CORONERS ACT (Northern Ireland), 1959

Deposition of Witness taken on the day of 19 , at inquest touching the death of ADAM STRAIN , before me Coroner for the District of 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

P.T.O.

red blood cells were given. His haemoglobin at the start of the procedure was 10.5 g/dl and fell to an estimated 6.1 g/dl during the case and was 10 g/dl at the end. The nurses were asked to weigh blood soaked swabs during the case so that they could be more correctly assessed. There was 328 mls of blood loss in the swabs, 500 mls of blood in the suction bottle and an unknown amount in the towels and drapes. I estimated this to be about 300 mls, but they were heavily soaked. Thus the total blood loss I estimated to be 1128 mls. The replacement for this included 2 packed cells (180-250 mls each) and 1000 mls of HPPF. The infusion of fluids was titrated against the CVP and BP to ensure that the blood volume was more than adequate to permit maximum perfusion of the donor kidney. This process was complicated by the fact that th( )onor kidney did not appear well perfused after an initial period)f apparently good kidney perfusion. A low dose dopamine infusion had)een commenced near the start of the case to improve the blood flow of :he donor organ. The pulse rate, CVP and arterial blood pressure gave ie no cause for concern throughout the case and a blood gas at 09.30am onfirmed good oxygenation and no sign of acidosis or any indication f problems. In view of the CVP, heart rate and BP I did not consider he fluids to be either excessive or restrictive. Indeed I regarded ne fluids to be appropriate and discussed this with other doctors ...t in the theatre. At the end of the case I reversed the neuromuscular .oc.. with neostigmine and anticipated the child awakening. When there is no sign of this I examined his pupils and found them to be fixed d dilated. I became extremely concerned that he had suffered brain em injury so I transferred him to the PICU for further ventilation his lungs and assessment. In the PICU hyperventilation and mannitol s administered the iv fluids restricted to permit fluid to be drawn t of the oedematous spaces. Along with Dr Savage I spoke to Adam's :her and offered my sympathy for the loss of her child, but could not)ply her with a clear explanation of what had happened to Adam. I :ompanied Adam to the CT-scan room later on that day and was informed

\KEN before me this day of 19

Coroner for the District of (61)

C.V.P. ! ? No concern WHY? NO RECORD OF SODIUM 123

2/

CORONERS ACT (Northern Ireland), 1959

Deposition of Witness taken on the day of 19 , at inquest touching the death of ADAM STRAIN , before me Coroner for the District of as follows to wit:–

The Deposition of R H TAYLOR of *(Address)*

who being sworn upon h oath, saith

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,

(62)

Keflex and erythropoietin. 2. Difficult i.v. access. The paediatric registrar had attempted on several occasions to erect i.v. fluids to further prevent dehydration prior to surgery. This proved impossible and the child came to theatre without iv access. I gained i.v. access on the first attempt and administered a 'routine' paediatric anaesthetic induction with thiopentone 125 mg, atropine 0.3 mg and atracurium 10 mg. A secure iv cannula was then placed on the first attempt as was intubation of the trachea and a right radial arterial line. A central venous line was attempted on 3 occasions in the left subclavian, once in the left internal jugular and then successfully in the right subclavian. With a child in the head-down position failure to locate the subclavian vein suggests that the child is dehydrated. A lumbar epidural was then placed without any difficulty and 'routine' drugs administered (bupivacaine 0.25% and fentanyl 5 mcg/kg). This enables minimal volatile anaesthetics to be given during the case and provides excellent postoperative pain relief. There is other evidence that it may prevent or lessen the 'stress response' which causes fluid retention (decreased urine output). 3. Haemodynamic considerations. On measuring the CVP the initial pressure reading was 17 mmHg. There were both cardiac and respiratory patterns to the waveform confirming correct intravascular placement. However, from the pressure reading, I concluded that the tip of the line was not in close relation to the heart (later confirmed by x-ray). I, therefore, used the initial reading (17 mmHg) as a baseline. The systolic BP at this time was 80-90 mmHg. This is low, but within the normal range for a child of his age without pre-existing hypertension. I, therefore, concluded that the child required more i.v. fluid to increase the CVP and BP from this baseline level. At 20 kg Adam had a calculated blood volume of 1600 mls and calculated fluid requirement of 60 ml/hr. However, he would 'normally' receive a sugar solution at 150 mls/hour. Thus I gave him the deficit of fluid 300-500 mls plus his ongoing requirements (50 mls/hour). During the following 30-40 minutes his CVP increased

TAKEN before me this day of 19 ,

Coroner for the District of (63)

070-015-063

IF MAY BE NECESSARY TO DISCUSS THIS 21a TOMORROW MORNING TO DECIDE ON ITS RELEVANCE

C.V.P. was high, I would have concluded that either apparatus was faulty or that there was fluid overload. C.V.P. was more sensitive B.P.

3/

CORONERS ACT (Northern Ireland), 1959

Deposition of Witness taken on the day of 19, at inquest touching the death of ADAM STRAIN, before me

Coroner for the District of

as follows to wit:–

The Deposition of R H TAYLOR

of *(Address)*

who being sworn upon h oath, saith

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

P.T.O.

