Witness Statement Ref. No. 038/1

NAME OF CHILD: Raychel Ferguson

Name: Peter Crean

Title: Doctor

Present position and institution: Consultant Paediatric Anaesthetist
Royal Belfast Hospital for Sick Children

Previous position and institution:
[As at the time of the child's death]

Membership of Advisory Panels and Committees:
[Identify by date and title all of those between January 1995-December 2004]

Member of Hyponatraemia Working Group 2001-02

Previous Statements, Depositions and Reports:
[Identify by date and title all those made in relation to the child's death]

Statement for Coroner's Inquest, 05.02.03 (012-016-119)
Deposition at Inquest (012-032-159-160)
Statement to RVH regarding Raychel Ferguson 22.01.02 (064-041-129)

OFFICIAL USE:
List of previous statement, depositions and reports attached:

Ref:	Date:	
064-041-129	22.01.02	Statement
012-146-119	Undated	Statement
012-032-159	05-02-03	Deposition at the Inquest into the death of Raychel Ferguson

Particular areas of interest
[Please attach additional sheets if more space is required]

1. Describe in detail your role in the treatment and care of Raychel Ferguson when she arrived at the Paediatric Intensive Care Unit at RBHSC on 9th June 2001, to include:
(i) your observations and concerns in respect of Raychel when you first examined her;
(ii) your thoughts at that time as to the probable cause of her condition;
(iii) your prognosis at that time for Raychel.

I was the consultant on duty in the Paediatric Intensive Care Unit (PICU), Royal Belfast Hospital for Sick Children on the day Raychel was transferred from Altnagelvin Hospital. It was my role to care for the children in the PICU and to liaise with other consultant colleagues in RBHSC regarding specialist management issues, as appropriate.

1. On arrival Raychel could not breathe for herself and she was intubated and ventilated. She did not move purposefully and her pupils were fixed and dilated and did not react to light. A CT scan had shown brain swelling (cerebral oedema) and my initial concern was of a catastrophic event leading to brain death.

2. The most likely cause of the cerebral oedema was a rapid fall in serum sodium.

3. At the time of her arrival at the PICU I felt that brain stem death had already taken place.

2. Describe in detail the further steps you took in relation to the treatment of Raychel both on the 9th and 10th June 2001, to include any discussions with colleagues that you had in relation to Raychel's condition.

As Raychel was unable to breath, mechanical ventilation was maintained as were other supportive measures. I asked my paediatric neurology colleague, Dr D Hanrahan, to assess Raychel on the afternoon of the 9th June. His initial finding was that of probable irreversible brain stem compromise. We then formally assessed Raychel's brain stem function on the 9th of June and again on the morning of the 10th of June (063-010-024). On both occasions the findings were consistent with brain stem death. Following a discussion with Raychel's mother and father ventilation was discontinued and she died at 12.09 on 10th June, 2001.

Particular areas of interest (Cont'd)

3. **Describe in detail your communications with the parents of Raychel Ferguson both before and after her death, to include:**
   (i) at whose request those communications took place;
   (ii) the dates and times of such communications;
   (iii) the subject matter of those communications.

It would be normal practice to meet with the parents of children in PICU on a regular basis so that they are continually updated on their child's condition.

Dr Hanrahan and I met with Raychel's parents following the completion of the first brain stem tests. This was on the 9th June some time after 17.30. An explanation of the findings of the brain stem tests was given (063-022-049).

After carrying out the second set of brain stem tests the following morning I again met with Raychel's mum and dad. I informed them that these second tests confirmed brain stem death (063-023-050).

I met with Mr and Mrs Ferguson on the 16th December 2001, at their request, to explain Raychel's post mortem results. Before this I had previously contacted Mr Leckey, the Coroner, regarding this request. He then wrote to Mr and Mrs Ferguson, confirming that I would speak to them. At the meeting I discussed the findings of the post mortem examination and answered their questions as best I could.

I may have met with Mr and Mrs Ferguson on other occasions when Raychel was in PICU but I am afraid that I am unable to remember specific instances.

4. **Describe in detail your knowledge in June 2001 of the condition known as hyponatraemia together with the source(s) of your knowledge and any steps you took prior to or after June 2001 to alert colleagues both at RBHSC and at other hospitals to the condition.**

Hyponatraemia is defined as a serum sodium of less than 135 mmol/L. This would have been known to me throughout my medical career. In 2001 I knew that a rapid fall in serum sodium could be associated with cerebral oedema. If respiratory arrest occurred as a consequence of cerebral oedema the outlook was grave.

