Witness Statement Ref. No. 012/1

NAME OF CHILD: Adam Strain

Name: Alison Armour

Title: Doctor Alison Armour.

Present position and institution: Consultant Pathologist at the Royal Preston Hospital. Preston. Lancashire and Consultant Home Office Pathologist MB BCh BAO FRCPath DMJ (Path).

Previous position(s) and institution(s):
[As at the time of the child's death]
Senior Registrar Forensic Medicine The State Pathologist's Department Belfast.

Membership of Advisory Panels and Committees:
[Identify by date and title all of those between January 1995-December 2010]
None.

Other Statements, Depositions and Reports:
[Identify by date and title all of those between January 1995-December 2010]
I am a Consultant Home Office Pathologist. As such I have compiled many reports and have appeared at many murder trials and trials regarding wounding offences. The reports and witness statements for these trials are numerous.

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

Ref:	Date:	
011-010-034	29.11.95	Report of Autopsy on Adam Strain
011-010-030	18.06.96	Deposition at the Inquest on Adam Strain
011-010-033		Transcript of oral evidence at the Inquest on Adam Strain
093-022	12.04.06	PSNI Witness Statement

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 autopsy report.
1a. Explain the basis upon which you report the blood loss to be ' approximately 1200 mls at the end of the procedure '.
I would have obtained this from the clinical notes.

1b. Describe and explain how the administration of " intravenous fluids of Hartman's, 100 mL HPPF and 500 mls of packed cells" constituted a replacement for Adam of blood loss calculate it to be approximately 1200 mls.
I would have obtained this from the clinical notes.

2a Dr M. Mirakhur is a Consultant Neuropathologist. The slides would have been shown to her for a second opinion.

2b. Dr Mirakhur is female. As far as I am aware what is written in my autopsy report was concurred by her.

3a. Again the histological slides were sent to Professor Berry -- Consultant Paediatric Pathologist for a second opinion. As far as I am aware his opinion concurred with mine.

3b. I cannot recall the exact site where I took the histological sample/samples. However it would have included a section of cortex and medulla.

3c. Any slides or tissue blocks will be held at the State Pathologist Department Belfast.

4a. Regarding Adam Strain's urinary output and that this was greatly increased this information would have been determined from the clinical notes. Without access to these notes it is not possible for me to state to what extent.

5a. I reported ligation of the left internal jugular vein as this is/was a fact. I refer you to my post mortem report under internal examination and specifically internal examination of neck on page 4.

5b. It was my understanding that this had been carried out after the removal of a long line.

5c. I have already clearly answered the question you ask of me. This has been clearly addressed in my autopsy report.

5d. I have clearly answered the question you ask of me. This has been clearly addressed in my autopsy report.

6a. Dead.

6b. Not sure.

6c. No.

II. Queries arising out of your deposition.

8a. I have clearly addressed the cause of this little boys massive cerebral oedema. This is clearly and concisely laid out in the cause of death on the very first page of my autopsy report. In a child undergoing renal transplantation/operative intervention this is extremely rare. I was unaware of any other case at the time. From my reading of the literature I am still of the view that it is extremely rare today. My understanding of dilutional hyponatraemia was that in the main it was a post-operative complication.

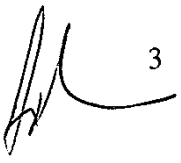
8b. From my reading of the literature.

8c. I am a pathologist and as such do not undergo training for the fluid management of patients. This is not my job.

8d. I had carried out many autopsies on children prior to that on Adam Strain. As this case occurred a long time ago I cannot recall whether cerebral oedema had caused or contributed to the death of these children. I cannot remember the details of every case I did in the past. However, as far as my memory serves me I am unaware of a case where dilutional hyponatraemia had caused the death of a child or contributed to it to any great extent. This was the first case I had undertaken of a child dying after renal transplant surgery. To date I have not carried out any such other case. Other than being allowed access to all my cases and reports that I carried out during my time in Northern Ireland it is not possible for me to answer the second part of this question accurately.

9a. By the expression haemodynamics I mean the management of his blood pressure, blood loss, fluid loss, replacement therapy and cardiovascular status.

10a. I cannot recall whether Dr Taylor was present during the autopsy on Adam Strain or not. This would be in my contemporaneous notes. I do not have access to these notes nor are they in my possession. They will be held at the State Pathologists Department Belfast. I cannot recall the circumstances in which Dr Taylor advised of the calculated blood loss without access to contemporaneous notes.



10b. Dr Taylor would have made this calculation and told me of his calculation.

11a. The notes I received prior to the autopsy would have been sent to me in the mortuary. I cannot list the notes now in 2011. I have no problem in recalling there were numerous hospital notes accompanying the body of this child.

