMS, and explore an alternative hypothesis: that reducing maintenance fluid volume would be equally or more effective as a prophylactic measure against hyponatraemia. Although previous authors have suggested a reduction in the amount of prescribed maintenance fluid, it should be emphasised that the merits of either proposal have yet to be tested in large prospective clinical trials.[1 12 13]

The case against a change to isotonic PMS as a prophylactic measure against hospital acquired hyponatraemia in children hinges on four key issues:

- Traditional volume recommendations for PMS are greater than actual requirements in children at risk of non-osmotic ADH production.[13 14]
- Electrolyte-free insensible water loss (IWL) should be included in the calculation of a tonicity balance in children.[15]
- The principal mechanism leading to hyponatraemia is the primary antidiuresis (dilution), not the secondary natriuresis (desalination).[16 17]
- In the absence of randomised controlled trials, there is insufficient evidence to support the safety, effectiveness, or relative merit of isotonic PMS in children.

PRINCIPLES OF MAINTENANCE FLUID THERAPY

In order to avoid hyponatraemia (or hypernatraemia) a tonicity balance must be preserved, by matching input and output of both water and electrolytes to maintain an isotonic final product.[18] Each input and output may be divided into two components, the volume of water, and the content of effective osmols (sodium and potassium), so that the net effect on tonicity may be calculated from the sum of these separate components.[18]

A nephro-centric approach to maintenance fluid therapy that ignores IWL will contain an inherent error, since *all inputs* and *all outputs* need to be considered.[18 19] Although such an approach might be acceptable in adults, children have greater proportional surface area, and the magnitude of error would increase with the proportion of IWL.[20]

It is important to note that IWL, the "perspiratio insensibilis" of Santorio, represents loss of *electrolyte-free* water.[21] Estimated IWL may be derived from data reported in hospitalised infants and smaller children, ranging from 29 to 54 ml/kg/day for a 10 kg infant.[22–28] After endogenous water of oxidation (270 ml/m^2 day) is subtracted, net IWL would amount to 30–35 ml/kg/day.[25] Approximately one third of IWL occurs via the respiratory tract, and two thirds via insensible evaporation from the skin.[29] Since cutaneous IWL is determined by body surface area, net IWL varies with age, and may be as little as 520 ml/day in adults under basal conditions.[30]

In 1956 Holliday and Segar devised a method for calculating maintenance fluid requirements, in which both insensible and urinary water losses were based on energy expenditure.[14] Maintenance electrolyte needs of 3 mmol/kg/day sodium and 2 mmol/kg day potassium were somewhat arbitrarily based on the amount delivered by human breast milk feeds (1 mmol kg/day sodium and 2 mmol/kg day potassium).[3 14]

Caloric expenditure was estimated as 100 kcal/kg/day for an infant weighing up to 10 kg, so that water loss could be calculated per kg body weight.[14] Using this approach, IWL for a 10 kg infant would be calculated as 50 ml/kg/day, with 16 ml/kg/day subtracted for endogenous water of oxidation, equating to net insensible loss of 34 ml/kg day.[14] Urinary losses, based on the water required to excrete the solute load of cows' milk, would be calculated as 66 ml/kg day, or 2.75 ml/kg/h.[14]

The sum of the net IWL (34 ml/kg) and renal water loss (66 ml/kg) produced

the arithmetically pleasing calculation of 100 ml/kg/day.[31] Moritz and Ayus assert that this formula for calculating water needs "clearly has passed the test of time".[1] However, even though almost half a century has passed, the formula has not been put to the test.

