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 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

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.[4 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).[13 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.[34] 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?[4 12] 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 …

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