The anaesthetic textbook that I used between 1979-80 (A Practice of Anaesthesia, Churchill-Davidson 1978) highlighted the fact that 'hyponatraemia is the commonest biochemical change postoperatively and dilution of the body sodium stores is the usual explanation' **(p681 - enclosed)**.

Soon after starting my anaesthetic career in 1977 I was aware of the TURP syndrome **(Ref Anesthesiology, Jan 1979 – copy enclosed)** which could occur during transurethral prostatectomy in adult males. During this procedure irrigating fluid, containing water and glycine, could be absorbed into the circulation in large amounts leading to dilutional hyponatraemia. If severe, this could give rise to cerebral oedema.

During my training in the Hospital for Sick Children, Toronto, from 1982-4, issues around fluid balance and hyponatraemia in children were discussed. The article by Arief in 1992 in the BMJ (059-059-140 to 144) and his editorial in Paediatric Anaesthesia in 1998 **(Arief 1998 - copy enclosed)** highlighted the potential problem of the use of hyponatraemic intravenous fluids in surgical patients. I was also aware of the Adam Strain case in 1995 and endorsed the guidelines for the prevention and management of hyponatraemia, which were drawn up by my anaesthetic colleagues. (060-018-036)

I set up a paediatric anaesthetic group in Northern Ireland in 1999 with the aim of sharing best practices relating to paediatric anaesthesia with anaesthetic colleagues across the province. At the meeting in Musgrave Park Hospital on Monday 26 November 2001 a case of hyponatraemic encephalopathy was discussed **(minute of meeting**

enclosed). This lead to a wider discussion on intravenous fluid management in children

I was a member of the Department of Health 'Hyponatraemia Working Group' from September 2001 to March 2002. This group produced a guideline on fluid balance management in children (007-003-004). I have also been involved in the review of this guideline in 2004 from which there has not yet been an outcome.

Other points you wish to make including additions to any previous Statements, Depositions and or Reports
[Please attach additional sheets if more space is required]

THIS STATEMENT IS TRUE TO THE BEST OF MY KNOWLEDGE AND BELIEF

Signed: [signature] **Dated:** 15 July 2005

(WYLIE AND CHURCHILL-DAVIDSON)

A PRACTICE OF ANAESTHESIA

EDITED BY

H. C. CHURCHILL-DAVIDSON

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volume of the extracellular fluid; in practice this simply means that both water and sodium will tend to be retained when they would normally be expected to be excreted.

The anterior pituitary secretes increased amounts of ACTH in response to surgical trauma. The afferent pathways are the peripheral somatic nerves from the site of injury and also autonomic afferents arising from intravascular pressor receptors. The main effect of ACTH is to stimulate the adrenal cortex to secrete cortisol (also called Compound F or hydrocortisone), but it is now also known to produce an increase in the adrenal secretion of aldosterone. Previously the anterior pituitary had been thought not to be concerned with aldosterone release, but two types of aldosterone activity are now recognised. The first is mediated by ACTH and results in proportionally similar increases in both cortisol and aldosterone. The other is mediated by volume-sensitive receptors thought to be in the renal juxtaglomerular bodies and results in increased levels of aldosterone and has no effect on cortisol levels. Zimmermann believes that in most surgical patients aldosterone release is predominantly due to the corticotrophin mechanism.

The Posterior Pituitary

Plasma levels of antidiuretic hormone (ADH) are consistently raised during surgery and for the first few post-operative days. During this time the normal osmoreceptor control of the hormone is overridden by other mechanisms with the result that the urine remains hypertonic whilst the plasma osmolarity tends to fall if excess water is given. Hyponatraemia is the commonest biochemical change post-operatively and dilution of the body sodium stores is its usual explanation (Singh and Flear, 1968). Zimmermann and his colleagues at West Virginia (Zimmermann, 1965; Moran and Zimmermann, 1967; Ukai *et al.*, 1968) showed that ADH levels fluctuated from minute to minute during operation in response to surgical manipulations: after operation the plasma concentration of the hormone falls but remains above the pre-operative level for up to five days. This is due to pain impulses transmitted by somatic nerves and accounts for the inability of the surgical patient to excrete hypotonic fluids at this time.