PROBLEMS WITH TRADITIONAL MAINTENANCE RECOMMENDATIONS

Urine output may be 1 ml/kg/h, or less, if determined by non-osmotic ADH production rather than solute load, and therefore children at risk of hyponatraemia may receive 40–50 ml/kg/day over and above their actual maintenance water needs.[3] It is also notable that hospital acquired hyponatraemia may be associated not only with hypotonic PMS, but with amounts of fluid that exceed, by up to 50%, even currently recommended maintenance volumes.[2 3 10 32 33]

Individual maintenance water needs also depend on motor activity, temperature, and biological work.[23 34] Since the energy expenditure of physically immobile, critically ill children may be less than 40 kcal/kg/day, their maintenance water requirement would be reduced.[34 35] We might expect a further 30% reduction of IWL in patients breathing warmed humidified air through a ventilator circuit, which illustrates an important aspect of fluid balance in critically ill ventilated children.[29] If their fluid requirement is dramatically reduced, by virtue of lower respiratory and cutaneous IWL, and the sodium requirement is unchanged, the concentration of PMS required to deliver that sodium increases.[29 34 35] However, this consideration does not apply to the vast majority of hospitalised children with non-osmotic ADH production, whose reduction in fluid loss is predominantly urinary (high electrolyte content), rather than insensible (zero electrolyte content).[1 3 6 29 34 35]

REDUCTION IN MAINTENANCE FLUID VOLUME

Previous authors have suggested a reduction of maintenance fluid volume in high risk patients, and fluid allowance of 50 ml/kg/day is standard practice for infants with bronchiolitis in some centres.[2 4 12 13] The rationale for avoiding such "fluid restriction" is that it may be disadvantageous to children with hypovolaemia.[12 36] Three prospective studies address this issue in meningitis.[7 37 38] Powell *et al* showed that plasma vasopressin fell with the administration of additional fluid, suggesting an appropriate ADH response to hypovolaemia.[7] Singhi *et al* showed that although (hypotonic) fluid restriction normalised serum sodium in hyponatraemic patients, it did not lead to a significant outcome advantage or disadvantage, except in post hoc subanalyses.[38] Duke *et al* compared oral fluid restriction and full intravenous maintenance, with no statistically significant difference in serum sodium or adverse outcome.[37]

Clearly, hypovolaemia and inadequate organ perfusion may be disadvantageous to patients with meningitis.[39] However, neither the volume nor composition of maintenance fluid should be a consideration in the treatment of hypovolaemia, which should be corrected immediately with rapid infusion of resuscitation fluid.[40] It has even been suggested that synthetic colloid, rather than saline, should be used to avoid sodium loading during resuscitation.[41]

MECHANISMS OF HYPONATRAEMIA

ADH increases the permeability of the distal renal tubule and collecting duct, resulting in renal conservation of water and inappropriately high urinary sodium concentration, so that children who develop hyponatraemia may excrete urine isotonic to plasma.[6 42 43] Excessive ADH production has also been termed a phenomenon of salt loss or "desalination", based on the secondary increase in net urinary sodium loss, possibly due to suppression of aldosterone, increased natriuretic peptide, or increased glomerular filtration, which occurs after over-expansion of the intravascular space.[6 16 17 42–45]

Experimental models show that the acute hyponatraemia is primarily dilutional, while the secondary natriuresis contributes to the maintenance of ongoing hyponatraemia.[16 17] In a model of 1-desamino-D-arginine vasopressin (DDAVP) infusion, two thirds of the acute hyponatraemia was ascribed to water retention, and one third to sodium depletion.[16] In a similar experiment, rats infused with DDAVP (but not arginine vasopressin) maintained constant sodium balance, and hyponatraemia resulted from water retention alone.[45]

It is important to note that the secondary "desalination" may be prevented by fluid restriction.[17] In normal adults given pitressin, there was no increase in natriuresis if fluid intake were restricted to prevent over-expansion of the intravascular space.[17] It follows that the administration of isotonic saline may be futile unless fluid volume is also reduced, since ongoing natriuresis may negate the effect of this intervention.[15 46 47]

Studies in surgical patients show that while the fall in sodium is related to the volume of electrolyte-free water administered, the sodium falls even if isotonic fluid is administered to produce a net positive sodium balance, evidence of the primary dilutional nature of the hyponatraemia.[44 48 49] Therefore, if the fundamental problem is antidiuresis, rather than natriuresis, surely the principle of treatment should be less fluid, not more salt?

