

Witness Statement Ref. No. (091/2

NAME OF CHILD: Adam Strain

Name: John Leckey

Title: Senior Coroner for Northern Ireland

Present position and institution:

Senior Coroner for Northern Ireland

Previous position(s) and institution(s):

[Since your Witness Statement of 15th July 2005]

HM Coroner for Greater Belfast

Membership of Advisory Panels and Committees:

[Identify by date and title all of those since your Witness Statement of 15th July 2005]

N/A

Other Statements, Depositions and Reports:

[Identify by date and title all those since your Witness Statement of 15th July 2005]

N/A

OFFICIAL USE:

List of previous statements, depositions and reports attached (*):

Ref:	Date:	
091/1	15.07.05	Witness Statement to the Inquiry

IMPORTANT INSTRUCTIONS FOR ANSWERING:

Please attach additional sheets if more space is required. Please identify clearly any document to which you refer or rely upon for your answer. If the document has an Inquiry reference number, e.g. Ref: 049-001-001 which is 'Chart No.1 Old Notes', then please provide that number. If the document does not have such a number then please provide a copy of the document.

I QUERIES ARISING OUT OF YOUR INQUIRY WITNESS STATEMENT

With reference to your Inquiry Witness Statement dated 15th July 2005, please provide clarification and/or further information in respect of the following:

(1) Answer to Question 1 at p.2

"My understanding was that so far as the Royal Belfast Hospital for Sick Children was concerned, the hospital would "learn" from what happened to Adam"

(a) Describe and explain the system of procedures and practices in Northern Ireland for the reporting of Serious Untoward Incidents or unexpected deaths in hospitals to the Coroner's Office at that time (November 1995)

As set out in Section 7 of the Coroners Act (NI) 1959

(b) Describe any differences between the procedure then (November 1995) and now

None

(c) State what you consider to be the scope for improvement in the current system of procedures and practices

It would not be appropriate for me to comment on this.

(2) Answer to Question 2 at p.2

"There was discussion at the inquest as to how the views of Dr Sumner could be disseminated amongst the medical profession in Northern Ireland. The consensus was that there was no effective means of doing so other than through the medical literature. Dr. Sumner ...undertook to arrange for Professor Arieff... to write an editorial on the issues."

(a) Identify the persons involved in the " discussion at the inquest"

I cannot fully recollect but believe that I had a discussion with Dr Sumner at the conclusion of his evidence whilst he was still in the witness box; no transcript of the inquest is available.

(b) State *"the views of Dr. Sumner"* that the participants in the discussion/you thought *"could be disseminated"*

See answer to (2) (a) above

(c) State whether Professor Arieff wrote the editorial

I believe so.

(d) If so, state whether you received it and if so, when and to whom you disseminated it and what, so far as you are aware, resulted from it. Please furnish a copy of the editorial

As far as I can recollect I did receive a copy but I did not disseminate it. A copy of what I believe is the editorial referred to is attached.

(e) Describe and explain the available means, in Northern Ireland, for the reporting and dissemination of information on the outcomes or lessons to be learned from Coroner's Inquests to the hospital where the patient was treated, other hospitals, the doctors and nurses, Trusts, Boards and the DHSSPS including the Chief Medical Officer

No formal system existed.

(f) Describe any differences between the procedure then (June 1996) and now

None known.

(g) State what you consider to be the scope for improvement in the current system of procedures and practices

It is not appropriate for me to comment on this.

(h) Describe and explain any differences between the position in Northern Ireland and that in England and Wales

I am not aware of any.

(i) Explain why you did not make a Rule 23(2) report in Adam's case

It is not appropriate for me to answer this question.

(3) Answer to Question 2 at p.2

"The position then and now is that there is no formal interface between coroners and Chief Medical Officers"

- (a) Describe and explain any informal methods of communication between Coroners and Chief Medical Officers

By correspondence

- (4) Answer to 'Other Points' at p.3

"I had assumed that the Royal Belfast Hospital for Sick Children would have circulated other hospitals in Northern Ireland with details of the evidence given at the inquest and, possibly, some "best practice" guidelines."

- (a) Explain the basis for your assumption *"that the Royal Belfast Hospital for Sick Children would have circulated other hospitals in Northern Ireland with details of the evidence given at the inquest and, possibly, some "best practice" guidelines."*

- (5) *"... I spoke to Dr. Murnaghan and said that it appeared imperative that the equipment was now independently examined...It was agreed that the equipment should be independently examined."*
(Ref: 011-025-125 and 011-025-126)

- (a) Explain what you meant by the examination of the equipment being *"independent"*

- (b) State what action you expected Dr. Murnaghan to take to have *"the equipment"* to *"be independently examined"*

- (c) State whether an examination of the equipment and report by the Royal's personnel would amount to an *"independent"* examination

- (d) State whether your request for *"a statement from the technician responsible for the equipment in theatre, confirming that it was functioning properly"* was in addition to or instead of the subsequent *"agreement"* *"that the equipment should be independently examined."*

It is not appropriate for me to comment further on these matters.

II ADDITIONAL INFORMATION

- (5) State whether you disseminated the paper on dilutional hyponatraemia sent to you under cover of a letter from Dr. Sumner dated 20th June 1996 (Ref: 011-088-223), and if so state to whom and when you disseminated it. Please furnish a copy thereof.

I cannot recall this.

(6) Provide any further points and comments that you wish to make in relation to lessons learned from Adam's death and any other relevant matter.

It is not appropriate for me to comment on this.

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

Signed:

h. l. w. k. e. y

Dated:

25/3/11

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.

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 PO₂ 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 osmolyte-mediated oedema is the loss of brain 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

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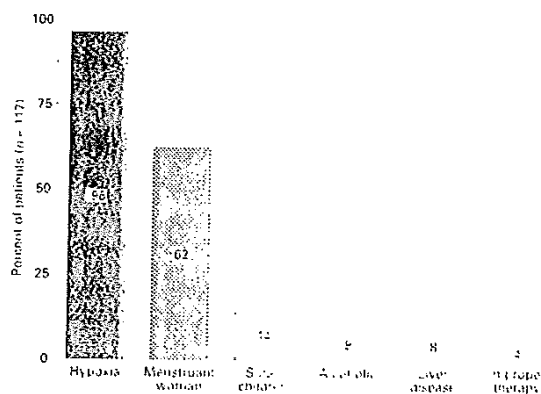


Figure 1
In nine published series from our laboratory comprising 847 hospitalized patients with postoperative hyponatraemia, 197 (158/847) developed hyponatraemic encephalopathy and 117 developed permanent brain damage or died. The major risk factors associated with permanent brain damage in these 117 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 ADH (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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