Dr Makar

D16.

RAYCHEL FERGUSON

In the preparation of this report I have carefully perused the documentation presented to me by the Police Service of Northern Ireland.

I must stress that the comments I make and the answers to questions posed are only my opinion.

My opinions have not changed since my report for the Coroner dated February 2002.

The verdict at the Inquest was cause of death: a) Cerebral oedema b) Hyponatraemia.

The letter of Dr Loughrey, Consultant Chemical Pathologist (page 168) to Dr Herron, consultant neuropathologist is extremely helpful, provides an excellent chronology and a very lucid explanation of the cause of the cerebral oedema from which Raychel died. I am in total agreement with her point of view.

Raychel was a previously fit little girl who underwent an appendicectomy late on the 7th June 2001. Postoperatively she was nursed on the children's ward and was therefore likely to have been under the care of the paediatricians as far as fluid management was concerned. The trainee surgeon who performed the surgery had written Raychel for Hartmann's solution while she was in the A and E Depar tment but this was changed to Dextrose/Saline at the request of the nursing staff on the children's ward (Staff Nurse Noble) as this was the regime in use there at that time.

She was given 200ml of Hartmann's solution in theatre by the anaesthetists.

Postoperatively she repeatedly vomited, though this was never quantified, nor was the volume of urine passed.
She had been given a total of 2220ml of the dextrose/saline solution over the first postoperative 24 hours.
On the 9th of June at 0315 Raychel had a fit and at that time the sodium was found to be 119, potassium 3 and magnesium 0.59 mmol/l, a picture of severe dilution. By 0630 her pupils were fixed and dilated.

After the operation she was seen by the trainee surgeon Mr Zatar the following day who found her well early in the morning, and did not see her again until the time of the resuscitation. She was also seen by Mr Makar in the morning.
According to the statement of Staff Nurse Rice, she had asked one of the paediatric SHOs to write up another bag of dextrose saline and then later the surgical JHO wrote Raychel for an anti-emetic around 6 pm, the signature for this is not clear

It is not clear from the notes whether she was seen again by the medical staff until 0315 when she suffered the seizure. At that time she was seen by Dr Johnston a junior paediatrician who wrote a good report (page158) and acted appropriately. He discussed the clinical situation with his next superior, Dr Trainor who informed the duty consultant paediatrician, Dr McCord. The surgeons were also informed and were present at the time of resuscitation, as were the anaesthetists.

In my opinion Raychel's death was caused by a systems failure, rather than by individuals at fault.
She was being nursed on the children's ward where the paediatricians would be in charge. The regimes operating in that ward at that time would prevail, as seen by the request of the nursing staff for the surgeon to change the type of intravenous fluid to that in used on that ward. I imagine that the role of the surgeon would be confined to looking after the surgical aspects of the postoperative management.

There was a failure on the part of the nursing staff to take the postoperative vomiting seriously and not to measure or at least estimate the sort of volumes being lost in this way. There was a great deal of discussion on this in the Coroner's Court. However, it might well be that a child having had "only" an appendicectomy would not be put on a strict intake/output regime.

There was a collective ignorance of the need to replace losses from vomiting with saline or Hartmann's solution, rather than dextrose/saline. This latter solution is only appropriate for use as a maintenance agent.
There was also a collective ignorance of the need to initially restrict fluids for the first 24 hours postoperatively because of the phenomenon of inappropriate ADH secretion and water retention.

I am enclosing a list of references relating to the phenomenon of hyponatraemia, the most important of which is the Arieff paper in the British Medical Journal from 1992. It might have been expected that earlier papers such as that in the New England Journal (the most prestigious medical journal in the world in my opinion) or that from Acta Paed Scand could have gone into the collective consciousness.

Dr Jenkins conclusion in his report for the Coroner, dated 30th January 2003 (page11) reads "Raychel's untimely death highlights the current situation whereby one sector of the medical profession can become aware of risks associated with particular disease processes or procedures through their own specialist communication channels, but where this is not more widely disseminated to colleagues in other specialties who may provide care for patients at risk from the relevant condition" I note he mentions the paper from Halberthal et al from the BMJ in 2001, rather than the BMJ paper from nine years earlier.

Dr Fulton, Medical Director of the Altnagelvin Trust at the time of Raychel's death set up a Critical Incident enquiry. Dr Nesbitt, Clinical Director of Anaesthesia suggested that dextrose/saline should not be used in paediatric surgical patients and from this stemmed the publication of Guidelines on Hyponatraemia which are now used in Northern Ireland (and elsewhere in the UK) His statement is on pages 26/7 and dated April 2002.

