Cof 77

Form 49/1

POLICE SERVICE OF NORTHERN IRELAND

FACSIMILE MESSAGE

To DR ARMOUR
Address DEPT OF CELLULAR PATHOLOGY, ROYAL PRESTON HOSPITAL
Fax. No. [redacted] Date/Time 16/3/06 1415
For Attention of/Message

DR ARMOUR

as discussed with secretary by telephone this date.

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Making Northern Ireland Safer For Everyone Through Professional, Progressive Policing

16 March 2006

Dr Armour
Consultant Pathologist
Royal Preston Hospital
Sharoe Green Lane North
Fulwood
Preston
PR2 4HG

Dear Dr Armour

RE ADAM STRAIN, DIED NOVEMBER 1995

I have spoken today to your secretary and she informed me that you wished a copy of the PM report. I have faxed it with this letter.

I have also faxed other documents which we will refer to when we meet. I have highlighted in the margin the relevant sections.

I have received your report to the Journal of Clinical Pathology. I have also tentatively arranged an interview for 11 April 2006, but will confirm by telephone.

If I can be of any further help, please contact me at the postal or email addresses below, or on [redacted]

Yours sincerely

William R Cross

WILLIAM R CROSS
D/SERGEANT

Fermanagh District Command Unit
48 Queen Street, ENNISKILLEN, BT74 7JR Web: www.psni.police.uk
Tel: [redacted] Fax: [redacted] E-mail: fermanagh[redacted]
Billy.Cross[redacted]

No. 20

CORONERS ACT (Northern Ireland), 1959

Deposition of Witness taken on TUESDAY the 18TH day of JUNE 1996, at inquest touching the death of ADAM STRAIN, before me MR J L LECKEY Coroner for the District of GREATER BELFAST as follows to wit:-

The Deposition of DR ALISON ARMOUR
of INSTITUTE OF STATE PATHOLOGY *(Address)*
who being sworn upon her oath, saith

On the instructions of HM Coroner for Greater Belfast Mr J L Leckey LLM, I Alison Armour, MB,BCh, MRCPath, DMJ (Path) registered medical practitioner and pathologist approved by the Northern Ireland Office made a postmortem examination on a body identified to me as that of Adam Strain. I now produce a copy of my report marked C1. 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.

Mr. Bingham: It was a complex case because of Adam's underlying condition, his previous surgery and the technical difficulty of the operation, he experienced substantial blood loss during the operation & after ~~caused~~ made his haemodynamics very difficult to manage. Adam was not a healthy child — he was a sick little boy.

139 mmol/l is within the normal range. So far as no significant oedema of any other organ my understanding is that fluid is absorbed into the brain in preference to any other organ. I distinguish between hyponatraemia & dilutional hyponatraemia. The latter is due to fluid given. Children are more susceptible to

TAKEN before me this 18th day of JUNE 1996

A.L. Leckey

Coroner for the District of Greater Belfast

17

CORONERS ACT (Northern Ireland), 1959 No.

Deposition of Witness taken on the d
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Coroner for the District of
as follows to wit:—

The Deposition of DR ALISON ARMOUR

of

who being sworn upon her oath, saith (Address

cerebral oedema and then adults and so far as dilutional hyponatraemia females are more susceptible than males. The paper I refused to refer to healthy children but it is still a good reference to this condition. There was impaired cerebral perfusion as there was a suture on the left side and a catheter hip on the right. 1200 mls blood loss during the operation. I do not know what problems this would have caused for the anaesthetist.

Mrs Higgins: A critical point was the fluids used by the anaesthetist to replace blood loss. At the autopsy I had 10.2kg weight relating to Adam and the clinicians' statements. The suture impaired the blood flow to the brain and the catheter hip on the right may have had a role to play. The suture had been there for some time. Dr Taylor advised me at the autopsy of the calculation he made to replace blood loss. Haematocrit = packed cell volume. In this case the reading could indicated he was bleeding at a dilutional state.

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P.T.O.

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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.

Mr Brangham: It was a complex case because of Adam's underlying condition, his previous surgery and the technical difficulty of the operation. He experienced substantial blood loss during the operation and that made his haemodynamics very difficult to manage. Adam was not a healthy child - he was a sick little boy. 139 mml/l is within the normal range. So far as no significant oedema of any other organ my understanding is that fluid is absorbed into the brain in preference to any other organ. I distinguish between hyponatraemia and dilutional hyponatraemia. The latter is due to fluids given. Children are more susceptible to cerebral oedema than adults and so far as dilutional hyponatraemia females are more susceptible than males. The paper I referred to refers to health children but it is still a good reference to this condition. There was impaired cerebral perfusion as there was a suture on the left side and a catheter tip on the right. 1200 mls blood loss during the operation. I do not know what problems this would have caused for the anaesthetist.

Miss Higgins: A critical point was the fluids used by the anaesthetist to replace blood loss. At the autopsy I had 10 sets of notes relating to Adam and the clinicians' statements. The suture impaired the 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. Dr Taylor advised me at the autopsy of the calculation he made to replace blood loss. Haematocrit = packed cell volume. In this case the reading could indicate he was bleeding or in a dilutional state.

C1

THE QUEEN'S UNIVERSITY OF BELFAST
NORTHERN IRELAND OFFICE

REPORT OF AUTOPSY

Name: Adam STRAIN **Sex:** Male **Age:** 4 yrs. **F.No:** 46,728

Date of Death: 28th November, 1995. MDEC

Date and Hour of Autopsy: 29th November, 1995. 2.40 p.m.

Place of Autopsy: The Mortuary, Royal Victoria Hospital, Belfast.

CAUSE OF DEATH:

I (a) CEREBRAL OEDEMA

due to

(b) DILUTIONAL HYPONATRAEMIA AND IMPAIRED CEREBRAL PERFUSION DURING RENAL TRANSPLANT OPERATION FOR CHRONIC RENAL FAILURE (CONGENITAL OBSTRUCTIVE UROPATHY)

On the instructions of H.M. Coroner for Greater Belfast, Mr. J. L. Leckey, LLM, I, Alison Armour, MB, BCh, MRCPath, DMJ(Path), registered medical practitioner and pathologist approved by the Northern Ireland Office, made a postmortem examination of the body of -

ADAM STRAIN
aged 4 years

identified to me at the Mortuary, Royal Victoria Hospital, Belfast, on Wednesday, 29th November, 1995, by Constable S. R. Tester, R.U.C. Grosvenor Road.