THEORETICAL EFFECTS OF VARIATION IN MAINTENANCE FLUID REGIMEN

It has been suggested that a tonicity balance should be used to predict changes in natraemia, rather than an electrolyte-free water approach.[18] Changes in sodium are related to the ratio between effective osmols (sodium and potassium) and total body water, and the term "isotonic" refers to a solution in which the sum of both sodium and potassium amounts to 154 mmol/l.[1 47 50] Changes in sodium may then be predicted by calculating a tonicity balance from the net gain or loss of effective osmols and water.[18 19] Five per cent dextrose is considered necessary for maintenance of normoglycaemia and cerebral metabolism.[51] However, although the additional dextrose increases the osmolality of PMS, we would not expect it to affect serum sodium, since glucose is not an effective osmol.[1 18]

From the examples in the box, it is apparent that giving 100 ml/kg/day of hypotonic (0.2% saline equivalent) PMS to a child with non-osmotic ADH production might result in a clinically significant fall in sodium from 140 mmol/l to 130 mmol/l. It can be seen from the large positive fluid balance, and small negative sodium balance, that this fall would be primarily dilutional. If tonicity were increased from 0.2% to 0.9% saline equivalent, with volume unchanged at 100 ml/kg/day, hyponatraemia might be prevented at the expense of a large positive fluid balance.

If instead of increasing sodium content, the amount of hypotonic PMS (0.2% saline equivalent) were decreased to 60 ml/kg/day, we might expect a clinically insignificant fall in sodium to 137 mmol/l over a period of 24 hours, but with no increase in total body water. This minor fall in sodium may not even occur if fluid restriction effectively reduces the natriuresis.[17]

LACK OF EVIDENCE FOR ISOTONIC PARENTERAL MAINTENANCE SOLUTIONS

Changes in sodium may be predicted by theoretical manipulation of tonicity balance, but it should be emphasised that current recommendations for water and

electrolyte needs have not been rigorously tested, and are based on estimated values for energy expenditure and IWL derived from small historical studies.[1 14 24–26] Although individual needs vary, recommendations for administration of PMS should be appropriate for the majority of all hospitalised children, while simultaneously safeguarding against hypo- or hypernatraemia in high risk conditions.[1 13 14 34 51]

If we consider increasing the tonicity of PMS to prevent hyponatraemia, several fundamental questions are yet to be answered.[1] Would isotonic PMS be safe?[41 51] Crucially, does it work? Is isotonic PMS actually effective in reducing the incidence of hyponatraemia? Would reducing the maintenance volume of hypotonic PMS be equally effective?[34 42] Given that hyponatraemia may occur despite isotonic fluid administration, and despite a positive sodium balance, isotonic PMS may not be effective in preventing hyponatraemia, unless fluid volume is also reduced.[44 47]

A large multicentre randomised trial is needed to compare the current standard of care (hypotonic PMS) with (a) isotonic PMS, (b) isotonic PMS at reduced volume, and (c) hypotonic PMS at reduced volume, in children at risk of hyponatraemia. A recent review considers the ethical aspects of such a trial, in which equipoise must be maintained.[52] Clearly, it would be unethical to perform a study in which the balance of evidence suggests that one treatment arm is inferior to the other. It would be equally unethical to perform a study in which lack of scientific rigour jeopardises the validity of the findings.[52]

The morbidity and mortality associated with hypotonic PMS is not disputed, but also underlines the pitfalls of adopting a standard of care without robust evaluation.[1–3 8–11 14 52] For the reasons outlined above, we may not assume that isotonic PMS would be superior to the current regimen, nor that isotonic PMS is without potential disadvantages.[41 51] A prospective trial to compare the effect of different maintenance fluid regimens on sodium and fluid balance would be both feasible, and ethically acceptable, if serial measurement of sodium and effective data safety monitoring could be ensured. Therefore, until it can be shown that isotonic maintenance fluid is both safe, and effective, in preventing hospital acquired hyponatraemia, calls for widespread change in practice are premature.[1 8 9]

SUMMARY

The morbidity and mortality associated with hospital acquired hyponatraemia should prompt re-evaluation of measured energy expenditure, water loss, and electrolyte needs in hospitalised children. Traditional recommendations for maintenance fluid volume exceed actual requirements and contribute to the development of hyponatraemia in children at risk of non-osmotic ADH production. Reducing the volume of maintenance fluid may be a more effective prophylactic measure than an increase in sodium content, and a prospective clinical trial should be performed to resolve this issue. Unless the evidence of such a trial were to support the use of isotonic maintenance fluid in children, an injudicious change in clinical practice may not correct the errors of the past 50 years, but compound them.

