

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 his 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.

UTV

069A-094-373 TOTAL P.05

lood 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 the donor kidney did not appear well perfused after an initial period of apparently good kidney perfusion. A low dose dopamine infusion had been commenced near the start of the case to improve the blood flow of the donor organ. The pulse rate, CVP and arterial blood pressure gave me no cause for concern throughout the case and a blood gas at 09.30am confirmed good oxygenation and no sign of acidosis or any indication of problems. In view of the CVP, heart rate and BP I did not consider the fluids to be either excessive or restrictive. Indeed I regarded the fluids to be appropriate and discussed this with other doctors present in the theatre. At the end of the case I reversed the neuromuscular block with neostigmine and anticipated the child awakening. When there was no sign of this I examined his pupils and found them to be fixed and dilated. I became extremely concerned that he had suffered brain stem injury so I transferred him to the PICU for further ventilation of his lungs and assessment. In the PICU hyperventilation and mannitol was administered the iv fluids restricted to permit fluid to be drawn out of the oedematous spaces. Along with Dr Savage I spoke to Adam's mother and offered my sympathy for the loss of her child, but could not supply her with a clear explanation of what had happened to Adam. I accompanied Adam to the CT-scan room later on that day and was informed

TAKEN before me this day of 19

Coroner for the District of

C.V.P.
 of No CONCERN
 WHY?
 No RECORD
 of Sodium
 123

UTV

069A-094-374

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 his 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 button at night as he slept. The delivery of such large quantities of food would have profound effects on his metabolism (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 gastrostomy) fluids (Diacoralyte= 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 otherwise 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,

P.T.O.

UTV

069A-094-375

CORONERS ACT (Northern Ireland), 1959

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

3/

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.

and erythropoietin. 2. Difficult i.v. access. The paediatric

*

IF MAY BE

registrar had attempted on several occasions to erect i.v. fluids to

NECESSARY

further prevent dehydration prior to surgery. This proved impossible

TO DISCUSS

and the child came to theatre without iv access. I gained i.v. access

THIS

on the first attempt and administered a 'routine' paediatric anaesthetic

2/a

induction with thiopentone 125 mg, atropine 0.3 mg and atracurium 10 mg.

TOMORROW

A secure iv cannula was then placed on the first attempt as was

MORNING

intubation of the trachea and a right radial arterial line. A central

TO DECIDE

venous line was attempted on 3 occasions in the left subclavian, once

ON ITS

in the left internal jugular and then successfully in the right

RELEVANCE

subclavian. With a child in the head-down position failure to locate

the subclavian vein suggests that the child is dehydrated. A lumbar

caudal 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

85-90 mmHg. This is low, but within the normal range for a child of

this 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

(150 mls/hour). During the following 30-40 minutes his CVP increased

TAKEN before me this

day of

19

Coroner for the District of

X

UTV

X
C.V.P. was high
I should have concluded
that either apparatus was faulty or he was for
had been overloaded
V.P. was more than 3

069A-094-377

'act' occurred. When the transducer was 're-zeroed' to take account
 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 mild 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's my practice and teaching that fluids must be carefully calculated
 in relation to the child's size and requirements. Furthermore Colloid
 or 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

3/a

*

3/3/22

UTV

TAKEN before me this day of 19

Coroner for the District of

069A-094-378

CORONERS ACT (Northern Ireland), 1959

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

4/

Coroner for the District of

as follows to wit:-

The Deposition of R H TAYLOR

of (Address) who being sworn upon his 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/glycopyrrolate) I am familiar with all the anaesthetic equipment used, which was checked prior to the case. Records show they were recently and routinely service 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),

DESCRIBED AS 'PUFFY' BY PS BERRY
↓
THERE WAS NO FACIAL SWELLING

UTV

069A-094-379

P.T.O.

ent log-books and printed records of actual monitoring measurements.

there had been an equipment malfunction (and there is NO evidence in

this case) then back-up systems would show it. For instance an arterial

blood gas at 09.30 confirms that both the CO2 and Oxygen monitors (SaO2)

were accurate in this case. If the BP was lower than that displayed

(malfunction) then the blood gas would have indicated a metabolic acidosis

(hypo-perfusion of tissues). In fact the blood gas did NOT indicate a

metabolic acidosis confirming that the BP was adequate for full tissue

perfusion. The heart rate and BP are also consistent between the theatre

and PICU monitors in this case. Conditions likely to precipitate

'osmotic' fluid shifts were not present. Adam's preoperative albumin

was 38 mmol/l and other electrolytes were in an acceptable range.

Although blood sugar was not measured during the case the final blood

sugar was 4 mmol/l. There is no reason to believe that it was much

different from this during the case as he was receiving basic sugar

containing fluids. Appropriate quantities and types of fluid were

given, ~~as I have set out above~~. This is confirmed by the fluid

calculations, Heart rate, CVP, BP, haemoglobin concentration, blood sugar

and autopsy (no evidence of fluid overload). In fact there is no evidence

that excessive quantities or incorrect types of fluid were given.

Another difficulty in attempting to explain the cerebral oedema is the

fact that Adam received cerebral-protective drugs during the operation,

not for specific reasons, but for other purposes. Thiopentone was used

for induction and, being a barbituate, has well documented cerebral-

protective effects, especially when given prior to the brain 'insult'.

Prednisolone was given for 'anti-rejection' therapy and, being a steroid,

is also recognised as a cerebral-protective agent. Conclusion; By the

careful exclusion of possible causes I can only assume that 'something'

occurred during this case which defies physiological explanation. I

remain totally devastated by this unexpected, unexplained and tragic

death of a 4 year old boy during a complicated operation. My only

consolation is that I consider the management to have been caring,

TAKEN before me this day of 19 ,

Coroner for the District of

4/a

UTV

069A-094-380

of
ADAM STRAIN

, before me
Coroner for the District of

as follows to wit:-

The Deposition of R H TAYLOR

(Addre.

of
who being sworn upon h oath, saith

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

Lined area for deposition text.

TOT

UTV

069A-094-381