11b. I need the contemporaneous notes to answer this question. I cannot recall this from memory all those years ago.

12a. I cannot answer this question.

12b. I have already clearly answered this question in my autopsy report.

12c. I have already clearly explained this.

III. Queries arising out of your PSNI statement.

13a. I cannot answer the question regarding the sodium content of the fluid administered other than to state that dextrose saline contains small amounts of sodium. I have clearly explained the jugular ligation and its role in cerebral perfusion. The third part of this question is only indeed part of what I have clearly said as already stated laid out in my autopsy report at the top of page 4.

IV. Additional information.

14a. All records held at the State Pathologists Department Belfast.

14b. I published a paper in the Journal of Clinical Pathology as a specific case report and using this case. The object of the publication was to ensure that this should not happen again. However, I am aware that the Journal is read mainly by pathologists. I enclose a copy for completeness. Dilutional hyponatraemia: a cause of massive fatal intra operative cerebral oedema in a child undergoing renal transplantation. Dr A. Armour. The Journal of Clinical Pathology May 1997. Volume 50. Number 5. Pages 444-446.

14c. I am a pathologist and do not treat patients nor do I give fluid replacement therapy.

14d. None.

V Declaration of Interest.

15. Confirm that you have completed and signed the attached declaration of interest. Yes.

I have had no contact with the individuals that you have listed on the declaration of interest form. However, you will see from my published paper that I acknowledged Dr Sumner Consultant Paediatric Anaesthetist for his expert opinion and Dr Robert Taylor for his helpful comments. I also acknowledged Her Majesty's Coroner for Greater Belfast Mr John Lecky for his permission to use the case. This was my only contact with these individuals regarding this paper.

This statement is true to the best of my knowledge and belief.

Signed: 

Dated: 15th March 2011

DECLARATION OF INTEREST FORM

TO Solicitor to the Inquiry

FROM Dr. Alison Sumner

I confirm that I have read the list set out below and have marked on the attached sheet those individuals with whom and (where those individuals represent an organisation, firm or government department) that organisation, firm or government department with which I declare an interest:

I confirm that: (please delete as appropriate)

a) I have disclosed on an attached sheet the existence and particulars of any personal or professional interest that I have had with the following individuals and organisations:

Dr. Maurice Savage
Dr. Mary O'Connor
Dr. Robert Taylor
Dr. Terence Montague
Mr. Patrick Keane
Mr. Stephen Brown

The RBHSC and its administrators and management, including Dr. G. A Murnaghan, Dr. J. Gaston, Dr. S. McKaigue, Dr. P.M. Crean
Belfast Health and Social Services Care Trust formerly the Royal Group of Hospitals and Dental Hospital Heath and Social Services Trust

"Professional interest" includes contact through collaboration on research, other investigations and committee work.

b) I have no such interest to declare

I have had no contact with the individuals you have listed on the declaration of interest form. However, you will see from my published paper that I acknowledged Dr Sumner Consultant Paediatric Anaesthetist for his expert opinion and Dr Robert Taylor for his helpful comments. I also acknowledged Her Majesty's Coroner for Greater Belfast Mr John Lecky for his permission to use the case. This was my only contact with these individuals regarding this paper.

I acknowledge that I am under a continuing duty to declare any personal or professional interest with those listed above that may arise hereafter.

SIGNED:

DATE : 15th March 2011

An in situ hybridisation procedure to reveal albumin mRNA on formalin fixed hepatic tissue using a digoxigenin labelled oligonucleotide probe has been developed by Murray *et al.*[4] In our case a 2 kb cDNA sequence corresponding to a coding sequence of human albumin provided by ATCC (Rockville, Maryland, USA) was used to produce a SP6 transcribed, digoxigenin 11 UTP labelled anti-sense riboprobe. The cytospin preparations were formalin fixed and the method of in situ hybridisation developed by Stewart *et al*[5] was employed. The patient had clinical signs strongly suggestive of hepatocellular carcinoma, supported by serological, ultrasound, and computerised tomography findings. The peritoneal effusion contained single neoplastic cells and groups of neoplastic cells showing nuclear pleomorphism and granular chromatin surrounded by moderate amounts of rather granular cytoplasm. These cytological features were certainly in keeping with the appearances of hepatocellular carcinoma as described in fine needle aspirate,[6] and after demonstrating human albumin mRNA in these cells we felt that the diagnosis of hepatocellular carcinoma in the ascitic fluid was certain.

We feel that application of this rapid, reliable, and specific technique of in situ hybridisation for human albumin can confirm the diagnosis of hepatocellular carcinoma on cytospin preparations of ascitic fluid, thus making further invasive diagnostic procedures unnecessary.