MAINTENANCE OF BODY OSMOLALITY

In health the body fluids, both cellular and extracellular, are maintained at a concentration of approximately one seventh molar:* this is chiefly due to the presence of potassium in the intracellular fluid (ICF) and sodium in the extracellular fluid (ECF), each milli-equivalent (mmol) of which will hold 7 ml of water in the cells or the extracellular fluid respectively. This water "binding" is shared equally between the cations and the anions which necessarily accompany them, but the cations are the vital constituents which determine the relative volume of the two major divisions of the body fluid. If it is known that plasma urea and sugar concentrations are within normal limits the osmolarity of the body fluids can be calculated by doubling the plasma sodium concentra-

* This is true for electrolytes which are completely dissociated. Strictly speaking the body fluids are about M 3/5. Thus isotonic solutions of electrolytes are M 1/5 and of non-electrolytes M 3/5. Contain 286 mosmol/l.

Anesthesiology
50:355–356, 1979

Water Intoxication after 15 Minutes of Transurethral Resection of the Prostate

BARBARA J. HURLBERT, M.D.,* AND DANIEL W. WINGARD, M.D.†

Water intoxication from intravascular absorption of non-electrolyte irrigating fluid is a well-known and often serious complication of transurethral resection of the prostate (TURP). The amount of fluid absorbed is related to the time elapsed and number of venous sinuses open during resection.[1] It is commonly accepted that a resection time of an hour is relatively safe for the prevention of this syndrome.[2,3] To our knowledge, acute water intoxication with grand mal seizures secondary to severe iatrogenic hyponatremia following only 15 minutes of resection has not been reported. Because of this, we feel the following case is of interest.

REPORT OF A CASE

A relatively healthy, 77-year-old, 59-kg white man, ASA II, had a history of chronic alcoholism. Past medical history, review of systems, and family history were essentially unremarkable. Two weeks prior to admission, the patient had complained of urinary retention and occasional urinary incontinence. Results of routine preoperative physical examination were normal except for an enlarged prostate with 820 ml residual volume after voiding. TURP was scheduled. Liver function studies, clotting studies, complete blood count, and renal screening studies disclosed no abnormality. Routine determination of serum electrolytes the night before the operation revealed CO_2 content, 30 mEq/l; *p*H 7.38; Cl⁻ 100 mEq/l; Na^+ [illegible] mEq/l; K^+ 4.8 mEq/l; Ca^{++} 9.3 mg/dl.

The patient was brought to the operating room unmedicated. An epidural catheter was placed at the L3–L4 interspace and advanced approximately 2 cm into the epidural space. A 3 ml test dose of 3 per cent chloroprocaine was given at 7:50 A.M. Five minutes later 16 ml 3 per cent chloroprocaine were injected, followed 10 min later by an additional 10 ml, for a total dose of 23 ml. Satisfactory analgesia was established to a level of T8. Cystoscopic examination progressed uneventfully. At 8:30 A.M. 10 ml 3 per cent chloroprocaine were injected, without change in vital signs. TURP began at 8:34 A.M. with continuous irrigation of the bladder with 1.5 per cent glycine. At 8:46 A.M. the patient began complaining of "dizziness," a "tight feeling" in his throat, and "inability to breathe"; administration of 100 per cent O_2 by mask was begun. Heart rate remained 80/min and blood pressure was 120/80 torr. Within 2 min the patient lost consciousness and had a tonic-clonic seizure above the block. The seizure abated in 2 min leaving the patient mottled and somnolent. Serum electrolytes were determined and a 3 per cent saline drip was instituted. A second seizure occurred 5 min later, and 50 ml 50 per cent glucose were given IV to rule out hypoglycemia as the cause of the seizures. Vital signs remained unchanged. A total of 1,200 ml of 5 per cent dextrose in lactated Ringer's solution had been given by this time. A systemic toxic reaction to chloroprocaine was discounted when the results of serum electrolyte determinations became available: CO_2 content 21 mEq/l; *p*H 7.20; Cl⁻ 81 mEq/l; Na^+ 104 mEq/l; K^+ 4.2 mEq/l; Ca^{++} 6.3 mg/dl. Over the next 15–20 min, 300 ml 3 per cent saline solution were given, and the procedure was completed. One unit of packed erythrocytes was given to replace an estimated 600 ml blood loss. In the recovery room 45 min later, repeated electrolyte determinations and revealed CO_2 21 mEq/l; *p*H 7.23; Cl⁻ 90 mEq/l; Na^+ 114 mEq/l; K^+ 5.0 mEq/l; Ca^{++} 6.6 mg/dl; hemoglobin was 10.4 mg/dl. Transfusion of a second unit of packed erythrocytes was begun, and 200 ml 3 per cent saline solution were given IV because the patient was still restless, nauseated and vomiting. During this period values obtained for central venous pressure ranged from 9 to 11 cm H_2O. At 2:00 P.M., repeated electrolyte determinations revealed Na^+ 129 mEq/l and K^+ 3.5 mEq/l. The patient felt better. A total of 256 mEq Na^+ had been given. The following day the serum electrolytes had returned to normal ranges with CO_2 25 mEq/l; *p*H 7.37; Na^+ 139 mEq/l; Cl⁻ 111 mEq/l; K^+ 3.6 mEq/l. The remainder of the postoperative course was uneventful.