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NEWS AND NOTES FROM THE UK

When I did resident on-call, every now and then a colleague would discover me in front of children's TV on a Saturday or Sunday morning, usually consuming a hurried on-call breakfast. The excuse "It was on when I came in" wore a bit thin, but fortunately now I have another, much better one should I need it. I'm doing market research. It is the same thing that a previous boss would claim he was doing when he read the local newspaper—a rag of doubtful value and variable accuracy. "This is part of my job" he'd say, and believe. "This is our constituency—a fact we forget at our peril. This is what the people who pay our wages think. Or what they are being told to think."

Back to the weekend morning, and if you haven't tried this recently, you should. Watch television as an anthropologist. Count the adverts and examine their strange internal logic. Look at the link between the adverts and the programme content. Find out what our children are being told to think. I'm told that the best way to advertise to children is to pitch the message just beyond their level of understanding. My guess is that this somehow appeals to both their and their parents' sense of premature ability, or it confuses the child while appealing to the parent, or maybe it just confuses both. I do know that after about 20 minutes of watching I'm at least 10 IQ points the poorer.

The next thing to do is to pick up a teen magazine. As I've said, it is market research, so you have a perfect excuse. Look at the seamless segue between content and advertisement. Look at the lifestyle articles telling our teens what they should be buying and where, how they should look and feel, what they should do and when. Actually, it is on this last issue that I find the single redeeming feature of some of these magazines. The problem pages often offer such sensible, down to earth, useful advice that I'm left wondering whether the agony aunts and uncles inhabit a different planet to the rest of the content providers.

How is this excuse for how I spend the occasional 20 minutes on a Saturday morning at all relevant to being a paediatrician? Well, it must be part of our role as child advocates to see that young people at least have a fighting chance of interpreting this deluge of information in a sensible manner. Our response could be to bring up our children in isolation—in a hut in the Scottish Highlands or Australian Outback. We could deny them access to television, magazines, and no unvetted book written since, say, 1950. Then we could release them into the world at 18 and see how they got on, secure in the knowledge that at the very least they'd had a wholesome childhood.

The other alternative, if we accept that the world that we live in is riddled with the media and, by association, advertising, then we could try to teach them a little bit about what we're beginning to understand about how advertising works. Media literacy sounds like a wishy-washy concept, but it is a powerful idea. Discussing with a 10 year old, for example, "Why are the people in this photograph smiling?" Yes, it might be because they're happy, but it might also be because they're being paid to smile, and that this helps you interpret the essential falseness of the photograph. Extend this to why the people in the photograph are thin, or holding cigarettes, and you can see the power.

It is easy to get carried away with this, but it is also very easy to fall into an advertising trap ourselves. If it weren't, if we were completely media savvy, then why would the otherwise extremely sensible and money conscious pharmaceutical companies take us out to dinner? I don't think I was a particularly stupid child, but when I was 10 and saw an aunt smoking John Player Special cigarettes, I did think that they must have been a great brand if they were named after a formula one racing car. It took me a few years to figure out the many falsehoods in that assumption.

You wouldn't take a child outside on a rainy day without making sure they were wearing a coat, would you? Why, then, would we allow a child out into a world populated with anorexic models, cigarettes, guns, fallible rock stars, soft drinks, and fast food, without comparable defences? The mental environment has become very complex, and our children need some sort of protection in order to be able to survive. Now, you'll excuse me please, as my favourite cartoon is about to start ...

I D Wacogne
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