Medicolegal Report

On

Adam Strain (deceased)

Prepared for: Police Service of Northern Ireland
Fermanagh District Command Unit
48 Queen Street
Enniskillen BT74 7JR

By: Edward Sumner MA BM BCh FRCA
[redacted]

September 2005

Thanks you for asking my opinion on this case. I am a retired consultant paediatric anaesthetist from Great Ormond Street where I worked from 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, Pediatric Anesthesia.

For the preparation of this report I have carefully perused the recent medical and nursing notes, but realise, because of Adam's previous medical history there are several older bundles of notes. I have also read the statements and depositions of those involved presented to me by the Police Service of Northern Ireland. The report of the autopsy by DR Alison Armour (pages78-85) is excellent and informative.

The verdict at the Inquest states the cause of death to be cerebral oedema due to Dilutional Hyponatraemia and impaired cerebral perfusion during renal transplant operation for chronic renal failure (congenital obstructive uropathy)

I must stress that the comments I make and the answers to questions posed are only my opinion.

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.
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 200ml Nutrizon during the day and 1500 ml at night, ie. A total volume of 2100ml 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 18.10.1995 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 0700, the anaesthetists were consultant Dr Taylor and trainee Dr Montague and the surgeons Mr Keane and Mr Brown. Adam weighed approximately 20kg, had a haemoglobin of 10.5 g/dl with reasonable electrolytes (urea 16.8, but sodium 139) at 11pm on 26.11. Overnight he was given 900ml Diorolyte (4% dextrose, 0.18% saline) via the gastrostomy, instead of his feed, but nothing for the two hours leading up to anaesthesia. Peritoneal dialysis

(PD) was as usual. I can find no note of how much urine per hour he was passing nor of any electrolytes results just prior to anaesthesia. They had tried to obtain blood before the operation in the morning to measure electrolytes but had been unable to do so.
The dialysis sheet is page 810 and I note that the fluid balance for the peritoneal dialysis has not been filled in. This is a pity because it gives some idea of how fluid depleted, or otherwise Adam was likely to have been after the dialysis.

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

Central venous access was not easy to achieve. There were three attempts at the left Subclavian vein, 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 500mg, methylprednisolone 200mg, Asathioprin 25mg (anti-rejection) and a low, renal vasodilating dose of dopamine by continuous infusion of 5mcg/kg/min, though there is no record of this on the anaesthetic form.

There was considerable blood loss – in excess of 1100ml (two thirds of the estimated blood volume)as the operation was more difficult than usual because of all the previous surgery. The systolic blood pressure started at 85 –90 mmHg 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 0930 but later rose again from1000. 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 0.18%) 1000ml from 0700-0830 and a further 500ml thereafter, 500ml Hartman's solution, 1000 .ml albumin and 500ml of packed red blood cells. A blood gas result taken at 0932 showed mild hypoventilation with PaCO2 44 mmHg (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.

The arterial supply for the kidney came from the iliac artery which is routine for this procedure. The vascular clamps were removed at 1030 and initially the kidney was well perfused with blood and started to produce urine, but by the end of the procedure was not so well perfused which is why dopamine was commenced. In the intensive

care unit postoperatively the kidney was described as "bluish" when the wound was closed

At the end of the procedure, around 1100, 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 nifedipine 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 ie. he had coned.

Electrolyte results from 27.11 not timed, but in the early postoperative period showed a sodium of 119 mmol/l. A chest Xray 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. The transplanted kidney was infarcted

I would like to make the following comments:

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

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 when a "full" blood pressure is beneficial to perfuse the new kidney.

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 (approx 100ml/kg/day - 4ml/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 approximately 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 – ie. 75ml/kg/day - 3.5 ml/kg/hour on average.

Preoperatively, instead of his feed he was given 900ml Dioralyte (dextrose-saline solution) until two hours before anaesthesia. If we take his average intake as 4ml/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 preoperative starvation and then fluid is given for normal maintenance (4ml/kg/hour) plus some extra to replenish evaporation from exposed surfaces and fluid shifts into the physiological third space. It was also thought necessary to give some dextrose to prevent hypoglycaemia, but increasingly dextrose solutions are not used as hyperglycaemia is readily produced. It is better to give isotonic solutions such as Hartman's or lactated-Ringer's solution.

In cases of renal transplant it is usual to be generous with fluids to maintain a CVP of 10-12 to optimise perfusion of the new kidney and to establish its urine-producing function. I think Dr Taylor over-estimated the deficit somewhat, but was reasonable in suggesting 150ml/hour for maintenance, but in fact he gave 500 ml D/S in just 30 minutes (0700-0730) and a further 500 ml over the next hour of this solution – on top of the 900 ml that Adam had been given overnight. A further 500ml over 2 ½ hours is also greater than his calculations.