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THE QUEEN'S UNIVERSITY OF BELFAST
NORTHERN IRELAND OFFICE

REPORT OF AUTOPSY

Name: Adam STRAIN Sex: Male Age: 4 yrs. F.No: 46,728

Date of Death: 28th November, 1995. MDEC

Date and Hour of Autopsy: 29th November, 1995. 2.40 p.m.

Place of Autopsy: The Mortuary, Royal Victoria Hospital, Belfast.

HISTORY:

He was a child and lived with his mother and grandparents in a bungalow in the town. He was born with a renal abnormality - an obstructive uropathy which resulted in polyuric renal failure. He had five ureteric reimplant operations, a fudoplication for gastro-oesophageal reflux and more recently in October, 1995 an orchidoplexy. He ate nothing by mouth and was fed via a gastrostomy button 1,500 mls. at night and 900 mls. during the day. He also received peritoneal dialysis. He was being prescribed calcium carbonate, Keflex, iron, one alpha vitamin, sodium bicarbonate and erythropoietin.

On 26th November, 1996, he was admitted to the Royal Belfast Hospital for Sick Children at 11.30 p.m. for a renal transplant operation. His blood pressure was 108/56 and a haemoglobin of 10.5 g/dl with a sodium of 139 mmol/1, potassium 3.6 mmol/1 and urea 16.8 mmol/l. Overnight he was given 900 mls. dioralyte (4% dextrose 0.18% saline). Peritoneal dialysis was performed as usual , 750 ml. fluid volume 1.36% dextrose solution. He was given 8 cycles before going to theatre the next morning.

He arrived in theatre at 6.45 a.m. and general anaesthesia was induced using thiopentone, atropine and atracium. Intravenous access was difficult and attempts were made to pass a central venous pressure catheter. Three attempts were made with the left subclavian vein, one with the left internal jugular vein and then the catheter was successfully passed into the right subclavian vein. A lumbar epidural between L1 and L2 was also sited with 0.25% bupivacaine and Fentanyl 5 mcg/kg. Apart from the anaesthetic drugs Augmentin an antibiotic, prednisolone, asathioprin (anti-rejection drug) and a continuous infusion of dopamine were administered intravenously. An initial central venous pressure reading was taken at 17 mm.Hg. Intravenous units were administered from 7.00 a.m. to 8.30 a.m., of three 500 ml. bags of dextrose saline (4% and 0.18%). The operation technically was difficult due to previous surgical procedures and there was an increase in blood loss, calculated to be approximately 1,200 mls. at the end of the procedure. Further fluids of 500 mls. Hartman's solutions 1,000 mls. of HPPF (human plasma protein fraction) and 500 mls. of packed cells were administered. At 9.32 a.m. a blood gas analysis revealed a sodium of 123 mmol/l (normal 135 - 145) and a haematocrit of 18% (normal. 35 - 40%). During the procedure the CVP rose to 20 -21 mm.Hg, the Hb was 6.1 g/dl which was 10.1 g.dl. at the end of the procedure and the blood pressure rose and the pulse rate gradually decreased. The donor kidney perfused and the operation was completed. At the end of the procedure the neuromuscular block was reversed with neostigmine but this boy did not wake up. His pupils were noted to be fixed and dilated at midday. He was transferred from theatre to the paediatric Intensive Care Unit at 12.05 p.m. He was intubated and hand ventilated on admission. He was treated with intravenous mannitol and intravenous fluids were restricted. An emergency CT scan at 1.15 p.m. revealed gross cerebral oedema. His body temperature was 36.5°C. the CVP was 30, heart rate 120 beats per minute and systolic blood pressure 120. Electrolytes revealed a

sodium of 119 mmol/l; and a chest X-ray revealed pulmonary oedema with the CVP catheter tip in a neck vessel. Neurologists carried out brain stem tests and life was pronounced extinct by a hospital doctor on 28th November, 1995 at 9.15 a.m.

EXTERNAL EXAMINATION:

The body of a young male child, 104 cm. in length and weighing 20 kilograms. Rigor mortis was present. Hypostasis of light purple colour stained the back of the body.

Back: There was a needle puncture mark in the midline, centred 11 cm. above the natal cleft, corresponding to an epidural cannula.

Eyes: The corneas had been taken for transplantation.

Ears: Normal.

Nose: Normal.

Neck: There was a needle puncture mark on the left side. There was a healed operation scar, 3 cm. long, on the left side. There were two further healed operation scars on the right side, 2.5 cm. long.

Chest : There was a needle puncture mark on the left upper chest, in the region of the subclavian vein. There were a number of bruised needle puncture marks on the right upper chest, corresponding to a subclavian line .There was a bruise, 1.5 x 1 cm., in the left upper chest, centred 3 cm. lateral and 1 cm. above the left nipple. There was a bluish-blackish bruise on the right chest, 2.5 x 1 cm., diameter, centred 3 cm. lateral to the right nipple.

Abdomen: There was a gastrostomy button situated in the left hypochondrium. The gastrostomy hole measured 6 mm. diameter. There was a healed operation scar, 18 cm. long, horizontally in the upper abdomen, corresponding to previous fundoplication. There was a further healed operation scar, 18 cm. long, traversing the mid-abdomen. There was a peritoneal dialysis tube in situ in the left upper abdomen. There were two further puckered scars, one situated in the left side of the lower abdomen, 5 cm. lateral and 2 cm. below the umbilicus. The other puckered scar was situated 4.5 cm. beneath the umbilicus. There was a recent elliptical surgical incision, 15 cm. long, on the right side of the lower abdomen with a drain protruding from its upper margin. Its edges were slightly bruised. A bladder catheter protruded from the lower end on the left side of the abdomen. There was a further drain in situ just at the level of the pubic bone, corresponding to the donor ureteric catheter.

Left Upper Limb: There were a number of bruised needle puncture marks in the fold of the elbow and a healed operation scar, 5 cm. long, again in the fold of the elbow.

Right Upper Limb: There were a number of bruised needle puncture marks in the fold of the elbow.

Left Lower Limb: There were a number of petechial bruises on the inner aspect of the thigh, in an area 4 x 1 cm. There was a bruise, 1 cm. diameter, on the front of the shin. There was a bruised needle puncture mark on the dorsum of the foot.

Right Lower Limb: There was a healed operation scar, 4 cm. long, in the right groin, corresponding to an orchidoplexy. There was a fading bruise, 0.5 cm. diameter, on the outer aspect of the upper thigh. There was a bluish bruise on the outer aspect of the thigh, 0.5 cm. diameter, and there were a number of fading bruises on the front of the shin. There were two bruised needle puncture marks on the dorsum of the foot.