DISCUSSION

Arterial hypertension, slowing of the pulse rate, widened pulse pressures, mental agitation, headache, confusion, nausea, dyspnea, cyanosis, progressive obtundation, convulsions, pulmonary edema, and cardiovascular collapse are seen in the classic picture of water intoxication following the use of distilled water for irrigation in TURP.[1,3] The patient's reaction depends upon the osmolarity of the fluid used, the amount of fluid absorbed, and the anesthesia administered.[3] The amount of absorption is governed mainly by three factors: 1) the hydrostatic pressure of the irrigating solution, 2) the number and sizes of the venous sinuses opened, and 3) the duration of exposure.[3,6] The osmolarity of the irrigating fluid is generally dependent on the surgeon's preference, although the solution must be transparent, non-electrolytic and nontoxic. Isotonic solutions are considered safer than hypotonic solutions.

In our case an isotonic solution was utilized. Although it was not measured, the hydrostatic pressure probably exceeded the 70 cm H_2O. Taylor advocates,[7] Taylor

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points out that often the resectionist is not a good judge of fluid accumulation, and the amount of tissue removed does not correlate with the extent of fluid uptake.[7] As would be expected, fluid absorption is more closely correlated to the progress and extent of the operative procedure than to time *per se*. Our case points out there are exceptions to the classically taught "safe one hour" resection time advocated by Fillman *et al.*[3] Time is only a relative guide for each specific surgeon. Some surgeons reach the capsule and venous sinus plexus very early, while others never reach the capsule after 2–4 hours of resection time. Numerous reports cite one hour as a relative safe time with use of distilled water as the irrigating solution. Still and Modell[8] point out many feel a much longer time is safe when isotonic solutions are used. They were surprised that their patient developed this syndrome after only 75 min of resection time. Our case points out the need for constant vigilance during the entire procedure, as water intoxication may occur at any time.

In this case serum Na^+ fell precipitously in 15 min to 104 mEq/l, resulting in grand mal seizures. Seizures are most likely to occur when serum Na^+ drops below 120 mEq/l. However, Maluf found that the more rapid the decrease in serum Na^+, the more likely were seizures to develop; a 20–30 mEq/l reduction in serum Na^+ was an ominous sign, and indicated that large amounts of fluid had been absorbed.[6] Fluid may enter the intracellular space rapidly, and serum Na^+ may not accurately reflect the true dilution that has occurred. One cannot necessarily correlate serum Na^+ level with the total amount absorbed.[4,6,7] In our case the volume absorbed appeared to be greater than the 20 ml/min that Hagstrom suggests as an average.[1]

Often the anesthetist relies on changes in vital signs to diagnose water intoxication prior to the outset of seizures, pulmonary edema and cardiovascular collapse. In this case, there was no significant change in vital signs prior to the grand mal seizures. Had general anesthesia been used, we probably would not have determined serum electrolytes so early. Pulse and blood pressure often do not reflect the true clinical picture[9]; for this reason, conduction anesthesia is preferred for the early detection of the syndrome.

Grand mal seizures as a result of water intoxication following short transurethral resection times can occur. The classic signs of water intoxication, widened pulse pressures, slowing heart rate, and hypertension, may not occur prior to central nervous system alterations. An alert anesthetist, ever aware of the possibility of early occurrence along with conduction anesthesia, allows for early detection and treatment of this syndrome.