1 Falconieri G, Zanconati F, Colautti I, Dudine S, Bonifacio-Gori D, Di Bonito L. Effusion cytology of hepatocellular carcinoma. *Acta Cytol* 1995;39:893–7.
2 Ma C-K, Zarbo RJ, Frierson HF, Lee MW. Comparative immunohistochemical study of primary and metastatic carcinoma of the liver. *Am J Clin Pathol* 1993;99:551–7.
3 Papotti M, Pacchioni D, Negro F, Bonino F, Bussolati G. Albumin gene expression in liver tumours: diagnostic interest in fine needle aspiration biopsies. *Mod Pathol* 1994; 7:271–5.
4 Murray GI, Paterson PJ, Ewen SWB, Melvin WT. In situ hybridisation of albumin mRNA in normal liver and hepatocellular carcinoma with a digoxigenin labelled oligonucleotide probe. *J Clin Pathol* 1992;45:21.
5 Stewart CJR, Farquharson MA, Kerr T, McCorriston J. Immunoglobulin light chain mRNA detected by in situ hybridisation in diagnostic fine needle aspiration cytology specimens. *J Clin Pathol* 1996;49:749–53.
6 Pilotti S, Rilke F, Claren R, Milellan M, Lombardi L. Conclusive diagnosis of hepatic and pancreatic malignancies by fine needle aspiration. *Acta Cytol* 1988;32:27–38.

Dilutional hyponatraemia: a cause of massive fatal intraoperative cerebral oedema in a child undergoing renal transplantation

A Armour

State Pathologists Department, Institute of Forensic Medicine, Grosvenor Road, Belfast, United Kingdom
A Armour

Correspondence to:
Dr Alison Armour, Consultant Pathologist, Directorate of Pathology, PO Box 202, Royal Preston Hospital, Sharoe Green Lane North, Fulwood, Preston PR2 4HG, United Kingdom.

Accepted for publication 12 February 1997

Abstract

A four year old boy with polyuric renal failure resulting from recurrent urinary tract infections and vesicoureteric reflux from birth underwent renal transplantation. In the past he had had five ureteric reimplant operations and a gastrostomy, as he ate nothing by mouth. He required peritoneal dialysis 13 hours a night, six nights a week. His fluid requirements were 2100 ml per day. This included a night feed of 1.5 litres Nutrizon. Before operation he received 900 ml of Dioralyte instead of the Nutrizon feed, and peritoneal dialysis was performed as usual. The operation itself was technically difficult and there was more blood loss than anticipated, requiring intravenous fluids and blood. The operation ended about four hours later but he did not wake up. Urgent computed tomography revealed gross cerebral oedema. He died the next day. At necropsy the brain was massively oedematous and weighed 1680 g.

(*J Clin Pathol* 1997;50:444–446)

Keywords: cerebral oedema; operation; intravenous fluids

There are various causes of cerebral oedema including inflammatory conditions, ischaemia, trauma, space occupying lesions, anoxia, toxins, and metabolic disorders—in particular hyponatraemia[1] and water intoxication.[2] Cerebral oedema has been defined as an increase in brain volume due to an increase in its water content.[3] It can be localised or generalised. In the conscious patient it produces symptoms of raised intracranial pressure, but in the unconscious the symptoms are masked. Cerebral oedema developing as a result of hyponatraemia is well documented[4–6] but most of these cases have developed postoperatively or following intravenous administration of fluids in a conscious patient. The event described here occurred during anaesthesia, and at the end of the operation—about four hours later—the patient, a child, did not wake up and had developed papilloedema. Urgent computerised tomography showed gross cerebral oedema with slit-like ventricles. Brain stem tests were carried out and he was declared dead the next day, about 26 hours from the start of the operation. This case illustrates the complexity of fluid management in an intraoperative fatality. To pathologists carrying out these necropsies—most probably at the behest of the coroner—it is important to realise that asymptomatic dilutional hyponatraemia can occur intraoperatively when the symptoms of hyponatraemia and cerebral oedema are masked due to anaesthesia and unconsciousness. Arieff *et al*[4] studied 16 cases of symptomatic postoperative

hyponatraemia. In 13 cases symptoms were present, one was too young to assess, and two were intubated. This is the first case to document this well recognised postoperative complication occurring rapidly during an operation with fatal results. The clinical management and the treatment of hyponatraemia will not be discussed.

Past medical history and preoperative care

This child developed recurrent urinary tract infections and vesicoureteric reflux from birth, which resulted in polyuric renal failure. He had had five ureteric reimplant operations, a fundoplication for gastro-oesphageal reflux, was fed through a gastrostomy because he ate nothing by mouth, and his most recent operation was an orchidopexy in the month before his death. All of these were uneventful. He required peritoneal dialysis 13 hours a night, six nights a week. His normal fluid requirements were 2100 ml a day, including a night feed of 1.5 litres of Nutrizon. The night before the operation routine investigations showed blood pressure 108/56 mm Hg, haemoglobin 10.5 g/dl, sodium 139 mmol/l, potassium 3.6 mmol/l, and urea 16.8 mmol/l. On anaesthetic advice he was given 900 ml Dioralyte (4% dextrose, 0.18% saline) instead of the Nutrizon feed. Peritoneal dialysis was performed as usual.