Scrotum: There was a healed operation scar, 3 cm. long, on the right scrotal sac. The right testis had been removed. The left testis was present

INTERNAL EXAMINATION:

HEAD:

Brain: To be described after fixation.

Mouth: There were natural teeth in good condition in each jaw. The lips were dry and parchmented. The tongue was held between the clenched teeth.

Tongue, Pharynx: Normal.

NECK AND CHEST:

Hyoid Bone and Laryngeal Cartilages: Intact.

Thyroid Gland: Normal.

Pericardial Sac: Normal.

Heart: 120 gm. The organ was taken for transplantation.

Aorta: Normal.

ABDOMEN:

Abdominal Cavity: Was crossed by a number of adhesions. There was a little blood clot formation around the renal transplant on the right side.

Stomach: A gastrostomy hole was present. The stomach contained a little bile.

Intestines: Externally appeared normal.

Duodenum: Normal.

Liver: Weighed 875 gms. A little congested.

Gall Bladder: Normal.

Pancreas: Normal.

Native Kidneys: Both were markedly contracted, scarred and contained a number of cysts. Little normal functioning kidney remained. Both ureters were hugely distended and dilated.

Transplanted kidney: Was in situ in the right pelvis, the ureter drained freely and the vascular attachments were intact.

Bladder: Contained a little straw-coloured urine.

Prostate: Normal.

SPINAL CORD: To be described after fixation.

INTERNAL EXAMINATION OF NECK:

There was no evidence of congestion or obstruction of the major blood vessels or the carotid arteries and jugular veins. There was no evidence of superior vena caval obstruction. The carotid arteries were normal. There was a suture in situ on the left side of the neck at the junction of the internal jugular vein and the sub-clavian vein.

DESCRIPTION OF ORGANS AFTER FIXATION:

Brain - Was cut on 12.1.96

External Examination: Fixed weight of brain 1,680 gm; cerebellum and brain stem 176 gm; cerebellum only 154 gm. The brain was grossly swollen with loss of sulci and uncal swelling. This was symmetrical. There was no uncal necrosis. There was swelling of the cerebellar tonsils but no necrosis. There was no cortical venous thrombosis. The anatomy of the circle of Willis was normal.

On cut section there was massive brain swelling and constriction of the ventricles. There was no ventricular haemorrhage. There was no asymmetrical lesion. There was severe white matter congestion and marked congestion of the blood vessels in the basal ganglia, white matter and deep grey matter. There was no necrosis of the mid-brain or brain stem.

Blocks were taken from:

1. Right frontal white matter
2. Left cingulate gyrus
3. Left basal ganglia
4. Right and left hippocampus
5. Left occipital lobe
6. Cerebellum
7. Pons in toto
8. Thalamus

The brain was photographed sequentially

Cervical Cord: No macroscopical lesion seen.

Blocks were taken from:

1. Cervical
2. Thoracic
3. Lumbar

MICROSCOPY:

Lungs: There was congestion of the capillaries and there were moderate numbers of alveolar macrophages. There was no evidence of embolism or infarction.

Larynx: There was ulceration of the mucosa, in keeping with intubation.

Liver: There was no evidence of cyst formation within the portal tract. There were scattered foci of clear cell change.

Kidney: There was widespread scarring and cyst formation, interstitial fibrosis and chronic inflammation. There was widespread glomerulo-sclerosis and the arterioles were thickened.

Transplanted Kidney: There was complete infarction.

Spleen: There was congestion of the red pulp.

Lymph Node: Normal.

(The above slides were seen by Professor J. Berry, Consultant Paediatric Pathologist).

Brain: There was massive cerebral oedema of the cortex and white matter. There was no evidence of terminal hypoxia. There was no evidence of myelinolysis.

Spinal Cord: No specific pathological features were noted.

(The brain, spinal cord and histological slides were seen by Dr. M. Mirakhur, Consultant Neuropathologist)

COMMENTARY:

This little boy with a past medical history of polyuric renal failure, numerous hospital admissions and operations was admitted to hospital one evening for a renal transplant operation. He was fed via a gastrostomy and ate nothing by mouth. Usually he would receive 1,500 mls. a night and ~~900~~ 600 mls. during the day. That night investigations included blood pressure 108/56, sodium 139 mmol/l and haemoglobin 10.5 g/dl. Overnight he was given 900 mls. dioralyte (4% dextrose 0.18% saline) and peritoneal dialysis was performed as usual. He went to theatre the next morning.

General anaesthesia was induced. Intravenous access was difficult and four attempts were made to pass a central venous pressure catheter before it was successfully passed into the right subclavian vein. A lumbar epidural was also sited with .25% bupivacaine and fentanyl. An initial CVP reading was taken at 17 mm.Hg. and intravenous fluids were given of 3 x 500 ml. bags of dextrose saline (4% and .18%). The operation itself was technically difficult due to the previous surgical procedures and there was an increased blood loss calculated to be approximately 1,200 mls. This was replaced by intravenous fluids of 500 mls. of Hartman's, 1,000 mls. HPPF and 500 mls. of packed cells. At 9.32 a.m. a blood gas analysis revealed a sodium of 123 mmol/l (normal 135-145) and a low haematocrit. During the operation the CVP increased to 20-21 mm.Hg., the haemoglobin fell to 6.1 g/dl., the systolic blood pressure rose to 150 mm.Hg. and the pulse gradually fell but rose steadily from 10.15 a.m. onwards. When the procedure was completed and the neuromuscular block was reversed this little boy did not wake up. A CT scan of the brain revealed gross cerebral oedema. Brain stem function tests were carried out and he was declared dead a little over 26 hours from the start of the operation.

The autopsy revealed gross cerebral oedema. The fixed weight of the brain at postmortem was 1,680 gms., the average weight for a boy of this age being 1,300 gms and the average weight of a man's brain being 1,450 gms. It was the effects of this massive swelling of the brain which caused his death. There was no significant oedema of any other organ.

This is a highly complex and difficult case. To try to understand the underlying cause for this cerebral oedema first some physiological mechanisms for maintaining fluid and electrolyte balance will be reviewed.

In healthy people the composition of body fluids vary within narrow limits. The kidneys are largely responsible for maintaining this constancy and the excretion of waste products of metabolism represents merely one aspect of this task. The control of water volume and sodium are maintained by the hormones A.D.H. (anti-diuretic hormone) and aldosterone.