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nesthesiology
):356–359, 1979

An Anesthesia Circuit Monitor

ROBERT FOREST, M.D.,* AND YVES LAMARCHE, M.D.†

Most new ventilators incorporate a low-pressure alarm as standard equipment. There are also available several commercial versions of high- and low-pressure alarms as separate units.[1,2] There is no question that some sort of disconnection-warning device is desirable. We describe below a multiple-function anesthesia circuit monitor. The model described adds three useful features to the basic concept of a high- and low-pressure alarm.

There is general agreement that exhaust gases should be removed.[3,5] Two problems are associated

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Editorial
Postoperative hyponatraemic encephalopathy following elective surgery in children

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Introduction

In the United States, there are an estimated 15 000 deaths per year as a consequence of postoperative hyponatraemia (1) (Figure 1). There have been a number of recent studies which have described postoperative hyponatraemic encephalopathy with death or permanent brain damage (2–6). From these studies, it appears that brain damage associated with postoperative hyponatraemic encephalopathy primarily affects menstruant women (1) and prepubertal children (6).

Postoperative hyponatraemic encephalopathy in prepubertal children

There are multiple reports of prepubertal children suffering brain damage from postoperative hyponatraemic encephalopathy (6–9). The aetiology of the hyponatraemia usually involves a combination of: a) intravenous hyponatraemic fluids; b) elevated plasma antidiuretic hormone (ADH); c) respiratory insufficiency secondary to hyponatraemic encephalopathy. It has been demonstrated in several series that plasma levels of ADH (vasopressin, antidiuretic hormone) are elevated in virtually every postoperative child (7,10–13). If such patients are given intravenous free water (any solution with a sodium concentration below 140 mmol·l^{-1}), there will always be a tendency towards postoperative hyponatraemia (14). When compared with other groups, prepubertal children are far more susceptible to brain damage from hyponatraemia than are adults (6), and recent experimental evidence demonstrates why this may be the case.

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Effects of hyponatraemia on the paediatric central nervous system

Nattie & Edwards (15) studied the effects of acute hyponatraemia on the brain of puppies. They found that acute lowering of plasma sodium from 140 to 120 mmol·l^{-1} resulted in severe hypoxaemia (arterial PO_2 fell from 11.4–6.9 kPa (88 to 53 mmHg)) and cerebral oedema. In contrast to adults, the brains of paediatric animals (three day old puppies and neonatal rats) were unable to adapt to hypo-osmotic stress by extrusion of cation (15,16).

Adaptation of the brain to hyponatraemia occurs as a consequence of the following sequence of events. First, hyponatraemia leads to a movement of water into brain cells as a result of osmotic forces. In addition, vasopressin which is usually elevated in the plasma of hyponatraemic patients (17) may lead to a direct movement of water into brain cells independent of the effects of hyponatraemia (18). The early response of the brain to this hyponatraemia-mediated oedema is the loss of blood and cerebrospinal fluid, followed by extrusion of sodium from brain cells by several pathways (19). Loss of potassium and possibly organic osmolytes follows later, in an attempt to decrease brain cell osmolality without a gain of water (20).

Effects of hormones and physical factors on brain adaptation to hyponatraemia

There is a significantly higher intracellular brain water content in prepubertal rats in comparison with adult rats, suggesting that the brain occupies a greater percent of the available intracranial volume in young rats (16). Such physical factors may be important determinants of outcome in hyponatraemic rats. As individuals age, there is a progressive decline in the volume of brain, while skull size remains constant in adult life (21). Thus, elderly individuals of both

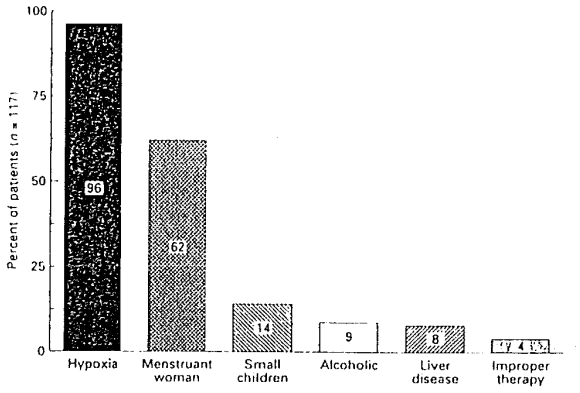

Figure 1
In nine published series from our laboratory comprising 847 hospitalized patients with postoperative hyponatraemia, 19% (158/847) developed hyponatraemic encephalopathy and 117 developed permanent brain damage or died. The major risk factors associated with permanent brain damage in these 117 patients with hyponatraemic encephalopathy are shown. Most patients (96%) suffered an hypoxic episode because of failure to initiate active therapy in a timely manner. In 4% of patients suffering permanent brain damage, improper therapy for hyponatraemia was implicated in the outcome.

genders have more room in the rigid skull for the brain to expand than do younger ones. This finding is more marked in males (21).