Operation

The child arrived in theatre at 0645. General anaesthesia was induced using thiopentone, atropine, and atracurium. Intravenous access was difficult and attempts were made to pass a central venous pressure catheter. Three attempts were made into the left subclavian vein and one into the left internal jugular vein, and then the catheter was successfully passed into the right subclavian vein. A lumbar epidural was sited between L1 and L2 with the administration of bupivicaine and fentanyl. In addition to the anaesthetic drugs, co-amoxyclav (Augmentin), prednisolone, azathioprine, and a continuous infusion of dopamine were given intravenously.

Central venous pressure was recorded as 17 mm Hg. Three 500 ml bags of intravenous dextrose-saline (4%/0.18%) were given between 0700 and 0830. The operation was technically difficult because of previous surgical procedures, and blood loss was calculated at 1200 ml. Further fluids given were 500 ml Hartmann's solution, 1000 ml of human plasma protein fraction, and 500 ml of packed red blood cells. At 0932 a blood analysis showed a sodium concentration of 123 mmol/l (normal 135–145) and a packed cell volume of 18% (normal 35–40%). During the operation the central venous pressure rose to 20–21 mm Hg and the haemoglobin fell to 6.1 g/dl, rising again to 10.1 g/dl at the end of the operation. The systolic blood pressure rose to 150 mm Hg and the pulse rate gradually fell, but rose steadily from 1015 onwards.

After perfusion of the donor kidney the operation was completed. The neuromuscular block was reversed with neostigmine but the child did not wake up. At midday his pupils were fixed and dilated. He was transferred to the paediatric intensive care unit at 1205, intubated, hand ventilated, treated with intravenous mannitol, and intravenous fluids were restricted. On cerebral computerised tomography there was gross cerebral oedema. The central venous pressure was now 30 mm Hg, heart rate 120 beats/min, and systolic pressure 120 mm Hg. Plasma sodium was 119 mmol/l. A chest *x* ray showed pulmonary oedema. Neurologists carried out brain stem tests and the child was declared dead the next day, about 26 hours after the start of the operation.

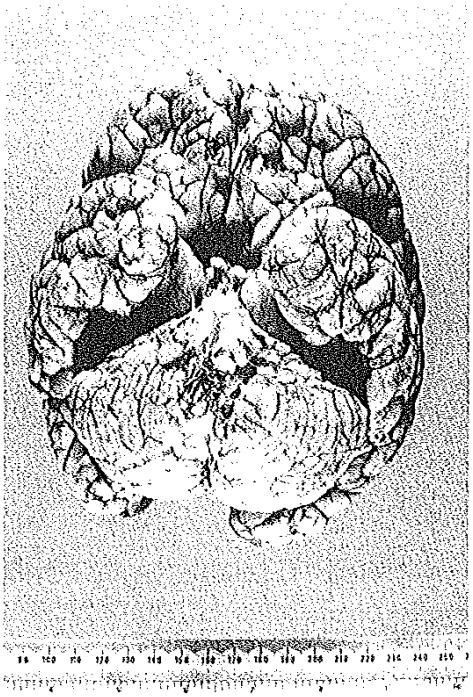


Figure 1 Marked uncal swelling and swelling of cerebellar tonsils.

Pathological findings

The brain was grossly swollen with loss of sulci and uncal swelling. The swelling was symmetrical, with swelling of the cerebellar tonsils (fig 1). There was no evidence of cortical venous thrombosis or uncal necrosis or necrosis of the cerebellar tonsils. The brain, after fixation, weighed 1680 g; the cerebellum and brain stem weighed 176 g and the cerebellum alone 154 g. On cut section there was massive brain swelling with constriction of the ventricles (fig 2). There was congestion of the white matter and blood vessels in the basal ganglia and deep grey matter. There was no evidence of necrosis of the midbrain or brain stem.

There was a suture in situ on the left side of the neck at the junction of the internal jugular vein and the subclavian vein.

There was no evidence of pulmonary oedema. The native kidneys were markedly contracted, scarred, and contained a number of cysts. Both ureters were hugely distended and dilated. The transplanted kidney was in the right pelvis, the ureter drained freely, and the vascular attachments were intact.