3

BMJ 2001;322:780-782 (31 March)

Clinical review

Lesson of the week

Acute hyponatraemia in children admitted to hospital: retrospective analysis of factors contributing to its development and resolution

Do not infuse a hypotonic solution if the plasma sodium concentration is less than 138 mmol/l

Michael Halberthal, *fellow* [a], **Mitchell L Halperin**, *professor* [b], **Desmond Bohn**, *professor* [c].

[a] Department of Critical Care Medicine, Hospital for Sick Children, Toronto, Ontario, Canada M5G 1X8, [b] Division of Nephrology, St Michael's Hospital Toronto, Toronto, Ontario, Canada M5B 1A6, [c] Department of Critical Care Medicine, Hospital for Sick Children, Toronto

Correspondence to: D J Bohn dbohn@sickkids.on.ca

Hyponatraemia (plasma sodium concentration less than 136 mmol/l) is acute if the decrease in natraemia occurs within 48 hours. The major dangers from this are brain cell swelling and herniation. [1 2] Two factors are required for hyponatraemia to develop: a source of electrolyte free water and vasopressin to prevent the excretion of that water.[3] Electrolyte free water is given routinely as maintenance fluids based on formulas developed in studies in healthy children more than 40 years ago. [4 5] There are many reasons to anticipate that vasopressin will be released in sick patients (box).[6] Patients with an acute illness may arrive in hospital with a low plasma sodium concentration because of previous water intake. Hence, to minimise the potential threat of brainstem herniation it is important to measure the plasma sodium concentration if intravenous solutions are to be given.

Causes of vasopressin release

- Hypernatraemia (most important stimulus, but not in these patients)
- Low "effective" circulating volume (greater than 7% decrease in extracellular fluid volume)
- Nausea, pain, anxiety
- Drugs (some act through inducing nausea)
- Afferent stimuli by way of the vagus nerve [×] for example, lung lesions

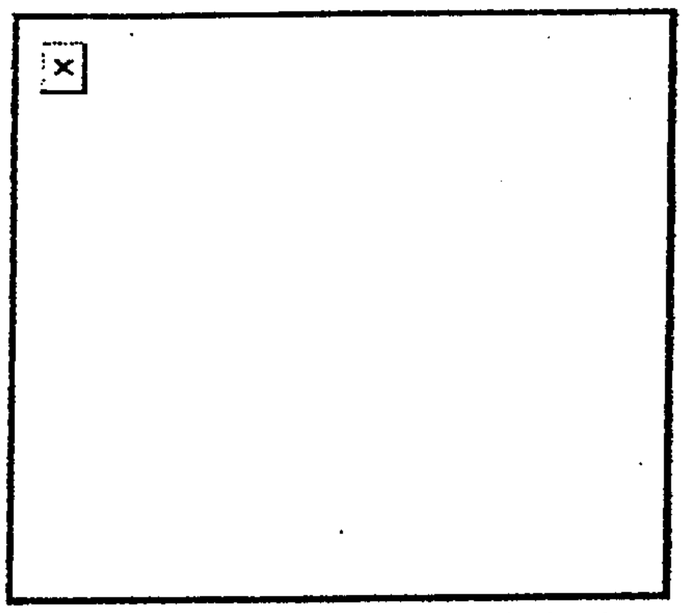
- Disturbances of the central nervous system (meningitis, encephalitis)
- Metabolic and endocrine disorders for example, hypothyroidism, hypoadrenalism, porphyria

We describe symptomatic hyponatraemia developing over 48 hours in children. In each patient, hypotonic solutions were infused using current guidelines.[7] We related the volume of electrolyte free water given to the decrease in natraemia and assessed whether actions of vasopressin persisted to guide emergency corrective therapy.[8]

We reviewed all patient charts (306 charts) with a recorded diagnosis of hyponatraemia for the past 10 years. Patients were included if their decrease in natraemia was to less than 130 mmol/l and this occurred within 48 hours, if intravenous fluids were given, and if an underlying disease did not compromise renal handling of sodium or water. Thirty patients had acute hyponatraemia. Crucial information was missing for seven, leaving 23 patients in the study group. The median age was five years (range one month to 21 years), with males predominating (18 of 23); 13 developed hyponatraemia in the postoperative period. Fifteen patients were referred to the critical care unit after the development of symptomatic hyponatraemia while receiving intravenous fluids 11 were from the hospital wards and four were transferred from other institutions. Symptoms included seizures (18 patients) and vomiting,17 a warning sign of an increased intracranial pressure. Treatment was withdrawn from five patients after brainstem coning. One patient sustained permanent, severe neurological damage.

