MEDICAL REPORT

ON

ADAM STRAIN (DECEASED)

Prepared for: John L Leckey LL.M.
H M Coroner
Coroner's Office
Courthouse
Crumlin Road
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Thank you for asking my opinion on this case. I have been a consultant paediatric anaesthetist at Great Ormond Street since 1973, with a particular interest in paediatric intensive care. I am the author of several textbooks on the subject and am the Editor-in-Chief of the journal, Paediatric Anaesthesia. For the preparation of this report I have carefully perused the recent medical and nursing notes, but realize, because of Adam's previous medical history there are several older bundles of notes.

Adam was born on 4.8.1991 with vesico-ureteric reflux causing repeated, damaging urinary tract infections. He had five operations for reflux ending up with one ureter connected to the other with only one draining into the bladder. He also had a fundoplication for gastro oesophageal reflux and marked vomiting. Nutrition was a problem and it became necessary to give him gastrostomy feeds. Eventually he refused all feeds and it is my understanding that he took nothing by mouth at all.

He gradually went into renal failure to the point that dialysis was commenced using the peritoneal route. Dialysis took place at night, but Adam also passed urine, presumably of a poor quality, and has been described as polyuric. However, he was generally progressing quite well having gastrostomy feeds of 3 x 200 ml Nutrizon during the day and 1500 ml at night, i.e. a total volume of 2100 ml per day. He was on the 50th centile for height but on the 95th for weight. In July 1995 he was admitted for a pyrexial illness which was extensively investigated and was

probably an infected gastrostomy site. On 14th July he was given a blood transfusion. At the time leading up to his renal transplant in November 1995, he was taking Keflex, Fersanel, vitamin D, bicarbonate and erythropoietin in addition to his feeds and dialysis regime.

He was not hypertensive as his blood pressure on 18.10.95 was 106/61 when he had his orchidopexy and on 26th November, when admitted for the transplant the following day, the BP was 108/56.

The renal transplant took place on 27.11.1995 beginning at 07.00, the anaesthetist being Dr Taylor and the surgeons Mr Keane and Mr Brown. Adam weighed approximately 20 kg, had a haemoglobin of 10.5 g/dl with reasonable electrolytes (urea 16.8, but sodium 139) at 11 pm on 26/11. Overnight he was given 900 ml Diorolyte (4% dextrose, .18% saline) via the gastrostomy, instead of his feed, but nothing for the two hours leading up to anaesthesia. P.D. was as usual: I can find no note of how much urine per hour he was passing nor of any electrolyte results just prior to anaesthesia.

The anaesthetic technique was appropriate for a renal transplant and involved mechanical ventilation, paralysis with atracurium and epidural, though the space is not noted. Dr Taylor estimated the blood volume as 1600 ml (80 ml/kg), an estimated fluid deficit of 300 ml and calculated an intraoperative maintenance of 200 ml/hr.

Central venous access was not easy to achieve. There were three attempts at the left subclavian, one in the left internal jugular, but successful access was achieved in the right subclavian vein using a triple-lumen catheter. There were also cannulas in a vein on the left hand and in the right radial artery. Apart from anaesthesia drugs, also administered intravenously were the antibiotic Augmentin, 500 mg, methyl prednisolone 200 mg, Asathioprin 25 mg (antirejection) and a low, renal vasodilating dose of dopamine by continuous infusion of 5 mcg/kg/min, though there is no record of this on the anaesthetic form.

There was considerable blood loss - in excess of 1100 ml as the operation was slightly more difficult than usual because of all the previous surgery. The systolic blood pressure started at 85 - 90 mm Hg and gradually rose, according to the charting, to 120, whereas the pulse rate started high (145/min) presumably because of the IV atropine and gradually fell, dipping to 80/min around 09.30. There are no entries in the space available on the anaesthesia record for central venous pressure measurements. Body temperature was well maintained.

