[redacted]

HER MAJESTY'S CORONER

DISTRICT OF GREATER BELFAST

Telephone: [redacted]
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John L Leckey LL.M.
H. M. Coroner
Coroner's Office
Courthouse
Crumlin Road
Belfast
N. Ireland
BT14 6AL

9.1.96
I have told
Dr. RH Taylor
of Dr Alexander's
conclusions.
Agreed that I
will send him
all 3 reports when
other 2 (Prof Berry
& Dr L. Simms) arrive
GAM

Dr G A Murnaghan
Senior Administrator
King Edward Block
Royal Victoria Hospital
Grosvenor Road
BELFAST
BT12 6BA

5 January 1996

Dear [redacted]

RE: ADAM STRAIN, DECEASED

I am enclosing a copy of Dr Alexander's report. I faxed a copy to Dr Maurice Savage in RBHSC.

On Thursday last I had a meeting with Miss Strain and her brother-in-law. I agreed to send her a copy of each report as it arrives with me and I have sent a copy of Dr Alexander's report to her.

I explained that these reports are likely to contain complex medical language and she would require an explanation of the contents. I stressed that I did not have sufficient expertise to do that. She wondered whether Dr Savage might be willing to and I informed her that I would channel that suggestion through your goodself.

Subsequently I spoke to Dr Savage and advised him of this request. I explained that you were on sick leave at the present time and that he could discuss it with you on your return.

Best wishes for 1996.

Yours sincerely

J L LECKEY
HM CORONER FOR GRATER BELFAST

9.1.96
Discussed c Dr. M. Savage
He has agreed to discuss reports c
Ms. Strain - recommend that he
... when all received

AS – ROYAL

059-057-134

[redacted]

This report has been prepared by me, Dr John Alexander, on the instructions of Mr John L Leckey LL.M., HM Coroner for the District of Greater Belfast. I have studied the relevant case notes and anaesthetic record.

Re: ADAM STRAIN, DECEASED

This little boy suffered from congenital vesico-ureteric reflux and dysplastic kidneys and had had multiple surgical operations in the past, many under general anaesthesia, and all apparently uneventful as far as the anaesthesia was concerned. At the time of his last report on the 24th November 1995 he was in renal failure with a high volume of dilute urine from his own (native) kidneys. His renal failure was being treated by Continuous Ambulatory Peritoneal Dialysis (CAPD) and feeding difficulties had been overcome by fashioning a feeding gastrostomy.

He was 4 years 3 months of age, weighed 21 kg, and was well nourished. Relevant blood tests that evening were haemoglobin 10.5 g/dl, packed cell volume 0.32, sodium 139 millimoles per litre (mmol/l), potassium 3.6 mmol/l, albumin 40 mmol/l, urea 16.8 mmol/l and creatinine 702 μmol/l. The latter two results are very high, an expression of his renal failure, the remainder within normal limits. He was given 952 ml 'clear fluid', presumably water, overnight, into his gastrostomy, and this was stopped at 0500 on the 27th. The child was taken to the operating theatre at 0700 for a renal transplant.

Dialyte - Dextrose & Saline

Anaesthesia was induced at 0700 in the standard manner and the child intubated and artificially ventilated. Venous access was secured, a triple-lumen central venous pressure catheter inserted into the right subclavian vein and a fine catheter into the right radial artery to continuously monitor arterial blood pressure. The child's estimated blood volume was 1600 ml, estimated fluid deficit 300 ml and calculated intraoperative maintenance 200 ml/hour. Infact a great deal more fluid was infused, which included 1500 ml of one fifth isotonic saline in 4% dextrose, 500 ml Hartmanns solution, and eventually 800 ml of Human Plasma Protein Fraction and 2 units of packed red cells to replace a blood loss during the operation of about 1200 ml.

The operation proved to be technically difficult and took 4 hours to complete. During that time the heart rate decreased from 140 to 90 beats per minute, the systolic blood pressure increased from 90 to 120 mmHg and arterial blood saturation with oxygen remained consistently at 99 - 100 %. There were no dramatic changes and no evidence of either hypoxia or hypotension, as documented by Dr Taylor's meticulous records, and confirmed by the computerised print-out obtained at the end of the operation. Central venous pressure remained very high throughout the procedure; this may have been partly due to a technical problem with the pressure transducer but was also partly deliberate, since releasing the clamps on a transplanted near-adult sized kidney in a child can divert most of the cardiac output into the new organ with a dramatic fall in blood pressure; a high venous pressure will encourage a high cardiac output and avoid this problem.

A 21 kg child has an extracellular fluid volume of about 5 litres. This is made up of the blood volume inside the intravascular space (red cells and plasma) and the interstitial fluid which lies outside the vascular space and also outside the cells. Infused fluids will distribute themselves through the intravascular and interstitial spaces. A simple calculation reveals that if 1500 ml 1/5 isotonic or 'normal' saline is infused into a child of this size, plasma (or serum) sodium will fall to about 120 mmol/l. Since it takes some time for infused fluids to leave the vascular compartment,

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serum (or plasma) sodium is likely to be even lower than this and the situation may be made worse by increased levels of antiduretic hormone produced during anaesthesia which will cause water retention by the kidneys. There is very little firm information available concerning dilutional hyponatraemia (low serum sodium) in children, either in standard textbooks or in the recent literature, although the condition is well recognised in neonates and in adults who have certain operations which result in an excess of water entering the circulation. Arieff and colleagues published a paper entitled "Hyopnatraemia and death or permanent brain damage in healthy children" (BMJ 1992; 304: 1218-22) which is informative. These workers described how, after hypotonic fluid administration, serum sodium can fall to levels around 115 mmol/l and lead to vague non-specific symptoms and then an explosive onset of respiratory arrest, cerebral oedema and coma. They also discuss the reasons why a child's brain has less room than an adult's to expand inside a rigid skull and suggest that developing brain cells are less able to protect themselves. One might speculate as to whether a child suffering from chronic renal failure could have increased vulnerability. In the discussion Arieff et al states: "These cases show that generally healthy children with symptomatic hyponatraemia (101-123 mmol/l) can abruptly develop respiratory arrest and either die or develop permanent brain damage". Of the 16 cases they described, 10 died and the others suffered permanent brain damage. A copy of this paper is attached.

At the end of the procedure, Adam was apnoeic and had widely dilated pupils. He was transferred to the intensive care unit. Serum sodium was 119 mmol/l and did not rise above 125 mmol/l in the next 20 hours. CT scan of the brain showed cerebral oedema and lung oedema was also evident. Tests for brain stem function were negative and active therapy was discontinued on the morning of the 28th November.

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SUMMARY The complex metabolic and fluid requirements of this child having major surgery led to the administration of a large volume of hypotonic (0.18%) saline which produced a dilutional hyponatraemia and subsequent cerebral oedema. The operation was difficult and prolonged and the problem could not be recognised until the surgery was completed. At no time during the procedure was there any suggestion of hypoxia nor is there the slightest indication of a malfunction in the anaesthesia apparatus. Dr Taylor is to be commended on the detailed notes and records he kept throughout the anaesthetic.