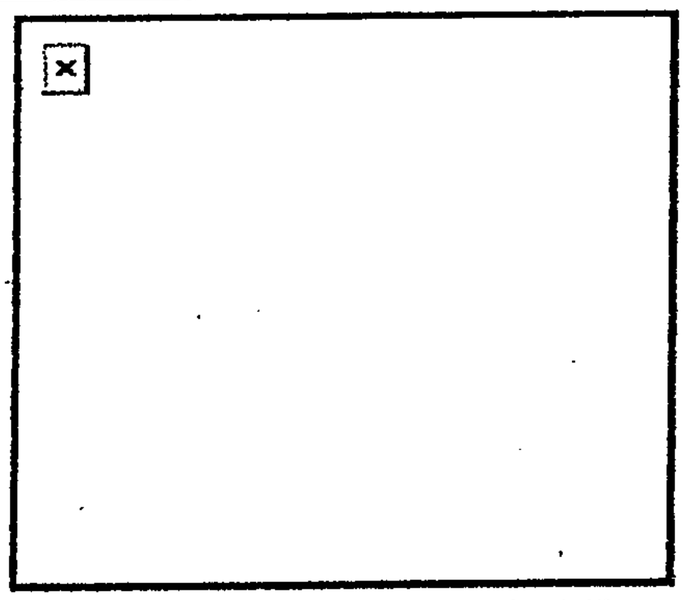
Results

All the children received hypotonic fluids while their plasma sodium concentration was less than 140 mmol/l, because of the wide belief in paediatric practice that "maintenance fluids" should be hypotonic.[9] In fact the volume of maintenance fluid given was 50% greater than recommended values in 16 of the 23 patients. This infusion of hypotonic fluids increased the risk of acute hyponatraemia and brain swelling because vasopressin is typically present in this setting.[1 2 10 11] In quantitative terms, some of the electrolyte free water infused was retained in six of the patients because their urine sodium plus potassium concentration was less than 25 mmol/l (fig 1). In six patients more electrolyte free water was infused than needed to cause the observed decline in natraemia (points above line of identity in fig 2). The remainder of the patients had a decrease in natraemia that exceeded the decline if the entire volume of electrolyte free water infused was retained (points below broken line in fig 2). Therefore there was either another non-recorded input of water or the excretion of a large volume of hypertonic urine (a desalination of infused isotonic saline[12]).



View larger version (19K):
[in this window]
[in a new window]

Fig 1. Concentration of sodium and potassium in urine at nadir observed for plasma sodium concentration in 17 patients (numbers represent rate of urine flow)



View larger version (26K):
[in this window]
[in a new window]

Fig 2. Comparison of decline in plasma sodium concentration with amount of electrolyte free water given in 17 patients. Difference in sodium concentration was between initial value and that at its nadir

Discussion

Top
Results
Discussion

References

One objective of our study was to assess the renal actions of vasopressin. Because six patients had very hypotonic urine at their recorded nadirs of natraemia, their plasma sodium concentration might have been much lower before water diuresis began (fig 1). Had their plasma sodium concentration been measured after this large water diuresis, the erroneous conclusion might have been drawn that acute hyponatraemia had never been present. Hence its incidence may be much higher than shown by an analysis of hospital records. Therefore acute hyponatraemia could have been an occult cause of morbidity and mortality.

Another implication of cessation of the release of vasopressin concerns treatment. Treatment for acute, symptomatic hyponatraemia causes a prompt decline in the size of brain cells.[10] Hypertonic saline (3%) is the commonest treatment for shrinking brain cell volume, thereby lowering intracranial pressure. Treatment must be prompt because deterioration may be rapid and irreversible, even when symptoms are mild. Enough hypertonic saline (a total of 5 mmol of