In this case the volume of urine output was greatly increased and the urine was also dilute. This was probably due to the fact that the kidneys did not function and their ability to concentrate the urine was minimal.

Generalised cerebral oedema in children has many causes including hypoxia. In this case this has been excluded. The history indicates that during the operation this little boy received a quantity of intravenous fluids. There was also a considerable blood loss during the operation of 1,200 mls. However a CVP, central venous pressure, catheter was in situ in the right subclavian vein and is usually in place to avoid overloading of the circulation by intravenous fluids. A rise in the CVP indicates an excessive load and a fall can be an early sign of haemorrhage. In this case the initial reading was 17 mm.Hg. (for an operation such as this 10-12 mm.Hg. is the norm) and this was taken as the base line. A subsequent reading was a little higher again. Also during the operation the sodium was low along with the haematocrit. It is known that a condition called dilutional hyponatraemia can cause rapid and gross cerebral oedema. This is no doubt in this case that the sodium level was low during the operation. A study revealed that in children undergoing operations there was substantial extra renal loss of electrolytes and with a minimal positive balance of hypotonic fluid could lead to fatal hyponatraemia. This study however must be taken in context as it refers to healthy children undergoing operations like tonsillectomies. Thus they had normally functioning kidneys which was not the situation in this case. It seems likely therefore that the hyponatraemia in this case was the cause of the cerebral oedema and most of the intravenous fluids given in the cases sited in this paper were administered as 280 mmol glucose per litre in water or in sodium chloride 38 mmol/l.

Another factor to be considered in this case is cerebral perfusion. The autopsy revealed ligation of the left internal jugular vein. The catheter tip of the CVP was situated on the right side. 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.

The autopsy also revealed changes in the kidneys, in keeping with chronic renal failure and total infarction of the transplanted kidney. These played no part in the fatal outcome.

There were marks due to treatment and bruises to both legs. They were trivial however.

REFERENCES:

Arieff et all
"Hyponatraemia and death or permanent brain damage in healthy children"
British Medical Journal 1992; 304; 1218-22

State Pathologist
Professor J Crane
MB BCh MRCPath DMJ (Clin et Path)

The Queen's University of Belfast &
Northern Ireland Office

State Pathologist's Department,
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Tel. [redacted] (Direct) or
Ext. [redacted] and [redacted]
Fax. [redacted]

JC/MDEC

20th December, 1995.

Dr. E. Sumner,
Hospital for Children,
NHS Trust,
Great Ormond Street,
LONDON WC1N 3JH

Dear Dr. Sumner,

Following our recent telephone conversation I should be grateful if you would provide an expert opinion on my case - Adam Strain, for H. M. Coroner for Greater Belfast, Mr. J. L. Leckey, LLM.

Please find enclosed:

1. The original hospital notes. (TAB 2)
2. Two reports from the consultant anaesthetist involved.
3. A report from the consultant paediatric nephrologist.
4. Equipment check report. (TAB 7)

To summarise:

This 4-year old child with a history of polyuric renal failure due to posterior urethral valves was admitted for a renal transplant. He had had a number of operations in the past including five fundoplications and more recently an orchidoplexy. All were uneventful. He ate nothing by mouth and as such was fed via a gastrostomy button which would include a night feed of 1,500 mls.

The operation itself produced a little more bleeding than expected and technically it was apparently a little more difficult than usual because this child was well nourished. When the operation was completed this child did not wake up. An urgent CT scan one hour later showed gross cerebral oedema. He was ventilated for about another 24 hours before the ventilator was turned off.

011-032-145

Findings at autopsy:

1. Gross cerebral oedema (brain still fixing along with spinal cord) with the brain bulging through the dura.

2. No substantial pulmonary oedema or oedema of any other organ.

I should be grateful if you could provide us with an opinion in this case.

Yours sincerely,

Alison Armour
Senior Registrar

Encls:

c.c. Mr. J. L. Leckey, H. M. Coroner for Greater Belfast.

011-032-146

tions. Home monitoring of blood glucose concentrations is economically impracticable for most patients, but easier access to urine dipsticks would probably increase patients' interest and motivation in improved control and would not add greatly to total direct costs.

The need for inpatient admission should also be considered carefully, especially for newly presenting patients. Wherever possible admission is best avoided if the patient and family are able to receive initial daily outpatient education and supervision.[11] Patients should be admitted only if they require nursing care or circumstances do not permit easy attendance at outpatient clinics. Admission rates for diabetic patients in Tanzania are six times higher than in the general population.[4] When patients are admitted careful consideration should be given to the need for investigations. Testing urine four times or more daily for example, may be unnecessary if blood glucose concentrations are also being measured. Consideration should also be given to the period of admission since patients are often kept in the wards until most urine results are glucose free.

The small proportion of direct costs due to nurses' and doctors' services reflects the low rates of pay of medical staff in most sub-Saharan countries. A lecturer in medicine, for example, is paid $60 monthly. The reasons for such low rate of remuneration are understood, but attention must also be paid to this problem since the motivation and interest of those caring for patients can have a significant impact on the quality of care.

United Republic of Tanzania, the British Council, and the Overseas Development Administration.

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(Accepted 24 February 1992)

Hyponatraemia and death or permanent brain damage in healthy children

Allen J Arieff, J Carlos Ayus, Cosmo L. Fraser

Abstract

Objective—To determine if hyponatraemia causes permanent brain damage in healthy children and, if so, if the disorder is primarily limited to females, as occurs in adults.

Design—Prospective clinical case study of 16 affected children and a review of 24 412 consecutive surgical admissions at one medical centre.

Patients—16 children (nine male, seven female; age 7 (SD 5) years) with generally minor illness were electively hospitalised for primary care. Consultation was obtained for the combination of respiratory arrest with symptomatic hyponatraemia (serum sodium concentration ≤128 mmol/l).

Main outcome measures—Presence, gender distribution, and classification of permanent brain damage in children with symptomatic hyponatraemia in both prospective and retrospective studies.

Results—By retrospective evaluation the incidence of postoperative hyponatraemia among 24 412 patients was 0·34% (83 cases) and mortality of those afflicted was 8·4% (seven deaths). In the prospective population the serum sodium concentration on admission was 138 (SD 2) mmol/l. From three to 120 inpatient hours after hypotonic fluid administration patients developed progressive lethargy, headache, nausea, and emesis with an explosive onset of respiratory arrest. At the time serum sodium concentration was 115 (7) mmol/l and arterial oxygen tension 6 (1·5) kPa. The hyponatraemia was primarily caused by extrarenal loss of electrolytes with replacement by hypotonic fluids. All 16 patients had cerebral oedema detected at either radiological or postmortem examination. All 15 patients not treated for their hyponatraemia in a timely manner either died or were permanently incapacitated by brain damage. The only patient treated in a timely manner was alive but mentally retarded.