(64)

'artefact' occurred. When the transducer was 're-zeroed' to take account of the differences in levels the pressure returned to the previous 'stable' range (20-22 mmHg) consistent with no net increase in fluid load or circulating blood volume. When the child was taken to the PICU and his head placed in the midline his CVP was 10-12 mmHg suggesting that in theatre, with his head rotated there was some mile venous occlusion of the great veins. There are two small increases in the systolic BP at around 10.00am corresponding to two small boluses of dopamine (1 mcg/kg). The rationale for this was to increase the perfusion pressure (without fluid challenge) to the donor kidney, which at that stage was not 'looking good' and not producing urine. 4. Intraoperative Fluids. This is the area requiring the greatest consideration and I keep returning to it. It is my practice and teaching that fluids must be carefully calculated in realtion to the child's size and requirements. Furthermore Colloid (Hartmanns is preferred to Dextrose solution to replace blood losses. In this case HPPF and Hartmanns (500 mls) were given for volume expansion (to raise and maintain the CVP 3-4 mmHg above baseline). The blood loss (> 1211 mls) was carefully balanced by administration of colloid (HPPF 1000 mls and 2 units Packed Cells). This is also confirmed by observing the haemoglobin concentration. The initial haemoglobin was 10.5g/dl, fell to 6.1 during the case, confirming significant blood loss and was restored by careful calculation to 10.1 at the end of the procedure. The glucose containing crystalloid was given over 4 hours (1,500 mls 0.18 NaCl/4% Glucose), again carefully calculated to restore the deficit (>300 mls), supply maintenance 150ml/hr (in view of the polyuria) and insensible losses (large area of abdominal cavity exposed). The calculation was complicated and included many subjective factors not easily measured (skin colour, skin mottling, peripheral perfusion, pulse volume, pulse response to fluid bolus, etc.) which become 'natural' for an anaesthetist. In the final analysis the blood sugar gives a reliable indication of the quantity of glucose solution given. Since the blood sugar at the end of this case was 4

KEN before me this day of 19 ,

Coroner for the District of

3/a

65

4/

CORONERS ACT (Northern Ireland), 1959

Deposition of Witness taken on the day of 19 , at inquest touching the death of ADAM STRAIN , before me

Coroner for the District of

as follows to wit:–

The Deposition of .. R H TAYLOR

of *(Address)*

who being sworn upon h oath, saith

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),

P.T.O.

SCRIBED AS "J" BY PJ BERRY
V
THERE MOST CERTAINLY (*) AS FACIAL SELLING

66

quipment log-books and printed records of actual monitoring measurments.

f there had been an equipment malfunction (and there is NO evidence in his case) then back-up systems would show it. For instance an arterial lood gas at 09.30 confirms that both the CO2 and Oxygen monitors (SaO2) ere accurate in this case. If the BP was lower than that displayed malfunction) then the blood gas would have indicated a metabolic acidosis ıypo-perfusion of tissues). In fact the blood gas did NOT indicate a etabolic acidosis confirming that the BP was adequate for full tissue erfusion. The heart rate and BP are also consistent between the theatre ıd PICU monitors in this case. Conditions likely to precipitate ısmotic' fluid shifts were not present. Adam's preoperative albumin is 38 mmol/l and other electrolytes were in an acceptable range.

.tl gh blood sugar was not measured during the case the final blood ıgar was 4 mmol/l. There is no reason to believe that it was much fferent from this during the case as he was receiving basic sugar ntaining fluids. Appropriate quantities and types of fluid were ven, as I have set out above. This is confirmed by the fluid lculations, Heart rate, CVP, BP, haemoglobin concentration, blood sugar d autopsy (no evidence of fluid overload). In fact there is no evidence at excessive quantitites or incorrect types of fluid were given.

other difficulty in attempting to explain the cerebral oedema is the hat Adam received cerebral-protective drugs during the operation, t specific reasons, but for other purposes. Thiopentone was used r induction and, being a barbituate, has well documented cerebral- otective effects, especially when given prior to the brain 'insult'. ednisolone was given for 'anti-rejection' therapy and, being a steroid, also recognised as a cerebral-protective agent. Conclusion; By the eful exclusion of possible causes I can only assume that 'something' curred during this case which defies physiological explanation. I ain totally devastated by this unexpected, unexplained and tragic ith of a 4 year old boy during a complicated operation. My only ısolation is that I consider the management to have been caring,

AKEN before me this day of . 19 ,

Coroner for the District of

4/a

67

5/

CORONERS ACT (Northern Ireland), 1959

Deposition of Witness taken on the day of 19 , at inquest touching the death of ADAM STRAIN , before me Coroner for the District of as follows to wit:–

The Deposition of R H TAYLOR

of *(Address)*

who being sworn upon h oath, saith

appropriate, expert and representative of the highest quality and intensity of care that I can provide.

68

070-015-069

TRANSMISSION VERIFICATION REPORT

TIME: 20/06/1996 13:25
NAME: EDWARDS & EDWARDS
FAX : [redacted]
TEL : [redacted]

TIME	20/06 13:18
FAX NO./NAME	244215
DURATION	00:06:46
PAGE(S)	10
RESULT	OK
MODE	STANDARD

69.