If adaptation of the brain is not adequate, pressure of the swollen brain on the rigid skull leads to a decrease in cerebral blood flow (22) and cerebrospinal fluid production (23). If the ability of the brain to adapt is impaired, there will be increasing oedema, with eventual tentorial herniation and secondary cerebral ischaemia (24). This often leads to respiratory insufficiency (4), with reduced delivery of oxygen to brain because of the further decrease of cerebral blood flow, thereby exacerbating the existing cerebral ischaemia (22).

Sex steroid and certain neuropeptide hormones may influence brain adaptation to hyponatraemia. Male rabbits and cats are more efficient than females in extruding sodium to decrease brain cell osmolality during hyponatraemia, resulting in significantly less brain swelling in male than in female hyponatraemic animals (16,25). Oestrogens have also been reported to stimulate, and androgens to suppress, vasopressin release (26,27). Virtually all hyponatraemic patients have increased plasma levels of vasopressin (17,28), a neuropeptide which may exert multiple potentially deleterious cerebral effects. In normonatraemic animals vasopressin results in water accumulation in the brain (18), a significant decline in brain synthesis of ATP (29), and a decline of brain pH (29,30). Vasopressin also impairs the function of several important adaptive pathways to hyponatraemia (31,32).

Recent studies have demonstrated that the brains of prepubertal rats are unable to adapt to hyponatraemia (16). The greater mortality with hyponatraemia in prepubertal rats is associated with a greater accumulation of water in the intracellular space of the brain than in rats belonging to other age groups, as well as an inability of the prepubertal brain to extrude sodium from brain cells. The baseline intracellular sodium content in the prepubertal rats was greater by almost 50% than in control adult rats, a finding consistent with previous studies in newborn dogs (15,33).

Biochemical differences in paediatric vs adult brain with hyponatraemia

There are several possible reasons for the increased brain intracellular sodium in prepubertal rats. The Na^+-K^+ ATPase system appears to be the major early adaptive pathway for extrusion of sodium from brain cells during hyponatraemia (19,34) and its impairment results in decreased ability to pump sodium out of the brain. In prepubertal rats, the brain Na^+-K^+ ATPase activity is significantly lower than that observed in adults, both *in vitro* (35) and *in vivo* (36). Coupled with the higher brain sodium, these differences may reflect a limited ability to pump sodium out of the prepubertal brain. The increased intracellular sodium content may be a consequence of limited cerebral Na^+-K^+ ATPase function in young rats compared to adults. The decreased cerebral Na^+-K^+ ATPase activity may be responsible for the impaired adaptation to hyponatraemia in prepubertal rats. Testosterone stimulates Na^+-K^+ ATPase activity in rat brain (37,38). Pretreatment of prepubertal rats with testosterone resulted in a significant decrease in the brain intracellular content of both sodium and water while also reducing the mortality associated with acute hyponatraemia from 84% to zero (16).

Clinical effects of hyponatraemia in children vs adults

If one can extrapolate the above experimental findings to paediatric patients, then the implications would be that children are more susceptible to brain damage from postoperative hyponatraemia than are adults. The reasons include: a) decreased available

room for swelling of the paediatric brain in the rigid skull, leading to a propensity for brain herniation with what might appear to be a small decrement of plasma sodium (39); b) impaired ability of the paediatric brain to adapt to hyponatraemia when compared with adults (15,37); c) severe systemic hypoxaemia secondary to respiratory insufficiency frequently occurs in children with only modest hyponatraemia (6,15,39). The respiratory insufficiency is a consequence of increased intracranial pressure (3).