Conclusions—Symptomatic hyponatraemia can result in a high morbidity in children of both genders, which is due in large part to inadequate brain adaptation and lack of timely treatment.

Introduction

In previous studies from our laboratories we have described the symptomatology, clinical course, effects of treatment, and pathological findings in more than 225 adults (aged over 16) with symptomatic hyponatraemia.[1-4] Although the actual incidence of hyponatraemia seems to be similar among men and women,[3,5] almost all adult patients suffering hyponatraemic brain damage are women. Although there are a number of reported paediatric cases of hyponatraemia,[6-11] there are few reported cases of death or permanent brain damage among children with this disorder,[9,10] and most such children had pre-existing neurological disorders.[9,10] Neither the gender distribution nor the incidence of brain damage among children with hyponatraemia is known.[6,10,11] Among children suffering brain damage from hyponatraemia neither the type nor the gender distribution is known. We describe both a prospective and a retrospective analysis of generally healthy children who were elective

University of California School of Medicine, San Francisco, California
Allen J Arieff, *professor of medicine*
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lively hospitalised. Sixteen children who developed severe symptomatic hyponatraemia either died or suffered permanent brain damage. Unlike the situation in adults, both males and females were adversely affected among these children.

Patients and methods

Prospective studies—Over a period of six years (1984-90) we were consulted about 16 previously healthy children (aged under 16) who had developed symptomatic hyponatraemia and either died or suffered permanent brain damage. These 16 patients were seen in consultation from five tertiary and nine community hospitals. The age of the children was 7 (SD 5) years (range 1·5 to 15 years), and the gender distribution was nine males and seven females. The mean weight was 23·8 (12·9) kg (range 10 to 52 kg). Symptomatic hyponatraemia developed within five days of admission to the hospital.

Epidemiological studies—We retrospectively studied all surgical admissions to a 456 bed tertiary paediatric university teaching hospital over three years (1989-91). The records of all paediatric (age under 16) surgical patients were evaluated for those who had postoperative hyponatraemia (serum sodium concentration 128 mmol/l or less) and the number who either died or suffered permanent brain damage as a result of the hyponatraemia. The epidemiological data were generated by computer search of the hospital records using the SAS database[9] to obtain information on all paediatric surgical patients who had a postoperative serum sodium concentration of 128 mmol/l or less. There were 24 412 consecutive inpatient operations over the three years ended 31 December 1991. In addition, we calculated an approximation of the incidence of hyponatraemic brain damage in children in the United States from our epidemiological data plus a statistical database from the medical literature.[10-13]

Results

STUDY PATIENTS

The table shows the clinical circumstances which resulted in hospitalisation of the 16 patients. All data are presented as means (SD). Symptoms were not known in three patients, who were either too young (less than 18 months) or intubated and thus unable to vocalise any complaints. Of the remaining 13 patients, 11 had progressive lethargy, weakness, nausea, and emesis and 12 had headache. All patients suffered respiratory arrest after a mean of 37 hours (range three to 120 hours) from the start of intravenous fluid administration.

CLINICAL COURSE

At admission the serum sodium concentration was 138 (2) mmol/l. As early as two hours after starting hypotonic fluid administration those patients able to communicate became progressively more lethargic and complained of headache and nausea, with subsequent emesis. All such symptoms were generally unresponsive to conventional agents (phenothiazines and narcotics). After a mean of 37 hours all 16 patients suffered respiratory arrest, at which time the serum sodium concentration was 115 (7) mmol/l and urine osmolality 676 (66) mmol/kg. This level of urine hypertonicity in the presence of hyponatraemia suggests that the plasma antidiuretic hormone concentration was raised.[8] The onset of respiratory arrest was often explosive in nature, and hyponatraemia was generally not considered as a possible cause.

Immediately after respiratory arrest but before oxygen administration or intubation the arterial oxygen tension was evaluated in 11 patients and was 6·0 (1·5) kPa. During the 37 hours between the time of admission and onset of respiratory arrest the patients had received a mean of 125 (83) ml hypotonic intravenous fluids per kg daily. Urine output was 34 (34) ml/kg per day and other fluid losses averaged 28 (25) ml/kg per day (nasogastric suction, n=2; emesis, n=10; cerebrospinal fluid drainage, n=1; not charted, n=3) with mean net output of 74 (82) ml/kg daily and net positive fluid balance of only 27 (14) ml/kg per day. Hyponatraemia in these children was thus largely due to extensive extrarenal loss of electrolyte containing fluids with replacement by hypotonic fluids. Most of the intravenous fluids were administered as 280 mmol glucose per litre either in water or in sodium chloride 38 mmol/l, but the plasma glucose concentration was