Administered fluids were, dextrose-saline (4% and .18%) 1000 ml from 07.00 - 08.30 and a further 500 ml thereafter, 500 ml Hartman's solution, 1000 ml albumin and 500 ml of packed cells. A blood gas result taken at 09.32 showed mild hypoventilation with $PaCO_2$ 44 mm Hg (normal 40), very low sodium of 123 mmol/l (normal 135 - 145) and a very low haematocrit of 18% (normal 35 -

40%). I could find no note of an earlier result. There is no note of urine output during the case - there is note of a suprapubic catheter, but I do not know whether this was in use in the theatre.

At the end of the procedure, around 11.00 am, Adam was given neostigmine and glycopyrrolate to reverse the neuromuscular blockade, but he did not breathe and was found to have fixed dilated pupils and bilateral papilloedema with haemorrhages. He had obviously suffered a major cerebral insult. On the ICU he was hypertensive, requiring nifediprine to control this. He was described as 'puffy' and he had some pulmonary oedema. He was appropriately treated with mannitol and hyperventilation in an attempt to shrink the brain, but a CT scan showed severe cerebral oedema with obliteration of the ventricles and the neurologists confirmed that his signs were compatible with brain stem death, i.e. he had coned. Electrolyte results from 27/11 (not timed) showed a sodium of 119 mmol/l. A chest x-ray showed that the triple-lumen central venous line was going up into the neck vessel. Adam died the following day.

The findings at autopsy included gross cerebral oedema but no substantial pulmonary oedema or oedema of any other organ. It was noted that the left internal jugular vein was tied off where it becomes the innominate vein.

I would like to make the following comments:

1. I do not think that the epidural had any part to play. Dr Taylor does not say which level was used nor how much 0.25% marcain he gave, but there is nothing to suggest an untoward incident with this technique.

2. Adam was normotensive throughout his life and certainly did not require drugs to control his blood pressure until after the transplant. In that case a systolic BP of 85 - 90 during anaesthesia is well within the normal range for a child having had an epidural and should not require a fluid load to raise the blood pressure at that stage, particularly as it would be some time before the new kidney was inserted.

3. Nowhere could I find a note of how much urine Adam was passing even though he was described as 'polyuric'. However, he was in a stable state for several weeks, growing and gaining weight. He was given 2100 ml per day of feed, i.e. approx 100 ml/kg/day - 4 ml/kg/hour - in addition to this there would be some water of oxidation of the nutrients in the diet. In a stable state intake equals output and his output in urine, sweat, respiration must equal 2100 ml, in addition to this there would be some volume taken off by the PD. As he was passing urine, the PD would be mainly for electrolyte exchange -K+, urea, etc., but could be in the order of 1-200 ml per day in total. I do not think his urine output could therefore be more than 1500 ml per day, i.e. 75 ml/kg/day - 3.5

ml/kg/hour on average.

Preoperatively, instead of his feed he was given 900 ml Dioralyte (hypotonic dextrose-saline solution) until two hours before anaesthesia. If we take his average intake as 4 ml/kg.hour, then two hours without fluids would give a deficit of 160 ml. Intraoperative maintenance fluids for abdominal surgery are usually calculated at 10 ml/kg for the first hour, then 6 - 8 ml/kg for subsequent hours. The initial bolus contains extra fluids to make up any deficits from preop starvation and then fluid is given for maintenance (4 ml/kg/hour) plus some extra to replenish evaporation from cut surfaces and fluid shifts into the physiological third-space. It is also necessary to give some dextrose to prevent hypoglycaemia but increasingly dextrose solutions are not used as hyperglycaemia is readily produced. It is probably better to give isotonic solutions such as Hartman's or lacted-Ringer^s solution.