Gomola *et al.* have described a prepubertal (10 years old) female child with middle face hypoplasia who underwent elective maxillary reconstruction (40). The surgery went well and postoperatively, she was given primarily free water intravenously (280 mM glucose in 51 mM NaCl) at a rate of 2 l per day. The child weighed 30 kg with estimated total body water of 18.5 l. On the first postoperative day, the child became confused and developed headache and vomiting. Renal function was apparently normal on the basis of normal plasma urea and creatinine. The plasma sodium was found to be 117 mmol·l^{-1}. She was initially treated with sodium supplementation, but on the second post-operative day, the plasma sodium was still low at 120 mmol·l^{-1}. The urine and plasma osmolalities were 342 and 255 mOsm·kg^{-1}. An MRI of the brain was normal. The authors proposed three possible explanations for the hyponatraemia: a) dilutional hyponatraemia secondary to IV hypotonic fluid; b) pituitary insufficiency; c) inappropriate secretion of ADH. Pituitary insufficiency was ruled out by normal values for ACTH, cortisol, thyroid hormone and growth hormone. The ADH was 4 to 5 pg·ml^{-1}, which is 'normal' but inappropriately high for the extracellular hypoosmolality (41) and is essentially a universal finding in both paediatric and adult postoperative patients (7–13). The child received 2 l per day of hypotonic IV fluid in the presence of elevated plasma ADH. Although neither initial plasma sodium, urine output or total volume of IV fluids are provided, given the child's weight and rate of infusion, the plasma sodium of 117 mmol·l^{-1} appears very likely to have been the consequence of retention of about 3 l of IV hypotonic fluid over two days (6). The expression inappropriate secretion of ADS (SIADH) was originally used for elevated plasma ADH related to lung cancer (42) and has become a catch all term for virtually any patient with elevated plasma ADH. In particular, postoperative patients as well as those with heart failure or hepatic cirrhosis have elevated plasma ADH levels but are functionally hypovolaemic as well (41). Postoperative subjects are functionally hypovolaemic, so that the term SIADH may not be appropriate in this patient (11). There is also a perception that ADH, and by association SIADH, can somehow lower the plasma sodium. Although ADH leads to increased retention of ingested or infused water, in the absence of increased water intake, ADH by itself will have no effect upon the plasma sodium. Thus, the most likely explanation for the hyponatraemia in this patient is infusion of hypotonic fluid (51 mM NaCl/280 mM glucose) in the presence of the expected postoperative increase in plasma ADH. Adrenal insufficiency is ruled out by the normal plasma cortisol and the fact that she remained normal for six months without any steroid replacement therapy. Exactly why the plasma sodium rose following IV hydrocortisone is uncertain, but may have been related to the expected decline of ADH values to normal after four to five postoperative days. Pituitary insufficiency is ruled out by normal values for ACTH, IGF1 and growth hormone.

Symptomatic postoperative hyponatraemia carries a mortality of at least 15% (43), particularly in children and respiratory arrest is a frequent occurrence, but once this complication occurs, the morbidity is substantial (6,7). There is no obvious rationale for the administration of hypotonic fluid to a postoperative patient, unless the individual is hypernatraemic (14). If the patient becomes symptomatic, therapy with hypertonic NaCl is indicated (39). The syndrome can be prevented by administration of primarily isotonic fluids to postoperative patients.

Acknowledgements

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A meeting of the Paediatric Anaesthetic Group in Northern Ireland was held in Musgrave Park Hospital on Monday 26 November 2001

IN ATTENDANCE

Dr Dimascio	Altnagelvin Area Hospital
Dr Lowry	Craigavon Area Hospital
Dr McKaigue	Royal Belfast Hospital for Sick Children
Dr Renfrew	Ulster Hospital
Dr Crean	Royal Belfast Hospital for Sick Children
Dr Wilson	Musgrave Park Hospital
Dr Allen	Musgrave Park Hospital
Dr Hurwitz	Belfast City Hospital
Dr Turner	Musgrave Park Hospital

APOLOGIES

Dr Carlise	Daisy Hill Hospital
Dr Kelly	Tyrone County Hospital
Dr Ferguson	Antrim Area Hospital
Dr Prasad	Antrim Area Hospital

ITEMS DISCUSSED AT MEETING

A case of hyponatraemic encephalopathy in a child following surgery was discussed. This led on to a wider discussion of preventing hyponatraemia in children receiving intravenous fluids.
The final draft document being prepared by the Department of Health in Northern Ireland 'Prevention of Hyponatraemia in Children Receiving Intravenous Fluids', was presented and discussed.

Thanks were given to Dr Callum Wilson for hosting the meeting at Musgrave Park Hospital. The next meeting will take place in Spring 2002 at the Royal Belfast Hospital for Sick Children.