Clinical characteristics of 16 children with symptomatic hyponatraemia

Case No	Gender and age (years)	Weight (kg)	Serum sodium (mmol/l) Initial	Serum sodium (mmol/l) Lowest	Duration of intravenous fluid treatment (hours)	Net fluid intake (ml/kg)	Net fluid output (ml/kg)*	Clinical history	Hospital procedures	Respiratory arrest	Treatment after respiratory arrest	Clinical outcome
1	M 3·1	2·27	139	114	46	246	222	Fever, dysphagia, pharyngitis, tonsillitis	Antibiotics + fluids	Yes	154 mM sodium chloride	Vegetative, quadriplegia
2	F 5	18·0	141	123†	14	96	33	Tonsillitis	Tonsillectomy	Yes	None	Died
3	F 4	18·2	139	113	21	114	NA	Tonsillitis	Tonsillectomy	Yes	None	Died
4	M 15	44·6	134	101	74	164	73	Fever, dysphagia, pharyngitis, tonsillitis	Antibiotics + fluids	Yes	154 and 514 mM sodium chloride	Aspiration pneumonia, sepsis, died
5	M 3·5	15·0	138	124†	9	61	5	Tonsillitis	Tonsillectomy	Yes	None	Died
6	F 12	51·8	137	120†	33	57	11	Elbow fracture from car accident	Setting of fracture	Yes	514 mM sodium chloride; intubation	Ambulatory, mental retardation
7	M 4	16·4	139	118	22	109	88	Elbow fracture from fall	Setting of fracture	Yes	None	Died
8	M 3	10·0	137	113	1	300	NA	Stricture of urethra; tonsillitis	Urethral dilatation; tonsillectomy	Yes	None	Died
9	F 1·5	10·6	132	114	120†	283	253	Hydrocephalus	Ventriculoperitoneal shunting	Yes	None	Vegetative
10	M 9	27·0	137	120†	32	79	NA	Fractures from car accident	Operative setting of fractures	Yes	None	Vegetative
11	F 15	52·0	138	102	94	87	57	Fractures from car accident	Operative setting of fractures	Yes	154 mM sodium chloride; intubation	Vegetative and blind
12	F 4	16·8	138	107	16	88	56	Tonsillitis	Tonsillectomy	Yes	None	Died
13	M 2	11·4	138	116	3	123	NA	Undescended testicle	Orchiopexy	Yes	None	Died
14	M 6	15·0	138	119	12	40	11	Severe epistaxis	Posterior packing	Yes	None	Died
15	M 12	42·0	137	123†	19	34	9	Fever, appendicitis, ruptured appendix	Appendicectomy plus drainage	Yes	None	Died
16	F 12	28·5	134	116	66	113	72	Pneumonia	Antibiotics + fluids	Yes	None	Vegetative
Mean	7	23·8	138	115	37	125	74					
(SD)	5	12·9	2	7	34	83	82					
	4	3·2	1	2	9	21	24					

*Urine + emesis + gastric drainage + cerebrospinal fluid. NA = Not available.

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diagnosed. Four patients (case 1, 3, 6, and 9) subsequently developed the syndrome of central diabetes mellitus and central diabetes insipidus[5] with hyposmotic polyuria. In these four patients the mean serum sodium concentration rose (without treatment) from 114 (6) mmol/l to 164 mmol/l and the glucose concentration to 31·1 mmol/l. None of these patients had been treated for their hyponatraemia.

OUTCOME

All 16 patients either died or suffered permanent brain damage (table): one was mentally retarded, 10 died, and five were in a persistent vegetative state which persisted for follow up intervals of at least two years. Twelve patients received no specific treatment for their hyponatraemia. Of these, nine died and three remained in a persistent vegetative state.[11] Four patients were eventually treated with intravenous sodium chloride 154 and 514 mmol/l (table) such that the serum sodium concentration was increased from 108 (9) to 138 (4) mmol/l in 44 hours. The average delay from respiratory arrest to start of treatment was eight hours; all four patients were comatose, apnoeic, and intubated at the time treatment was begun, and none awoke either during treatment or for three days thereafter. Only one patient (case 6), who survived mentally retarded, was treated within 10 minutes of respiratory arrest.

NECROPSY FINDINGS

Postmortem examination of the brain was performed in 10 patients (three girls, seven boys). In nine patients who had received no treatment and died in less than 48 hours there was cerebral oedema and herniation on gross examination of the brain. The brain weight (unfixed) in six patients (three male, three female) whose mean age was 3·8 years was 1354 (95) g. For comparison, the normal brain weight in men is 1450 g, in women 1250 g, in 4-5 year old boys 1300 g, and in 4-5 year old girls 1150 g.[11] Thus brain weight was increased by more than 10% above control values for children of the age range studied.[11] That transtentorial herniation was present in all nine patients subjected to postmortem evaluation correlates well with the observation that the human brain can expand by only about 5-7% of its normal volume[12] before herniation occurs. We have shown that men's brains can usually adapt to hyponatraemia within a few hours whereas women's brains may not adapt within several days.[4] In all 16 children presented here the brains were unable adequately to adapt to hyponatraemia.

EPIDEMIOLOGICAL FINDINGS

Among 28 412 paediatric surgical admissions to a 456 bed university paediatric hospital there were 83 (0·34%) patients who developed hyponatraemia. Among these, seven (8·4%) died of complications of the hyponatraemia. Among the seven deaths, four were in boys and three in girls. Hence the incidence was 340 cases of paediatric postoperative hyponatraemia and 29 hyponatraemic deaths per 100 000 inpatient operations on children. There are 2·02 million paediatric inpatient operations a year in the United States.[13,14] The estimated yearly incidence in the United States is 7448 cases of paediatric postoperative hyponatraemia, with 626 such hyponatraemic deaths in children. The most common inpatient operations on children in the United States[13] are to the nose, mouth, and pharynx (17%); digestive system (17%); musculoskeletal system (15%); and nervous system (13%), of which 43% are performed in girls. This was essentially the distribution in our series, in which 92% of operations were in these four groups and 44% of the patients were female (table).

Almost every other paediatric patient with symptomatic hyponatraemia (101-123 mmol/l) can abruptly develop respiratory arrest and either die or develop permanent brain damage. The permanent brain damage can include pituitary infarction with resultant central diabetes insipidus and mellitus, a syndrome not previously described in children.[5] The incidence of postoperative hyponatraemia in children (0·34%) was less than in adults (1-4%).[1-3] However, among paediatric patients who developed symptomatic hyponatraemia the incidence of permanent brain damage was substantially higher than in adults.[1-3] Both the types of surgery and gender distribution among our 16 patients (table) were the same as the most common operations and gender distribution in the United States as a whole,[13] and thus our 16 patients were representative of the spectrum of elective paediatric surgical patients.

The hyponatraemia in these children seems to have been caused by excessive extrarenal loss of electrolyte containing fluids and intravenous replacement with hypotonic fluids (table) in the presence of antidiuretic hormone activity. Increased plasma concentrations of antidiuretic hormone are usually found in both children and adults with hyponatraemia,[6-10,15,16] and the hormone has multiple cerebral and vascular effects which can impair the ability of the brain to adapt to hyponatraemia.[17,18] However, the genesis of hyponatraemia in children is usually different from that in adults. In adults there has often been administration of very large quantities of intravenous fluid (net retention 63 ml/kg per day in adults *v* 28 ml/kg per day in children; $p<0·01$)[15] or diuretic induced loss of cations.[7,19] It is important to recognise that in children, when there is substantial extrarenal loss of electrolytes, a minimal positive balance of hypotonic fluid can lead to fatal hyponatraemia. Another major factor which may have contributed to the high morbidity among these children was the virtual absence of timely treatment in the presence of obvious symptoms.[8,11,20,21] Furthermore, the types of operations and the clinical conditions in this patient population were similar to those most common in the United States.[13] Thus the index of suspicion for electrolyte disorders in generally healthy children undergoing elective surgery may be quite low.