In cases of renal transplant it is usual to be generous with fluids to maintain a CVP of 10 - 12 to optimize perfusion of the new kidney and to establish its urine-producing function. I think Dr Taylor overestimated the deficit somewhat, but was reasonable in suggesting 150 ml/hour for maintenance, but in fact he gave 500 ml D/S in just 30 minutes (07.00 - 07.30) and a further 500 ml over the next hour of a hypotonic solution - on top of the 900 ml that Adam had been given overnight. A further 500 ml

over 2½ hours is also greater than his calculations. Up to 09.30 he was given 800 ml plasma and 500 ml Hartman's solution for replacement of blood loss. I am assuming that the bleeding was steady, with the odd bigger loss and if Hartman's is used for blood volume replacement, twice the volume as loss is required, Adam was thus given volume replacement by 09.30 of 1050 ml for a total blood loss over four hours of 1100+ ml. It should be noted that plasma is also low in sodium.

4. I think it was unwise not to have electrolyte values taken before going to theatre and after the PD had been completed. It might be that the serum sodium was already low at that stage. It is also strange that the first blood gas was not taken until 09.32 when Adam was already severely hyponatraemic and diluted (haematocrit 18) from a combination of excess crystalloid and blood loss. Arterial access had been gained early in the case and it seems logical to analyze the blood for gases and electrolytes as soon as the patient is put on the table. There is no note of urine output during the case.

5. It is not surprising that it proved impossible to cannulate the left internal jugular vein and left subclavian since the internal jugular had been tied off. There must have been scars on the skin from a previous surgical approach to the vein. I do not believe it is a sign of dehydration if there is difficulty in cannulating a central vein, unless

other signs of dehydration, such as cold peripheries are present. Cannulation of the right subclavian was achieved, but on subsequent chest x-ray the tip was found to be lying in a neck vein, rather than in the right atrium of the heart. Unfortunately, this is not an uncommon occurrence especially when the venous anatomy is deranged from multiple previous usage. My own philosophy is that while it is possible to freely aspirate blood, it can be used on a temporary basis, but should be changed at the earliest opportunity. It is not routine practice to x-ray for these lines when they are put in in the anaesthetic room prior to surgery. It is possible that the venous drainage from the head was not completely normal. Dr Taylor did not chart any CVP measurements and all the information on this I have from his letter. There were obvious problems with CVP readings. It is advisable to attach the pressure transducers to the side of the operating table so that when this is raised and lowered as it so often is during surgery, the zero is not changed. If the transducer is correctly put at zero, there is free flow of blood in and out of the central line, cardiac and respiratory patterns to the waveform then, in my opinion, the reading is correct. I do not agree with Dr Taylor that 'from the pressure reading I concluded that the tip of the line was not in close relation to the heart.' I believe that the pressure of 17 mm was the actual reading at the tip of the catheter. This is a high reading and the rise to 20 - 21 mm Hg is very high and actually difficult to achieve in a

child because the venous system (including the liver) is incredibly distensible. With hindsight, knowing that the tip of the catheter was up in the neck, these high figures for venous pressure imply there was some degree of obstruction to venous drainage from the head and with the knowledge that the left internal jugular vein had been tied off. This was possibly caused by having the head turned to one side as is usual in theatre, as the CVP came down to 10 - 12 in the ICU with the head in the neutral position. If gross obstruction to the venous flow had been present the head would have been suffused and swollen as suggested by Dr Taylor in his letter. However, Adam was described as 'puffy' by the ICU staff.

6. It is very interesting to have the monitoring data printed out from the machine. I assume that for the systemic blood pressure with a range of 200 mm Hg, the half-way line is 100 mm Hg. The trace shows much more clearly than Dr Taylor's anaesthetic record the consistent rise in BP from around 09.30, i.e. soon after the blood gas was drawn, peaking at 150 mm Hg. The pulse rate also rose steadily from 10.15 onwards. Again, with hindsight these could represent the cardiovascular changes of a coning patient under anaesthesia. The arterial trace shows that the line was not interrupted for sampling until just after 09.30.

