CORONERS ACT (Northern Ireland), 1959

Deposition of Witness taken on FRIDAY the 21 day of JUNE 19 96, 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 R H TAYLOR

of c/o R.B.H.S.C.

(Address)

who being sworn upon h oath, saith

On the 27th November 1995 at 06.45am, I was the Consultant Paediatric Anaesthetist on duty for the Royal Belfast Hospital for Sick Children. I commenced a general anaesthetic for a kidney transplant on a 4 year old boy known to me as Adam Strain. He was in polyuric renal failure as the result of congenital posterior urethral valves and had been receiving continuous peritoneal dialysis. He had been admitted to RBHSC on Sunday 26th Nov 1995 in preparation for the transplant. I was made aware of the preoperative problems of fluid administration, that he usually received night feeds and that iv fluids could not be given 2 hours prior to surgery so I had permitted clear gastric fluids to be given up to the last possible moment. I encountered no difficulties following his arrival in theatre accompanied by his mother. He weighed 20 kgs. General anaesthesia was induced uneventfully using thiopentone 125 mg, atropine 0.3 mg and atracurium 10 mg given by a 25G butterfly needle in his right antecubital fossa with his mother cuddling him. I.v. access, arterial access and a central venous catheter were all placed without undue difficulty and a lumbar epidural was sited under sterile technique to provide pain relief during and after the procedure. I administered iv fluids as is usual and calculated to correct his fluid deficit, supply his maintenance and replace operative losses. Crystalloid fluids (500 ml bags of 0.18 NaCl in 4% glucose x 3 and Hartmanns 500 mls over 4 hours) were continued to provide maintenance and supply sufficient fluid for the native polyuric kidneys. As there was a substantial ongoing blood loss from the surgery colloid fluids (HPPF) and eventually packed

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The Deposition of R H TAYLOR

of

who being sworn upon h oath, saith *(Address)*

by the neuro-radiologist that he had gross cerebral oedema and herniation of his brain. I remain extremely perplexed and concerned that this happened to Adam and cannot offer a physiological explanation for such severe pulmonary and cerebral oedema in the presence of normal monitoring signs. I wish to make the following observations:- 1. Polyuric renal failure. This required great attention to the details of calculating Adam's fluid requirements. It was usual to give this child 1,500 mls of food/fluid overnight to maintain his growth milestones and to compensate for polyuria from his native kidneys. This was given via his gastrostomy buttom at night as he slept. The delivery of such large quantities of food would have profound effects on his metabolisum (eg. sugar, insulin), normally we fast at night. It was, therefore, necessary to interfere as little as possible with his 'normal' fluids. I had discussed his preoperative fluids with Dr. Savage (Consultant Paediatric Nephrologist) and Mr Brown (Consultant Paediatric Surgeon) and had decided that 'usual' quantities of oral (or gastrotomy) fluids (Diaoralyte= 0.18 NaCl/4% Glucose solution) should be administered up to the last possible moment (2 hours before surgery) to minimise the likelihood of dehydration and hypoglycaemia. A great amount of consideration was given to maintaining this 'normality' during the operation. He had multiple previous anaesthetics, but was otherside well. His cardio-respiratory status (normotensive) and neurological status were normal. FBP, Coagulation Screen and U & E were all within acceptable limits Preoperative medication included bicarbonate and calcium supplements,

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CORONERS ACT (Northern Ireland), 1959

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as follows to wit:–

The Deposition of R H TAYLOR

of *(Address)*

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to 20-21 mmHG, corresponding to an actual increase of 3-4 mmHg. This is a relatively mild increase in CVP and is necessary in such cases to provide the child's tissues with sufficient water, sugar and electrolytes. The heart rate also gives evidence of fluid status. Although this is 'blocked' by the administration of atropine at the start of the case there was a gradual decrease throughout the procedure (120-100 beats/ minute) consistent with the clearance of atropine and gradual rehydration. All the more important in this case is the need to avoid dehydration that will deprive the donor kidney of sufficient fluid to produce urine. There are several feedback systems in the body which act to retain fluid (ADH, renin-angiotensin ANP etc). These decrease urine output, thus it is necessary to prevent these systems becoming activated for successful transplants. The systolic BP increased, in accordance with the CVP, and was stable at around 100 mmHg throughout most of the case. It is vital to provide sufficient BP to perfuse the vital organs and the donor kidney. A low-dose dopamine infusion (5 mcg/kg/min) was commenced near the beginning of the case to provide a renal vaso-dilating effect. This dose has minimal (if any) systemic effects and is regarded as routine practice in renal transplantation in centres where I have worked. The haemodynamics (HR, CVP, BP, SaO2) were remarkably stable (see print out) despite the ongoing blood loss (>1211 mls almost a full blood volume) which I discussed in my earlier letter. The sudden 'increase' in CVP to 28 mmHg occurred when the table was raised 5-6 inches for surgical reasons, but the transducer was attached to a drip-stand and thus an