BRAIN ADAPTATION TO HYPONATRAEMIA IN CHILDREN

In adults oestrogens seem to impair the ability of the brain to adapt to hyponatraemia and androgens may augment such adaptation.[20,22] However, prepubescent children have only minimal to absent concentrations of either hormone, thus negating such effects. Most adults suffering permanent brain damage from hyponatraemia are female,[11,24] but in the current series a minority of affected patients (43%) in both the prospective and retrospective studies were female. Thus unlike the marked gender differential in adults, male and female children seem to be at similar risk of developing hyponatraemia encephalopathy (NS (χ^2 test)). Furthermore, neither the actual concentration of serum sodium nor the rapidity of development of hyponatraemia seemed to predict the ultimate outcome in these 16 children (table). Hyponatraemia developed over a mean of 37 hours and the range of serum sodium values was 101-123 mmol/l, values quite similar to those previously reported in children with symptomatic hyponatraemia who did not develop brain damage.[25,26]

EFFECTS OF PHYSICAL FACTORS

When hyponatraemia was present all 16 children had radiological evidence (computed tomography, magnetic resonance imaging) of cerebral oedema

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whereas at necropsy nine of 10 evaluated had cerebral oedema with herniation. These findings show that adequate adaptation of the brain to hyponatraemia had not occurred. There are several unique characteristics of the paediatric central nervous system which may impair the ability to adapt to hyponatraemia. Such characteristics may include physical factors resulting from differences in the ratio of intracranial capacity to brain size, cerebrospinal fluid volume, and brain water and electrolyte content.

The early adaptation of brain to hyponatraemia involves a loss of blood and cerebrospinal fluid followed by extrusion of sodium from brain cells.[9,10] Later adaptation includes loss of potassium and possibly amino acids, which act further to decrease brain cell osmolality and limit the gain of water.[9,10] In humans and laboratory animals brain water content is more than 2·5 times higher in the young, decreasing progressively with age.[9,10] In children the ratio of brain to skull size is such that there is less room for expansion of the paediatric brain in the skull than there is in adults.[9] As adults age there is a progressive decline in the brain volume whereas skull size remains constant.[9] Hence anatomically there is decreased room for expansion of the brain within the skull in children as compared with adults.[9]

Adult brain size is reached at about age 6 whereas full skull size is not reached until age 16. Additionally, the intracerebral volume of cerebrospinal fluid is more than 10% greater in adults than in the young.[9] When brain swelling occurs the intracerebral loss of cerebrospinal fluid increases the available volume in which the brain can expand.[9,10] As the percentage of cerebrospinal fluid in the brain increases with age[9,10] adults of both genders have more room in the rigid skull for the brain to expand than do children.[9] Furthermore, the brain intracellular concentration of sodium is about 27% higher in children than in adults[10] and may reflect a relative decreased ability to pump sodium out of the brain in children. In the presence of hyponatraemia this will result in a greater osmolar gap between brain and plasma in the young. It has been shown that in newborn puppies with hyponatraemia the brain is unable to extrude cations[10] whereas adult animals with hyponatraemia can readily transport sodium out of the brain.[9,10,26]

PREVENTION AND TREATMENT OF HYPONATRAEMIC ENCEPHALOPATHY

Symptomatic hyponatraemia can best be prevented by not infusing hypotonic fluids to hospitalised children unless there is a clear cut indication for their use. Headache, nausea, emesis, weakness, and lethargy are consistent symptoms of hyponatraemia in children. If the condition is allowed to go untreated there can follow an explosive onset of respiratory arrest, coma, and transtentorial cerebral herniation. At present there is no way to predict which children may suffer respiratory arrest. As found recently in adults neither the magnitude of hyponatraemia nor its duration is the major determinant of brain damage.[3] Recent studies show that recovery from symptomatic hyponatraemia in children, even after the onset of seizures and apnoea, may be possible if appropriate treatment is instituted in a timely manner.[11]

When a paediatric patient receiving hypotonic fluids begins to have headache, emesis, nausea, or lethargy the serum sodium concentration must be measured. Although these symptoms are somewhat non-specific, the diagnosis is easily established at minimal cost and with virtually no risk to the patient by evaluating plasma electrolyte values. When symptomatic hyponatraemia is diagnosed the patient should be moved to a location where constant monitoring can be provided, such as the intensive therapy unit. Hypertonic sodium chloride (514 mmol/l) should be infused as described,[11,14] such that the serum sodium concentration is increased to 125–130 mmol/l but by no more than 25 mmol in the initial 48 hours. In addition to hypertonic sodium chloride, treatment may include intubation and assisted mechanical ventilation when required.

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Addendum

After submission of this paper a report appeared describing 34 paediatric patients with water intoxication.[11] Two of the patients became hyponatraemic secondary to intravenous hypotonic fluid administration (serum sodium concentrations 112 and 114 mmol/l). Both suffered respiratory arrest and died, and at necropsy both had cerebral oedema. These two patients had a clinical course similar to the 16 in our series. The other 32 patients had oral water intoxication, and all survived because of timely and appropriate treatment.

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First use of heroin: changes in route of administration over time

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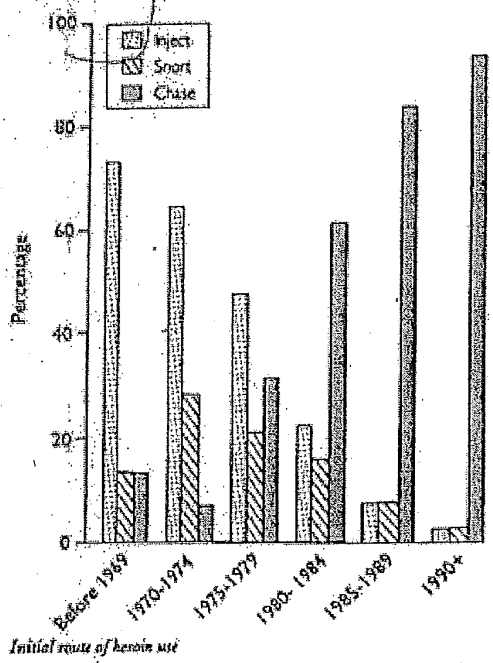
AIDS and drug misuse are linked mainly by the injection of many drugs. Major changes in the methods of heroin use, however, have fundamentally altered the importance of heroin use in the transmission of HIV. Recent reports describe the extent of "chasing the dragon" (inhaling sublimated heroin after heating it on tinfoil) as a new route of heroin use but give no information on the emergence of this pattern.[1,2] During the 1960s heroin use was by injecting.[3] What events occurred (and when) to account for this substantial change in the nature and the link with HIV of the heroin epidemic?