sodium chloride per litre of body water[13]) is needed acutely to lower intracranial pressure sufficiently to minimise this risk (the plasma sodium concentration should be increased by 5 mmol/l over several hours). Because an excessively rapid rate of correction of hyponatraemia might have deleterious effects,[6] hypertonic saline should not be given if there is a brisk water diuresis. For example, the plasma sodium concentration will also increase by 1.2 mmol/l/h if 6 ml of electrolyte free water are excreted per kilogram per hour (total body water is close to 600 ml/kg; 6 ml is a 1% change of 120 mmol/l). Whereas excretion of hypotonic urine indicates that electrolyte free water is being excreted (6 of 17 patients, fig 1), it is also important to consider the rate of urine flow. Little electrolyte free water was excreted in the index oliguric patient (flow 0.16 ml/kg/h). By contrast, the excretion of electrolyte free water was high enough to increase the plasma sodium concentration by close to 3 mmol/l/h in the polyuric index patient who recovered (15 ml/kg/h). Vasopressin continued to act in patients excreting isotonic or hypertonic urine, so hypotonic intake must be avoided in them. With these high urine tonicities a further decrease in natraemia would be anticipated if the urine output was high (index case designated with a urine output of 5.3 ml/kg body weight, fig 1).[12] Finally, vasopressin concentrations may decline abruptly, increasing the excretion of electrolyte free water.

Serious symptoms may become evident when hyponatraemia approaches 120 mmol/l, but there are cases where symptoms become evident with a higher plasma sodium concentration, whereas others tolerate this electrolyte disorder without developing seizures.[14] Apart from underlying conditions that might make a patient more susceptible to seizures, a possible important factor could be the extracellular fluid volume of the brain. If this volume was expanded by a large infusion of isotonic saline, a higher intracranial pressure might be present at a given degree of hyponatraemia. Moreover, because there is a relatively larger proportion of brain cell volume to extracellular fluid volume in young patients, they are more vulnerable to an increase in brain cell volume.

Study limitations

Because of a reporting and referral bias, the incidence of adverse outcomes from hyponaetremia cannot be deduced from these data. Our results highlight the dangers of the routine use of hypotonic solutions when vasopressin acts. The currently used guidelines for maintenance fluids in children admitted to hospital must be changed because they do not take into account the unpredictability of vasopressin secretion. We recommend that the concentration of plasma sodium should be measured when starting an intravenous infusion. If it is less than 140 mmol/l then isotonic and not hypotonic fluids should be given. The use of hypotonic solutions should be reserved for patients who have a plasma sodium concentration greater than 140 mmol/l. If a patient receives intravenous fluid that exceeds 5% of total body water (30 ml/kg) then their plasma sodium concentration should be measured. If an intravenous infusion is started to give drugs, a small volume should be used, and the solution should be isotonic if possible.

Acknowledgments

Contributors: MH collected the data and drafted the original manuscript. MLH analysed the data and coauthored the manuscript. DB had the original idea and coauthored the manuscript; he will act as guarantor for the paper.

Footnotes

Competing interests: None declared.

References

Top
Results
Discussion

References

1. Arieff AI. Hyponatremia, convulsions, respiratory arrest, and permanent brain damage after elective surgery in healthy women. *N Engl J Med* 1986; 314: 1529-1535[Abstract].
2. Arieff AI, Ayus JC, Fraser CL. Hyponatremia and death or permanent brain damage in healthy children. *BMJ* 1992; 304: 1218-1222[Medline].
3. Halperin ML, Oh MS. The dysnatremias: hyponatremia and hypernatremia. In: Glassock RJ, ed. *Current therapy in nephrology and hypertension.* 4th ed. Philadelphia: Mosby-Year Book, 1998:1-9.
4. Darrow DC, Pratt EL. Fluid therapy. *JAMA* 1950; 143: 365-373.
5. Halliday M, Segar W. The maintenance need for water in parenteral fluid therapy. *Pediatrics* 1957; 19: 823-832.
6. Sterns RH, Clark EC, Silver SM. Clinical consequences of hyponatremia and its correction. In: Seldin DW, Giebisch G, eds. *Clinical disorders of water metabolism.* New York: Raven, 1993:225-236.
7. Adelman RD, Solhung MJ. Pathophysiology of body fluids and fluid therapy. In: Nelson WE, ed. *Textbook of pediatrics.* 15th ed. Philadelphia: WB Saunders, 1996:207.
8. Rose BD. New approach to disturbances in the plasma sodium concentration. *Am J Med* 1986; 81: 1033-1040[Medline].
9. Chesney RW. The maintenance need for water in parenteral fluid therapy. *Pediatrics* 1998; 102: 399-400[Full Text].
10. Soupart A, Decaux G. Therapeutic recommendations for management of severe hyponatremia: current concepts on pathogenesis and prevention of neurologic complications. *Clin Nephrol* 1996; 46: 149-169[Medline].
11. Wattad A, Chiang M, Leighton-Hill L. Hyponatremia in hospitalized children. *Clin Pediatr (Phila)* 1992; 31: 153-157[Medline].
12. Steele A, Gowrishankar M, Abrahmson S, Mazer D, Feldman R, Halperin ML. Postoperative hyponatremia despite isotonic saline infusion: a phenomenon of `desalination.' *Ann Intern Med* 1997;126:20-5.
13. Spital A, Sterns RD. The paradox of sodium's volume of distribution: why an extracellular solute appears to distribute over total body water. *Arch Intern Med* 1989; 149: 1255-1257 [Medline].
14. Ayus JC, Wheeler JM, Arieff AI. Postoperative hyponatremic encephalopathy in menstruant women. *Ann Intern Med* 1992; 117: 891-897[Medline].