7. Blood transfusion is usually given to patients who are losing in excess of 15 - 20% of the blood volume (i.e. 250

- 300 ml in Adam's case). Until that point is reached volume is replaced using plasma and/or Hartman's. I think they were rather late in starting the blood transfusion as the haematocrit at 09.30 had fallen to 18% (normal 40). Overall, however, the haemoglobin was well managed as the result at the end of the case was 10 g/dl.

8. Dr Taylor suggests that cerebral oedema is difficult to explain because both thiopentone and methyl prednisone had been given albeit for other reasons. While methyl prednisolone is often given as a cerebral protector, for example for patients going on cardiopulmonary bypass, there is no hard data to support its efficacy. It is 10 years at least since thiopentone was used as a cerebral protector and in much higher doses than those used for induction of anaesthesia. Success with animal work was not borne out in the human clinical situation. Modern evidence suggests that barbiturates may even be detrimental.

To summarize, I believe that on the balance of probabilities Adam's gross cerebral oedema was caused by the acute onset of hyponatraemia (see reference) from the excess administration of fluids containing only very small amounts of sodium (dextrose-saline and plasma). This state was exacerbated by the blood loss and possibly by the overnight dialysis.

A further exacerbating cause may have been the obstruction to the venous drainage of the head. If drugs such as antibiotics were

administered through a venous line in a partially obstructed neck vein then it is possible that they could cause some cerebral damage as well.

Ref: Arieff AI, Ayus JC, Fraser CL. Hyponatraemia and death or permanent brain damage in healthy children. BMJ 1992, 304: 1218-1222.

Edward Sumner

22 January 1996

GLOSSARY CASE F 46728 (1)

CEREBRAL OEDEMA: SWELLING OF THE BRAIN

HYPONATRAEMIA: LOW SODIUM (NORMAL RANGE 135-145mmol/l)

RENAL FAILURE: FAILURE OF THE KIDNEYS

OBSTRUCTIVE UROPATHY: OBSTRUCTION TO THE OUTFLOW FROM KIDNEYS

CONGENITAL: BORN WITH

POLYURIA: PRODUCTION OF LARGE VOLUMES OF DILUTE URINE

URETERS: CONNECTING KIDNEYS TO BLADDER

FUNDOPLICATION: OPERATION TO STOMACH

GASTRO-OESPHAGEAL REFLUX: STOMACH CONTENTS REGURGITATING INTO THE GULLET (oesophagus)

ORCHIDOPLEXY: OPERATION TO FREE UNDESCENDED TESTICLE

PERITONEAL DIALYSIS: THE REMOVAL OF WASTE AND TOXIC PRODUCTS FROM BLOOD BY THE USE OF THE PERITONEUM AS A SEMIPERMEABLE MEMBRANE

GASTROSTOMY: ARTIFICIAL HOLE IN STOMACH CREATED BY SURGERY

CVP: CENTRAL VENOUS PRESSURE

CT SCAN: COMPUTERISED AXIAL TOMOGRAPHY

PULMONARY OEDEMA: FLUID ON THE LUNGS

EPIDURAL: INJECTION OF ANALGESIC INTO THE SPACE AROUND THE SPINAL CORD

LUMBAR: LOWER BACK

ELECTROLYTES: IN THE MAIN SODIUM AND POTASSIUM

A.D.H.: ANTI-DIURETIC HORMONE

ALDOSTERONE:	HORMONE CONTROLLING SODIUM BALANCE
HYPOXIA:	DIMINISHED AMOUNT OF OXYGEN IN TISSUES
HAEMORRHAGE:	BLEEDING
ISOTONIC:	SAME CONCENTRATION AS PLASMA i.e.. normal saline is isotonic with plasma
HYPOTONIC:	A SOLUTION HAVING A LOWER OSMOTIC PRESSURE THAN ANOTHER ONE
CEREBRAL PERFUSION:	THE PASSAGE OF BLOOD THROUGH THE BRAIN

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