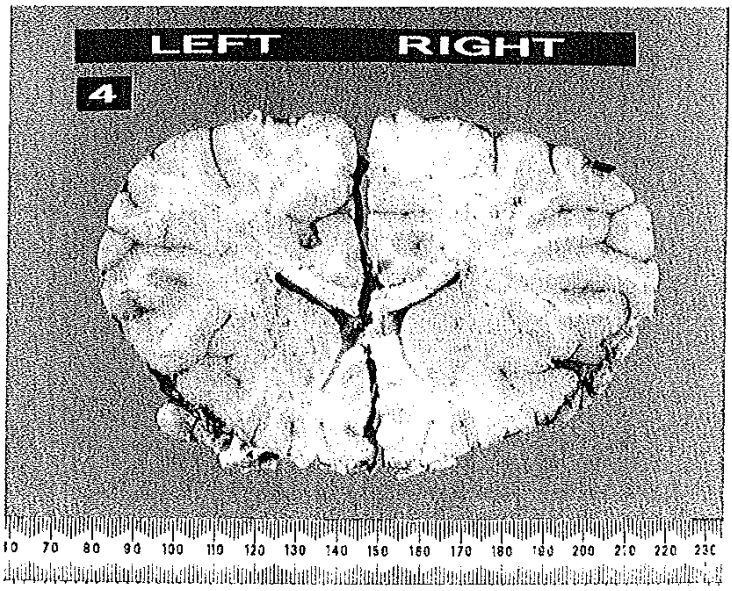


Figure 2 Cut section of brain showing slit-like ventricles.

Histologically there was massive oedema of the cerebral cortex and white matter. There was no evidence of terminal hypoxia or of central pontine myelinolysis.

Discussion

The signs of raised intracranial pressure include a rising blood pressure and falling pulse rate. Hyponatraemia, if chronic, may be asymptomatic and may not result in cerebral oedema.[3] However, the symptoms of hyponatraemia following intravenous administration of fluids include lethargy, headache, nausea, and emesis, with the onset of respiratory arrest.[4,5] In the anaesthetised and unconscious patient these symptoms are obviously masked. Hyponatraemia has been defined as a serum sodium level concentration 130 mmol/l[5] and severe hyponatraemia with a serum sodium concentration below 125 mmol/l.[6] The latter is associated with cerebral oedema[7,8] and it appears that it is the excess water content of the brain which is important in influencing consciousness.[9] In this case the serum sodium was 139 mmol/l at 2300 the night before the operation. At 0932 during the operation it had fallen to 123 mmol/l. It fell further to 119 mmol/l on admission to ICU around midday, and it did not rise again. At necropsy the brain was massively oedematous and when fixed it weighed 1680 g. The normal brain weight (unfixed) for boys aged four to five years is 1300 g. Thus the brain weight had increased by almost 30%, which is greatly in excess of other cases documented by Arieff *et al.*[4] In that study the investigators proposed that it was neither the actual concentration of the serum sodium nor the rapidity of the development of hyponatraemia that determined the ultimate outcome in these children. However, our case was associated with extremely rapid development of cerebral oedema and a correspondingly rapid fall in serum sodium concentration. It shows that dilutional hyponatraemia can occur intraoperatively, with rapidly fatal results. Dilutional hyponatraemia can occur with a minimal positive fluid balance when fluids containing small amounts of sodium are given, for example dextrose-saline as was given in this case.[4] It is the usual practice in routine elective operations to give Hartmann's solution, which contains no glucose. However, this case was not routine. It involved complex fluid management in a child who produced large quantities (75 ml/h) of dilute urine and was fed through a gastrostomy at night, therefore producing a high insulin requirement. Before the operation, to avoid the possibility of aspiration, the 1.5 litre feed was replaced by 900 ml of clear fluids. Therefore there was a fluid deficit before the operation. There was also concern about the intraoperative glucose management, and about the fact that renal transplant operations require adequate fluids for the graft to take. For these clinical reasons it was decided to use dextrose-saline though in retrospect it might have been better to give isotonic sodium chloride with glucose.

The brain was so grossly swollen at necropsy that it is possible that an additional factor was involved to account for the findings. The internal jugular vein on the left side had been tied off at its junction with the subclavian vein after the removal of a long line. This obstruction to the venous drainage from the brain could have led to increased engorgement of the brain which can be associated with oedema.[4] It is possible therefore that this factor may have played a role in the severity and rapid development of the cerebral oedema.

SUMMARY

Symptomatic dilutional hyponatraemia is a well recognised postoperative complication, particularly in children. This case shows that it can occur intraoperatively, thus masking the symptoms of cerebral oedema and the low serum sodium level. This makes its detection more difficult for the clinical staff involved, but for the pathologist the rapidity of onset with massive fatal cerebral oedema should be noted.