(*Accepted 26 July 2000*)

RF - PSNI

323

098-081-241

CROSS Billy

From: esumner [es015f8200[redacted]k]
Sent: 31 January 2007 13:16
To: CROSS Billy
Subject: Re: Ferguson

Yes - it came through!
I'm away next week. I'll ponder the points and get back to you.
All the best - Ted
----- Original Message -----
From: <Billy.Cross[redacted]>
To: <esumner[redacted]
Sent: Wednesday, January 31, 2007 12:04 PM
Subject: Ferguson

Dear Dr Sumner

Following our last meeting we have had discussions with the Public Prosecution Service. They have responded as below and have requested that we ask you to address clearly three specific points. Can I ask you:

1. Is it possible that we could meet to discuss these matters in London at a time convenient for you? I do not expect the meeting would take too long. I have other police business in London and can arrange it to suit a meeting with you.
2. Can you consider the points a) - c) below and reply to them in writing, perhaps in a preliminary fashion by email before we meet so that we are in a position to discuss your views, and then to finalise your opinion after the meeting.

We have previously discussed the proofs required to establish an offence of Gross Negligence Manslaughter. Whilst certain inferences could be drawn from the conclusion that Dr Sumner expressed in his report of Sept 2005, he does not specifically address the issue of whether in his opinion there is evidence of Gross Negligence. I suggest that he be asked to review the evidence and comment specifically on the following points:

a) In his opinion, was any member of medical or nursing staff responsible for any part of Raychel's care from the time of her admission to hospital negligent in any aspect of her treatment or care?
b) If so, did that negligence constitute gross negligence?
c) Was that negligence, if gross, causative of the death of Raychel?

Dr Sumner has cited "systems failure" as the cause of Raychel's death. You may wish to ask him to address specifically the parts of the system that he asserts failed, to enable specific further enquiries to be made.

For your consideration.

Billy Cross
DSgt

****

****

--

No virus found in this incoming message.
Checked by AVG Free Edition.
Version: 7.5.432 / Virus Database: 268.17.17/661 - Release Date: 30/01/2007
23:30

Edward Sumner
MA BM BCh FRCA

[redacted]

Telephone/Fax [redacted]

Mobile

E-mail

My initial thoughts on the questions posed, suggest that there was no one person – specific doctor or nurse, negligent during the course of Raychel's treatment.
By "systems failure" I mean a sequence of causes each of which contributed to the death which we believe to be dilutional hyponatraemia cause by excessive volumes of water in the face of prolonged and severe vomiting in a postoperative child.

There was no written protocol which doesn't surprise me but usually there is a general understanding of the correct line of treatment.

Raychel's care was in the hands of the surgeons but she was nursed on the paediatric (medical) ward and the paediatricians who would be in and out of the ward all day would only be involved if specifically requested to do so

The fluid postoperative fluids prescription written by the anaesthetist was crossed out – he said he didn't prescribe for children and understood that the nurses would ask one of the paediatricians of which there were several (and all very junior). For example we have no idea who prescribed Valoid; Mr Gilliland said "it could have been any doctor on duty"

The nurses failed to realise how severe and prolonged was the vomiting and did not pass the information on to the doctors.

It was therefore the system which placed Raychel under the care of the surgeons but on a paediatric ward where the care-givers were probably more familiar with paediatric medical conditions which is part of the problem. There was no realisation that a fluid regime for a medical condition in childhood might be inappropriate in the postoperative setting.

I cannot believe that the negligence in this case could possibly amount to Gross Negligence, however:

The system, though flawed was that in use at the time
The fluids prescribed were those still in general use by paediatricians
Several nurses and doctors were involved so no one person was able to assess the vomiting and general clinical progression
There would be a mind-set which suggested that it was only an appendicectomy and therefore nothing could go wrong.