Up to 0930 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 replacement, twice the volume as loss is required. Adam was thus given volume replacement by 0930 of 1050 ml for a total blood loss over four hours of 1100+ ml. Thus the bulk of the fluid administered had been done so one hour before the vascular clamps were removed

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. The delay in measurement of the blood gases and electrolytes until 0930 is hard to understand and I have never seen a satisfactory explanation for this.

Although a laboratory result would probably take one hour (possibly more at that time of day) instant information on sodium and potassium values is available from blood gas results. Although blood gas analysis is primarily intended for the measurement of the blood (usually arterial) acidity (pH) oxygen and carbon dioxide levels, it does also give readings for sodium and potassium with a fair degree of accuracy.

The machine for measuring this is usually in theatre or at least nearby in the intensive care unit.
I do not agree with Dr Alexander (Page 28) when he says "with the benefit of hindsight..." They had tried to obtain blood before the operation so it was in their mind to measure the electrolytes, which is the correct thing to do.

In my opinion this was an error of judgement.

By 0930 when the first blood gas was taken 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 analyse the arterial blood for gases and electrolytes as soon as the patient is put on the operating table. Blood loss not replaced with blood could also have contributed to the low haemoglobin.

There is no note of urine output during the case.

It is not surprising in retrospect, 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 Xray 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 convenient opportunity. It is not routine practice to Xray 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 CVP measurements and the information comes from the monitor print-out.

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 to 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. Dr Taylor said that "from the pressure reading I concluded that the tip of the line was not in close relation to the heart" I am not sure how he could say that. I believe that the pressure of 17 mmHg was likely to have been the actual reading at the tip of the catheter. This is a high reading and the rise to 20-21 mmHg is very high and actually quite difficult to achieve in a child because the venous system (including the liver) is very distensible.
With hindsight, knowing that the tip of the catheter was up in the neck and with the knowledge that the left internal jugular vein had been tied off, these high figures could imply there was some degree of obstruction to venous drainage from the head.

This was possibly caused by having the head turned to on side as is usual in theatre, as the CVP came down from 18, the last reading in theatre to 10-12 in the ICU with the head in the neutral position.
Again with the advantage of hindsight, the initial high reading of CVP was probably correct and could have been the result of the initial fluid bolus

If gross obstruction to the venous flow had been present the head alone would have been suffused and swollen as suggested by Dr Taylor in his letter. Adam was described as "puffy" by the ICU staff, but it is not clear whether this refers just to the head. Adam's mother described him as bloated which implies to me, whole body swelling.

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 200mmHg, the half-way line is 100 mmHg. The trace shows much more clearly than Dr Taylor's anaesthesia record the consistent rise in BP from around 0930, soon after the blood gas was drawn, peaking at 150 mmHg. The pulse rate also rose steadily from 1015 onwards. Again, with hindsight these changes 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 0930.

Blood transfusion is usually given to patients who are losing in excess of 15-20% of the blood volume (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 0930 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, the level at which it had started.

Dr Taylor suggests that the cerebral oedema is difficult to explain because both thiopentone and methylpredisolone had been given, albeit for other reasons. While methylprednisolone is often given as a cerebral protector, for example for patients going on cardiopulmonary bypass, there are 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 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.

There is no evidence that the surgical aspects of the renal transplant were not routine. The vascular anastomoses were to the iliac vessels which is normal for this procedure and although the perfusion of the kidney when they closed was not normal, this is often the case and it can be expected to improve with time. It is unthinkable that the wound would have been closed if the kidney had infarcted at that stage.

To summarise, Adam's gross cerebral oedema was caused by the acute onset of hyponatraemia form the excess administration of fluids containing only very small amounts of sodium. This state could have been exacerbated by the blood loss and possibly the overnight dialysis.

A further exacerbating cause could have been an obstruction to the venous drainage of the head but there is no way of knowing whether this was a contributing factor to the brain swelling.

My impression is that Dr Taylor acted in good faith but I believe he made some errors of judgement.

The fluid management in this situation is complex as the various requirements for replacement of deficits, ongoing hidden and obvious losses (evaporation and bleeding) and the amount being lost via urine all require minute-to-minute judgements based on clinical and biochemical findings.

As Adam died from dilutional hyponatraemia and its acute cerebral effects, then, in my opinion it must have been the volumes of intravenous dextrose-saline which contributed to this.

Dr Taylor had done initial calculations which he justifies, but in my opinion they were over-estimations but the administration of 500 ml dextrose-saline over a 30 minute period and a further 500ml over the next 75 minutes was too much of this solution, over too short a time and is far in excess of his calculations. Dextrose-saline strictly speaking is "isotonic" in the bag but basically becomes water in the body as the sugar is rapidly metabolised

I now understand that there was and indeed probably still is widespread ignorance concerning hyponatraemia, in spite of papers on the subject in prominent medical journals from the 1980s. The ignorance was that a dextrose-saline (0.18% saline) solution while being a reasonable choice for use strictly as a maintenance fluid, cannot be used to replace abnormal losses such as bleeding or diarrhoea and vomiting in which the losses are primarily sodium chloride.