Subjects, methods, and results

Four hundred heroin users were contacted and interviewed by trained peer group interviewers through a structured and tape recorded interview. A total of 204 (51%) were currently out of contact with any treatment service, 100 (25%) were currently attending a drug clinic, and 124 (31%) were currently attending a needle exchange scheme. A total of 136 (34%) had never had contact with either treatment services or an exchange scheme. Their ages ranged from 17 to 53 (mean (SD) 27·6 (6·3) years); 248 (62%) were male; 96 (24%) were in current employment. There was wide variation in first year of use of heroin use (1954 to 1991): 16 (4%) started during the '60s, 28 (7%) during the early '70s, 76 (19%) during the late '70s, 124 (31%) during the early '80s, 120 (30%) during the late '80s, and 36 (9%) during the '90s.

Three different routes of initial drug use were identified: injecting, snorting, and "chasing the dragon." Analysis of these data by year revealed a major change in the annual proportion who were initiated by either injecting or chasing (figure).

"Chasing" was a route of initiation for a minority of users up to the late 1970s but has become an increasingly common route of initiation since 1975. By 1979 there were as many initiations by chasing as by injecting, and by 1981 more than half of the initiations into heroin use were by chasing (with the annual proportion remaining above half since 1981). By 1985 more than three quarters of initiations were by chasing, and since 1988, 87 out of 93 initiations (94%) were by chasing. During most years, a tenth to a quarter of users were initiated by snorting.



Initial route of heroin use

Comment

Heroin use today is not what it was yesterday. Initiation no longer occurs by injecting but by the new route of "chasing the dragon." The emergence of new non-injecting routes of heroin use may partly explain not only the major heroin epidemic in the United Kingdom during the 1980s but also its apparent continuation despite the addition of AIDS as a potential consequence. Perhaps the protective societal taboo against injecting was circumvented and a less fettered epidemic has developed. In the 1990s virtually all initiations into heroin use in our London sample were by "chasing the dragon," even though heroin use in other countries (for example, the United States) and even in other British cities (for example, Edinburgh) continues to be by injection. Should the change in London be regarded as an isolated development in a few "chasing" cities, or is it an indication of likely future changes on a wider scale? And what is the significance for tomorrow's prevention and treatment programmes?

Our level of ignorance about changing routes of drug administration is not only scientifically disturbing but also interferes with the development of prevention and treatment programmes. Effective primary prevention strategies depend greatly on the adequacy of knowledge about the gateways into drug use, and yet our understanding of the phenomenon is informed largely by

011-009-077

Dr John Alexander
[redacted]

30 November 1995

Dear Dr Alexander

RE: ADAM STRAIN, DECEASED

The above-named four year old child died following kidney transplant surgery in the Royal Belfast Hospital for Sick Children on 28th November 1995. A post-mortem examination was carried out by Dr Alison Armour of the State Pathologist's Department and she has informed me that she found gross cerebral oedema - the worst she had ever seen in an autopsy on a child.

At the present time the information I have is somewhat sketchy but it would appear that the surgery itself was uneventful with nothing to cause concern to either the surgical team or the anaesthetist. I understand that the child was healthy and considered to be an ideal candidate for transplant surgery. No complications were anticipated. The child had undergone surgery on a number of previous occasions and therefore one could assume that if any complications had been encountered on one of those occasions they would have been noted.

The anaesthetist involved was Dr Bob Taylor and the Surgeons were Mr Stephen Browne and Mr Patrick Keane (of BCH).

I should be grateful if you would let me have an anaesthetic report for use at the Inquest and in accordance with my normal practice I would wish you to attend to give evidence. All the medical notes will be made available to you, for these you should contact Dr George Murnaghan, Senior Administrator, King Edward Block, Royal Victoria Hospital, Grosvenor Road, Belfast, BT12 6BA - telephone [redacted] As yet I do not have any statements from the

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clinicians but when these come to hand I will let you have copies. If I can be of any further assistance please let me know.

I am most grateful that you agreed to undertake another difficult case on my behalf.

Yours sincerely

J L LECKEY
HM CORONER FOR GREATER BELFAST

011-021-130

8/5/96

Dear Dr Murnaghan.

Thank you for forwarding the postmortem findings regarding Adam Strain. As you know I do not wish to cause any conflict or disagreement which would cause further distress or suffering to persons involved in this case. However there are several fundamental problems with the report which I must address.

I agree that death was due to cerebral oedema and that hyponatraemia was present but disagree with the causes.

Hyponatraemia
Towards the end of Commentary it states "the hyponatraemia in this case was the cause of cerebral oedema and most of the ...fluids given .....were.....sodium chloride 38 mmol/l." The facts are that 40% of the fluids contained this amount of sodium (1500 ml) 0.18 NaCl/4% Glucose compared to the remaining 60% of total fluids given which contained 130-150 mmol/l of sodium (HPPF, Blood, Hartmanns).
The PM statement therefore clearly misrepresents the facts in a prejudicial manner.

Impaired cerebral perfusion
There is no evidence that "Impaired cerebral perfusion" occurred in this case. Cerebral Perfusion is defined as Mean Arterial pressure (MAP) minus Intracranial pressure (ICP). Intracranial pressure was not monitored in this case, and is never monitored except in head injuries etc. as it involves an invasive monitor in the brain. Since MAP was maintained throughout the procedure it is unlikely that there was cerebral hypoperfusion. Perhaps a better logical explanation would be "Impaired Cerebral Drainage". However this is against known research especially in this case where a recent article suggests that complete jugular ligation does not cause an increase in ICP.

This is contradicted by the description of the postmortem findings. In the PM under **Examination of the Neck** it states "There was no evidence of congestion or obstruction of the major blood vessels...". This contradicts the conclusion that cerebral perfusion (or cerebral drainage) could have been impaired.

There is no premorbid nor postmortem evidence that excessive volumes of fluid were administered which produced a dilutional hyponatraemia. I still do not know what caused his death but I believe it is unacceptable to speculate on the cause of Adam's death without direct postmortem evidence and by misrepresenting the quantities and type of fluids given.

I would hope that reasons are not being generated or misrepresented to suit the diagnosis.

Yours sincerely,
Dr Robert Taylor

10.06.96

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