I thank Dr E Sumner, Consultant Paediatric Anaesthetist, Great Ormond Street Hospital, for his expert opinion, Dr Bob Taylor, Consultant Paediatric Anaesthetist, Royal Belfast Hospital for Sick Children, for his helpful comments, and HMC for Greater Belfast, Mr John Leckey, for his permission to use this case.

1 Arieff AI, Guisado R. Effects on the central nervous system of hypernatremic and hyponatremic states. *Kidney Int* 1976;10:104–16.
2 Wasterlain CG, Torack RM. Cerebral oedema in water intoxication: 11. An ultrastructural study. *Arch Neurol* 1968;19:79–87.
3 Fishman RA. Brain edema. *N Engl J Med* 1975;293:706–11.
4 Arieff AI, Ayus JC, Fraser CL. Hyponatraemia and death or permanent brain damage in healthy children. *BMJ* 1992;304:1218–22.
5 Arieff AI. Hyponatraemia, convulsions, respiratory arrest, and permanent brain damage after elective surgery in healthy women. *N Engl J Med* 1986;314:1529–35.
6 Worthley LIG, Thomas PD. Treatment of hyponatraemic seizures with intravenous 29.2% saline. *BMJ* 1986;292:168–70.
7 Rymer MM, Fishman RA. Protective adaptation of brain to water intoxication. *Arch Neurol* 1973;28:49–54.
8 Wasterlain CG, Posner JB. Cerebral oedema in water intoxication: 1. Clinical and chemical observations. *Arch Neurol* 1968;19:71–8.
9 Worthley LIG. Rapid correction of water intoxication by hypertonic saline and frusemide. *Aust NZ J Med* 1975;5:557–60.
10 Dekaban AS, Sadowsky D. Changes in brain weights during the span of human life: relation of brain weights to body heights and body weights. *Ann Neurol* 1978;4:345–56.

Witness Statement Ref. No. 012/1

NAME OF CHILD: Adam Strain

Name: Alison Armour

Title:

Present position and institution:

Previous position(s) and institution(s):
[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 2010]

Other Statements, Depositions and Reports:
[Identify by date and title all of those between January 1995-December 2010]

OFFICIAL USE:
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093-022	12.04.06	PSNI Witness Statement

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I QUERIES ARISING OUT OF YOUR AUTOPSY REPORT

With reference to your Autopsy Report dated 29th November 1995 (Ref: 011-010-034), please provide clarification and/or further information in respect of the following:

(1) *"The operation itself was technically difficult due to the previous surgical procedures and there was an increased blood loss calculated to be approximately 1,200mls. This was replaced by intravenous fluids of 500mls of Hartman's, 1,000mls HPPF and 500mls of packed cells"* **(Ref: 011-010-040)**

(a) **Explain the basis upon which you report the blood loss to be** *"approximately 1,200mls at the end of the procedure"*

I would have obtained this from the clinical notes.

(b) **Describe and explain how the administration of** *"intravenous fluids of 500mls of Hartman's, 1,000mls HPPF and 500mls of packed cells"* **constituted a 'replacement' for Adam's** *" blood loss calculated to be approximately 1,200mls"*

I would have obtained this from the clinical notes.

(2) *"(The brain, spinal cord and histological slides were seen by Dr. M. Mirakhur, Consultant Neuropathologist)"* **(Ref: 011-010-040)**

(a) **State the purpose for which Dr. M. Mirakhur was shown Adam's** *"brain, spinal cord and histological slides"*

Dr M. Mirakhur is a Consultant Neuropathologist. The slides would have been shown to her for a second opinion.

(b) **State what comments, analysis and/or reports Dr. M. Mirakhur provided in respect of** *"the brain, spinal cord and histological slides"* **that he saw, and identify where, if at all, you have incorporated/reflected them in your Report**

Dr Mirakhur is female. As far as I am aware what is written in my autopsy report was concurred by her.

(3) *"Transplanted kidney: There was complete infarction ... (The above slides were seen by Professor J. Berry, Consultant Paediatric Pathologist)"* **(Ref: 011-010-040)**

(a) **Describe, in respect of the transplanted kidney, the *"slides ... seen by Professor J Berry"***

Again the histological slides were sent to Professor Berry – Consultant Paediatric Pathologist for a second opinion. As far as I am aware his opinion concurred with mine.

(b) **Identify the site(s) on the transplanted kidney from which the tissue was taken for those slides and state what determined the selection of that site(s)**

I cannot recall the exact site where I took the histological sample/ samples. However it would have included a section of cortex and medulla.

(c) **State where any tissue blocks and/or slides in respect of the transplanted kidney are currently held**

Any slides or tissue blocks will be held at the State Pathologist Department Belfast.