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The Deposition of R H TAYLOR

of

who being sworn upon h oath, saith *(Address)*

mmol/l then there was not an excess of this type of solution given. In fact, if less had been given then there would have been a danger of HYPOglycaemia, a much more serious condition in early childhood. I can not explain what has happened. However, I can explain several things that could not have happened. The cerebral oedema was gross and there was x-ray evidence of pulmonary interstitial oedema (no cardiomegaly). Despite aggressive measures to reduce brain swelling, (mannitol x 2, hyperventilation, fluid restriction) he was confirmed brain stem dead. There were no intraoperative 'events' which could account for cerebral oedema eg, hypoxia, hypotension, arrest or anaphylaxis (see print out). There were no external signs of a suffusion of 'hanging' injury (no facial swelling, no petechiae, no sub-conjunctival haemorrhages) causing fluid to sequestrate in the brain. Also the presence of pulmonary oedema is against such a notion. Also there was no associated signs of raised Intracranial Pressure (ICP) such as Hypertension & Bradycardia. The heart rate 'drifted' lower over the first hour (120-100 beats per minute- see print out) of the operation consistent with the effects of atropine. Thereafter the heart rate remained stable until towards the end of surgery when neuromuscular reversal was given (neostigmine/glycopyrollate). I am familiar with all the anaesthetic equipment used, which was checked prior to the case. Records show they were recently and routinely serviced. As one of the paediatric anaesthetists working in the RBHSC my contribution to the vital aspect of equipment safety had been to order the purchase and installation of oxygen monitors (FiO2), capnographs (CO2),

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P.T.O.

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CORONERS ACT (Northern Ireland), 1959

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The Deposition of R H TAYLOR of

(Address)

who being sworn upon his oath, saith

appropriate, expert and representative of the highest quality and intensity of care that I can provide. With regard to the cause of death I cannot understand the finding of "impaired cerebral perfusion". I cannot understand why a fluid regime employed successfully with Adam previously, led on this occasion to dilutional hyponatraemia. I do not know if in fact there was impaired blood flow from the brain & if there was, whether it was a factor in this case. I had no knowledge of the state of death until Dr Savage told me. I believe the underlying cause of the cerebral oedema was hyponatraemia (not dilutional during renal transplant operation. In Adam's case it was not practical to carry out electrolyte tests at the commencement of surgery.

Miss Higgins: I believe I was involved in previous surgery concerning Adam. I saw the scar on Adam's neck. It was reasonable to attempt access to the same site. I believe it is possible to ~~have~~ place lines in ligated veins. On this occasion I was unable to speak to Miss Strain prior to surgery. Adam had not a sodium deficiency — it was being

P.T.O.

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No.

CORONERS ACT (Northern Ireland), 1959

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The Deposition of R.H. TAYLOR

of

who being sworn upon h is oath, saith (*Address*

I agree with the view expressed by Dr Alexander.

Witness asked if he believed death could have been avoided but claimed privilege.

Mr. Bingham: The purpose of the machine is to analyse blood gases. Electrolyte measurements are normally carried out in our labs. I would not rely on the machine accurately analyse sodium levels. That is a common practice in the RBHSC. We measured the total number of fluids given against those omitted. The bladder being opened did affect my calculations. I believed the tip of the catheter was not in close relation to the heart, I confirmed this manually by touching. There is no clear view on venous drainage from the brain. If there had been such a problem I would not have been able to be aware of it. If anything had gone to plan when the clamps were released surgery would have been completed soon afterwards. The fluids I gave were isotonic — the same potential as plasma which should

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TRANSCRIPTION OF DEPOSITION OF DR R H TAYLOR

With regard to the cause of death I cannot understand the finding of "impaired cerebral perfusion". I cannot understand why a fluid regime employed successfully with Adam previously, led on this occasion to dilutional hyponatraemia. I do not know if in fact there was impaired blood flow from the brain and if there was, whether it was a factor in this case. I had no knowledge of the other 9 deaths until Dr Savage told me. I believe the underlying cause of the cerebral oedema was hyponatraemia (not dilutional) during renal transplant operation. In Adam's case it was not practical to carry out electrolyte tests at the commencement of surgery.

Miss Higgins: I believe I was involved in previous surgery concerning Adam. I saw the scar on Adam's neck. It was reasonable to attempt access to the same site. I believe it is possible to place lines in ligated veins. On this occasion I was unable to speak to Miss Strain prior to surgery. Adam had not a sodium deficiency - it was being managed successfully. There was no reason to believe there would have been a change in electrolytes between 11.00 pm and 6.45 am. nothing in that period happened to change that. Adam was the only child with polyuric renal failure I have anaesthetized for renal transplant. He needed a greater amount of fluid because of the nature of the operation. I believe the fluids given were neither restrictive or excessive. The new kidney did not work leading to a re-assessment of the fluids given. This made us think we have underestimated fluid and we gave a fluid bolus at 9.32. I checked CVP as soon as I had inserted the line (about 7.30 am). The monitor gave a continuous display and there was a computerised print-out also. The electrolytes at 9.30 were not in an acceptable range. We felt we had taken adequate measures to stop the sodium falling further and to increase it. The skin closure stage of the operation was reached at 11.00 am. We were considering taking another electrolyte test in conjunction with other tests at the end of the operation. I was aware of the Arieff article when it was first published. In hindsight I cannot say what I would have done differently. I do not believe turning the head to one side impaired venous drainage. The catheter in the right subclavian vein - I do not know if it had any effect on drainage. I cannot explain the mercury reading of 17 but I agree with the views expressed by Dr Alexander.

Witness asked if he believed death could have been avoided but claimed privilege.

Mr Brangham: The purpose of the blood/gas machine is to analyse blood gases. Electrolyte measurements are normally carried out in our Labs. I would not rely on the machine to accurately analyse sodium levels. That is a common practice in the RBHSC. We measured the total number of fluids given against those emitted. The bladder being opened did affect my calculations. I believed the tip of the catheter was not in close relation to the heart. I confirmed the manually by touching. There is no clear view on venous drainage from the brain. If there had been such a problem I would not have been able to be aware of it. If everything had gone to plan when the clamps were released surgery would have been completed soon afterwards. The fluids I gave were isotonic - the same potential as plasma which should have mimicked those that Adam previously received. I produce a further statement C5.