(4) ***"In this case the volume of urine output was greatly increased"*** **(Ref: 011-010-041)**

(a) **Explain the basis upon which you state that Adam's *"volume of urine output was greatly increased"*, including:**

- **his volume of daily urine output**
- **the extent to which that represented a *"greatly increased"* volume**

Regarding Adam Strain's urinary output and that this was greatly increased this information would have been determined from the clinical notes. Without access to these notes it is not possible for me to state to what extent.

(5) ***"Another factor to be considered in this case is cerebral perfusion. The autopsy revealed ligation of the left internal jugular vein ... This would mean that the cerebral perfusion would be less than that in a normal child. This would exacerbate the effects of the cerebral oedema and should also be considered as a factor in the cause of death. Therefore the most likely explanation is that the cerebral oedema followed a period of hyponatraemia and was compounded by impaired cerebral perfusion."*** **(Ref: 011-010-041)**

(a) **Describe and explain in detail why you reported that there was a *"ligation of the left internal jugular vein"***

I reported ligation of the left internal jugular vein as this is/was a fact. I refer you to my post mortem report under internal examination and specifically internal examination of neck on page 4.

(b) **State and explain when you believe the *"ligation of the left internal jugular vein"* was carried out**

It was my understanding that this had been carried out after the removal of a long line.

(c) **Describe and explain the impairment in Adam's cerebral perfusion that you consider occurred secondary to this vein ligation**

I have already clearly answered the question you ask of me. This has been clearly addressed in my autopsy report.

(d)**Describe and explain the extent to which the *"ligation of the left internal jugular vein"* gave rise to *"cerebral perfusion ... less than that in a normal child"***

I have clearly answered the question you ask of me. This has been clearly addressed in my autopsy report.

(6) ***"The autopsy also revealed changes in the kidneys, in keeping with chronic renal failure and total infarction of the transplanted kidney."*** **(Ref: 011-010-041)**

(a) **Explain what you mean by *"total infarction of the transplanted kidney"***

Dead.

(b)**State what you consider caused that *"total infarction"***

Not sure.

(c) **Explain whether you were or are now able to estimate when that infarction occurred.**

No.

II QUERIES ARISING OUT OF YOUR DEPOSITION

With reference to your Deposition to the Coroner taken on 18th June 1996 (Ref: 011-010-030), please provide clarification and/or further information in respect of the following:

(8) ***"This was massive cerebral oedema and I have never come across anything of a similar degree. The cause of it in this case is extremely rare and never encountered by me previously. On a worldwide basis it would be equally rare."*** **(Ref: 011-010-033)**

(a) **Explain what you considered to have been the *"cause"* of Adam's *"massive cerebral oedema"* and the basis upon which you reported that the *"cause of it ... is extremely rare"***

I have clearly addressed the cause of this little boy's massive cerebral oedema. This is clearly and concisely laid out in the cause of death on the very first page of my autopsy report. In a child undergoing renal transplantation/ operative intervention this is extremely rare. I was unaware of any other case at the time. From my reading of the literature I am still of the view that it is extremely rare today. My understanding of dilutional hyponatraemia was that in the main it was a post-operative complication.

(b) **Explain the basis upon which you reported that *"On a worldwide basis it would be equally rare"***

From my reading of the literature.

(c) **Describe in detail the education and training you received in fluid management (in particular hyponatraemia) and record keeping through the following, providing dates and names of the institutions/bodies:**

- **Undergraduate level**
- **Postgraduate level**
- **Hospital induction programmes**
- **Continuous professional development**

I am a pathologist and as such do not undergo training for the fluid management of patients. This is not my job.

(d) **Prior to the date of the autopsy that you carried out on Adam on 29th November 1995, describe in detail your experience of conducting autopsies on children in the circumstance set out below, including providing the estimated number of such cases and the dates when they took place:**

- **who had undergone renal transplant surgery immediately prior to their death**
- **where cerebral oedema had caused or contributed to their death**
- **where hyponatraemia had caused or contributed to their death**

I had carried out many autopsies on children prior to that on Adam Strain. As this case occurred a long time ago I cannot recall whether cerebral oedema had caused or contributed to the death of these children. I cannot remember the details of every case I did in the past. However, as far as my memory serves me I am unaware of a case where dilutional hyponatraemia had caused the death of a child or contributed to it to any great extent. This was the first case I had undertaken of a child dying after renal transplant surgery. To date I have not carried out any such other case. Other than being allowed access to all my cases and reports that I carried out during my time in Northern Ireland it is not possible for me to answer the second part of this question accurately.

(9) *"He [Adam] experienced substantial blood loss during the operation and that made his haemodynamics very difficult to manage."* (Ref: 011-010-033)

(a) **Explain what you mean by *"made his haemodynamics very difficult to manage"* together with the effect, if any, that you consider it had on Adam's fluid management during his transplant**

By the expression haemodynamics I mean the management of his blood pressure, blood loss, fluid loss, replacement therapy and cardiovascular status.

(10) *"A critical point was the fluids used by the anaesthetist to replace blood loss ... Dr. Taylor advised me at the autopsy of the calculation he made to replace blood loss. Haematocrit = packed cell volume."* (Ref: 011-010-033)

(a) **State whether Dr. Taylor was present during the autopsy that you carried out on Adam on 29th November 1995 and if not explain the circumstances in which Dr. Taylor advised of the *"calculation he made to replace blood loss"***

I cannot recall whether Dr Taylor was present during the autopsy on Adam Strain or not. This would be in my contemporaneous notes. I do not have access to these notes nor are they in my possession. They will be held at the State Pathologists Department Belfast. I cannot recall the circumstances in which Dr Taylor advised of the calculated blood loss without access to contemporaneous notes.

(b) State what Dr. Taylor advised you was *"the calculation he made to replace blood loss"*, including the type and volume of the fluid(s)

Dr Taylor would have made this calculation and told me of his calculation.

(11) *"At the autopsy I had 10 sets of notes relating to Adam and the clinicians' statements"* (Ref: 011-010-033)

(a) Identify the *"10 sets of notes relating to Adam"* that you had at the autopsy

The notes I received prior to the autopsy would have been sent to me in the mortuary. I cannot list the notes now in 2011. I have no problem in recalling there were numerous hospital notes accompanying the body of this child.

(b) Identify the clinicians' statements that you had at the autopsy

I need the contemporaneous notes to answer this question. I cannot recall this from memory all those years ago.

(12) *"There was impaired cerebral perfusion as there was a suture on the left side and a catheter tip on the right ... The suture impaired blood flow to the brain and the catheter tip on the right may have had a role to play. The suture had been there for some time"* (Ref: 011-010-033)

(a) With reference to the *"suture had been there for some time"*, state the date when *"the suture on the left hand side"* was inserted

I cannot answer this question.

(b) Describe and explain the degree and extent to which *"The suture impaired blood flow to the brain"* and the basis on which you were able to estimate that degree and extent

I have already clearly answered this question in my autopsy report.

(c) Describe and explain what you meant by *"the catheter tip on the right may have had a role to play."*

I have already clearly explained this.

III QUERIES ARISING OUT OF YOUR PSNI WITNESS STATEMENT

With reference to your PSNI Witness Statement dated 12th April 2006 (Ref: 093-022-062), please provide clarification and/or further information in respect of the following:

(13)*"D/Sergeant Cross has shown me a letter numbered 059-036-072. I would state that my opinion was honestly held at the time and remains so now, based on the facts provided to me. I have not misrepresented any fact nor have I behaved in a prejudicial manner"* (Ref: 093-022-063)

(a) Comment on the claims he made in that letter in respect of:

- **sodium content of the fluids administered**
- **jugular ligation and both 'impaired cerebral perfusion' and 'impaired cerebral drainage'**
- **the examination of the neck showing no evidence of congestion or obstruction of the major blood vessels and the conclusion that cerebral perfusion could have been impaired**

I cannot answer the question regarding the sodium content of the fluid administered other than to state that dextrose saline contains small amounts of sodium. I have clearly explained the jugular ligation and its role in cerebral perfusion. The third part of this question is only indeed part of what I have clearly said as already stated laid out in my autopsy report at the top of page 4.

IV ADDITIONAL INFORMATION

(14)Provide any further points and comments that you wish to make, together with any documents, in relation to:

(a) Record keeping

All records held at the State Pathologists Department Belfast.

(b)Lessons learned from Adam's death and its effect on your work

I published a paper in the Journal of Clinical Pathology as a specific case report and using this case. The object of the publication was to ensure that this should not happen again. However, I am aware that the Journal is read mainly by pathologists. I enclose a copy for completeness. Dilutional hyponatraemia: a cause of massive fatal intra operative cerebral oedema in a child undergoing renal transplantation. Dr A. Armour. The Journal of Clinical Pathology May 1997. Volume 50. Number 5. Pages 444-446.

(c) Current 'protocols' and procedures

I am a pathologist and do not treat patients nor do I give fluid replacement therapy.

(d) Any other relevant matter

None.

V DECLARATION OF INTEREST

(15) Confirm that you have completed and signed the attached 'Declaration of Interest'

Yes. I have had no contact with the individuals that you have listed on the declaration of interest form. However, you will see from my published paper that I acknowledged Dr Sumner Consultant Paediatric Anaesthetist for his expert opinion and Dr Robert Taylor for his helpful comments. I also acknowledged Her Majesty's Coroner for Greater Belfast Mr John Lecky for his permission to use the case. This was my only contact with these individuals regarding this paper. This statement is true to the best of my knowledge and belief.

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

Signed: Dated: