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MEDICAL REPORT

ON

ADAM STRAIN (DECEASED)

Prepared for: John L Leckey LL.M.
H M Coroner
Coroner's Office
Courthouse
Crumlin Road
Belfast
N. Ireland
BT14 6AL.

By: Edward Sumner MA, BM, BCh, FRCA
Consultant Paediatric Anaesthetist
Great Ormond Street Hospital for Children
NHS Trust
Great Ormond Street
London WC1N 3JH

22 January 1996

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

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THE QUEEN'S UNIVERSITY OF BELFAST
NORTHERN IRELAND OFFICE

REPORT OF AUTOPSY

Name: Adam STRAIN **Sex:** Male **Age:** 4 yrs. **F.No:** 46,728

Date of Death: 28th November, 1995. MDEC

Date and Hour of Autopsy: 29th November, 1995. 2.40 p.m.

Place of Autopsy: The Mortuary, Royal Victoria Hospital, Belfast.

CAUSE OF DEATH:

I (a) CEREBRAL OEDEMA

due to

(b) DILUTIONAL HYPONATRAEMIA AND IMPAIRED CEREBRAL PERFUSION DURING RENAL TRANSPLANT OPERATION FOR CHRONIC RENAL FAILURE (CONGENITAL OBSTRUCTIVE UROPATHY)

On the instructions of H.M. Coroner for Greater Belfast, Mr. J. L. Leckey, LLM, I, Alison Armour, MB, BCh, MRCPath, DMJ(Path), registered medical practitioner and pathologist approved by the Northern Ireland Office, made a postmortem examination of the body of -

ADAM STRAIN
aged 4 years

identified to me at the Mortuary, Royal Victoria Hospital, Belfast, on Wednesday, 29th November, 1995, by Constable S. R. Tester, R.U.C. Grosvenor Road.

THE QUEEN'S UNIVERSITY OF BELFAST
NORTHERN IRELAND OFFICE

REPORT OF AUTOPSY

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Date of Death: 28th November. 1995. MDEC

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Place of Autopsy: The Mortuary, Royal Victoria Hospital, Belfast.

HISTORY:

He was a child and lived with his mother and grandparents in a bungalow in the town. He was born with a renal abnormality - an obstructive uropathy which resulted in polyuric renal failure. He had five ureteric reimplant operations, a fudoplication for gastro-oesophageal reflux and more recently in October, 1995 an orchidoplexy. He ate nothing by mouth and was fed via a gastrostomy button 1,500 mls. at night and 900 mls. during the day. He also received peritoneal dialysis. He was being prescribed calcium carbonate, Keflex, iron, one alpha vitamin, sodium bicarbonate and erythropoietin.

On 26th November, 1996, he was admitted to the Royal Belfast Hospital for Sick Children at 11.30 p.m. for a renal transplant operation. His blood pressure was 108/56 and a haemoglobin of 10.5 g/dl with a sodium of 139 mmol/1, potassium 3.6 mmol/1 and urea 16.8 mmol/l. Overnight he was given 900 mls. dioralyte (4% dextrose 0.18% saline). Peritoneal dialysis was performed as usual , 750 ml. fluid volume 1.36% dextrose solution. He was given 8 cycles before going to theatre the next morning.

He arrived in theatre at 6.45 a.m. and general anaesthesia was induced using thiopentone, atropine and atracium. Intravenous access was difficult and attempts were made to pass a central venous pressure catheter. Three attempts were made with the left subclavian vein, one with the left internal jugular vein and then the catheter was successfully passed into the right subclavian vein. A lumbar epidural between L1 and L2 was also sited with 0.25% bupivacaine and Fentanyl 5 mcg/kg. Apart from the anaesthetic drugs Augmentin an antibiotic, prednisolone, asathioprin (anti-rejection drug) and a continuous infusion of dopamine were administered intravenously. An initial central venous pressure reading was taken at 17 mm.Hg. Intravenous units were administered from 7.00 a.m. to 8.30 a.m., of three 500 ml. bags of dextrose saline (4% and 0.18%). The operation technically was difficult due to previous surgical procedures and there was an increase in blood loss, calculated to be approximately 1,200 mls. at the end of the procedure. Further fluids of 500 mls. Hartman's solutions 1,000 mls. of HPPF (human plasma protein fraction) and 500 mls. of packed cells were administered. At 9.32 a.m. a blood gas analysis revealed a sodium of 123 mmol/l (normal 135 - 145) and a haematocrit of 18% (normal. 35 - 40%). During the procedure the CVP rose to 20 -21 mm.Hg, the Hb was 6.1 g/dl which was 10.1 g.dl. at the end of the procedure and the blood pressure rose and the pulse rate gradually decreased. The donor kidney perfused and the operation was completed. At the end of the procedure the neuromuscular block was reversed with neostigmine but this boy did not wake up. His pupils were noted to be fixed and dilated at midday. He was transferred from theatre to the paediatric Intensive Care Unit at 12.05 p.m. He was intubated and hand ventilated on admission. He was treated with intravenous mannitol and intravenous fluids were restricted. An emergency CT scan at 1.15 p.m. revealed gross cerebral oedema. His body temperature was 36.5°C. the CVP was 30, heart rate 120 beats per minute and systolic blood pressure 120. Electrolytes revealed a

sodium of 119 mmol/l; and a chest X-ray revealed pulmonary oedema with the CVP catheter tip in a neck vessel. Neurologists carried out brain stem tests and life was pronounced extinct by a hospital doctor on 28th November, 1995 at 9.15 a.m.

EXTERNAL EXAMINATION:

The body of a young male child, 104 cm. in length and weighing 20 kilograms. Rigor mortis was present. Hypostasis of light purple colour stained the back of the body.

Back: There was a needle puncture mark in the midline, centred 11 cm. above the natal cleft, corresponding to an epidural cannula.

Eyes: The corneas had been taken for transplantation.

Ears: Normal.

Nose: Normal.

Neck: There was a needle puncture mark on the left side. There was a healed operation scar, 3 cm. long, on the left side. There were two further healed operation scars on the right side, 2.5 cm. long.

Chest : There was a needle puncture mark on the left upper chest, in the region of the subclavian vein. There were a number of bruised needle puncture marks on the right upper chest, corresponding to a subclavian line .There was a bruise, 1.5 x 1 cm., in the left upper chest, centred 3 cm. lateral and 1 cm. above the left nipple. There was a bluish-blackish bruise on the right chest, 2.5 x 1 cm., diameter, centred 3 cm. lateral to the right nipple.

Abdomen: There was a gastrostomy button situated in the left hypochondrium. The gastrostomy hole measured 6 mm. diameter. There was a healed operation scar, 18 cm. long, horizontally in the upper abdomen, corresponding to previous fundoplication. There was a further healed operation scar, 18 cm. long, traversing the mid-abdomen. There was a peritoneal dialysis tube in situ in the left upper abdomen. There were two further puckered scars, one situated in the left side of the lower abdomen, 5 cm. lateral and 2 cm. below the umbilicus. The other puckered scar was situated 4.5 cm. beneath the umbilicus. There was a recent elliptical surgical incision, 15 cm. long, on the right side of the lower abdomen with a drain protruding from its upper margin. Its edges were slightly bruised. A bladder catheter protruded from the lower end on the left side of the abdomen. There was a further drain in situ just at the level of the pubic bone, corresponding to the donor ureteric catheter.

Left Upper Limb: There were a number of bruised needle puncture marks in the fold of the elbow and a healed operation scar, 5 cm. long, again in the fold of the elbow.

Right Upper Limb: There were a number of bruised needle puncture marks in the fold of the elbow.

Left Lower Limb: There were a number of petechial bruises on the inner aspect of the thigh, in an area 4 x 1 cm. There was a bruise, 1 cm. diameter, on the front of the shin. There was a bruised needle puncture mark on the dorsum of the foot.

Right Lower Limb: There was a healed operation scar, 4 cm. long, in the right groin, corresponding to an orchidoplexy. There was a fading bruise, 0.5 cm. diameter, on the outer aspect of the upper thigh. There was a bluish bruise on the outer aspect of the thigh, 0.5 cm. diameter, and there were a number of fading bruises on the front of the shin. There were two bruised needle puncture marks on the dorsum of the foot.

Scrotum: There was a healed operation scar, 3 cm. long, on the right scrotal sac. The right testis had been removed. The left testis was present

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INTERNAL EXAMINATION:

HEAD:

Brain: To be described after fixation.

Mouth: There were natural teeth in good condition in each jaw. The lips were dry and parchmented. The tongue was held between the clenched teeth.

Tongue, Pharynx: Normal.

NECK AND CHEST:

Hyoid Bone and Laryngeal Cartilages: Intact.

Thyroid Gland: Normal.

Pericardial Sac: Normal.

Heart: 120 gm. The organ was taken for transplantation.

Aorta: Normal.

ABDOMEN:

Abdominal Cavity: Was crossed by a number of adhesions. There was a little blood clot formation around the renal transplant on the right side.

Stomach: A gastrostomy hole was present. The stomach contained a little bile.

Intestines: Externally appeared normal.

Duodenum: Normal.

Liver: Weighed 875 gms. A little congested.

Gall Bladder: Normal.

Pancreas: Normal.

Native Kidneys: Both were markedly contracted, scarred and contained a number of cysts. Little normal functioning kidney remained. Both ureters were hugely distended and dilated.

Transplanted kidney: Was in situ in the right pelvis, the ureter drained freely and the vascular attachments were intact.

Bladder: Contained a little straw-coloured urine.

Prostate: Normal.

SPINAL CORD: To be described after fixation.

INTERNAL EXAMINATION OF NECK:

There was no evidence of congestion or obstruction of the major blood vessels or the carotid arteries and jugular veins. There was no evidence of superior vena caval obstruction. The carotid arteries were normal. There was a suture in situ on the left side of the neck at the junction of the internal jugular vein and the sub-clavian vein.

DESCRIPTION OF ORGANS AFTER FIXATION:

Brain - Was cut on 12.1.96

External Examination: Fixed weight of brain 1,680 gm; cerebellum and brain stem 176 gm; cerebellum only 154 gm. The brain was grossly swollen with loss of sulci and uncal swelling. This was symmetrical. There was no uncal necrosis. There was swelling of the cerebellar tonsils but no necrosis. There was no cortical venous thrombosis. The anatomy of the circle of Willis was normal.

On cut section there was massive brain swelling and constriction of the ventricles. There was no ventricular haemorrhage. There was no asymmetrical lesion. There was severe white matter congestion and marked congestion of the blood vessels in the basal ganglia, white matter and deep grey matter. There was no necrosis of the mid-brain or brain stem.

Blocks were taken from:

1. Right frontal white matter
2. Left cingulate gyrus
3. Left basal ganglia
4. Right and left hippocampus
5. Left occipital lobe
6. Cerebellum
7. Pons in toto
8. Thalamus

The brain was photographed sequentially

Cervical Cord: No macroscopical lesion seen.

Blocks were taken from:

1. Cervical
2. Thoracic
3. Lumbar

MICROSCOPY:

Lungs: There was congestion of the capillaries and there were moderate numbers of alveolar macrophages. There was no evidence of embolism or infarction.

This is a highly complex and difficult case. To try to understand the underlying cause for this cerebral oedema first some physiological mechanisms for maintaining fluid and electrolyte balance will be reviewed. .

In healthy people the composition of body fluids vary within narrow limits. The kidneys are largely responsible for maintaining this constancy and the excretion of waste products of metabolism represents merely one aspect of this task. The control of water volume and sodium are maintained by the hormones A.D.H. (anti-diuretic hormone) and aldosterone.

In this case the volume of urine output was greatly increased and the urine was also dilute. This was probably due to the fact that the kidneys did not function and their ability to concentrate the urine was minimal.

Generalised cerebral oedema in children has many causes including hypoxia. In this case this has been excluded. The history indicates that during the operation this little boy received a quantity of intravenous fluids. There was also a considerable blood loss during the operation of 1,200 mls. However a CVP, central venous pressure, catheter was in situ in the right subclavian vein and is usually in place to avoid overloading of the circulation by intravenous fluids. A rise in the CVP indicates an excessive load and a fall can be an early sign of haemorrhage. In this case the initial reading was 17 mm.Hg. (for an operation such as this 10-12 mm.Hg. is the norm) and this was taken as the base line. A subsequent reading was a little higher again. Also during the operation the sodium was low along with the haematocrit. It is known that a condition called dilutional hyponatraemia can cause rapid and gross cerebral oedema. This is no doubt in this case that the sodium level was low during the operation. A study revealed that in children undergoing operations there was substantial extra renal loss of electrolytes and with a minimal positive balance of hypotonic fluid could lead to fatal hyponatraemia. This study however must be taken in context as it refers to healthy children undergoing operations like tonsillectomies. Thus they had normally functioning kidneys which was not the situation in this case. It seems likely therefore that the hyponatraemia in this case was the cause of the cerebral oedema and most of the intravenous fluids given in the cases sited in this paper were administered as 280 mmol glucose per litre in water or in sodium chloride 38 mmol/l.

Another factor to be considered in this case is cerebral perfusion. The autopsy revealed ligation of the left internal jugular vein. The catheter tip of the CVP was situated on the right side. This would mean that the cerebral perfusion would be less than that in a normal child. This would exacerbate the effects of the cerebral oedema and should also be considered as a factor in the cause of death. Therefore the most likely explanation is that the cerebral oedema followed a period of hyponatraemia and was compounded by impaired cerebral perfusion.

The autopsy also revealed changes in the kidneys, in keeping with chronic renal failure and total infarction of the transplanted kidney. These played no part in the fatal outcome.

There were marks due to treatment and bruises to both legs. They were trivial however.

REFERENCES:

Arieff et all
"Hyponatraemia and death or permanent brain damage in healthy children"
British Medical Journal 1992; 304; 1218-22

A. Amer

CURRICULUM VITAE

OF

EDWARD SUMNER, MA BM BCh FRCA

FEBRUARY 1995

NAME: Edward Sumner

ADDRESS: [redacted]

DATE OF BIRTH: [redacted]

MARITAL STATUS: [redacted]

DEGREES HELD:

MA, BM, BCH Oxford 1966
FRCA London 1971

ACADEMIC DISTINCTIONS:

First Class Honours Degree
Animal Physiology
Oxford 1963

Nuffield Prize
Primary Fellowship
London 1969

DATE OF MEDICAL REGISTRATION: March 1968

PRESENT APPOINTMENT:

Consultant Anaesthetist - Great Ormond Street Hospital for Children NHS Trust
Great Ormond Street
London WC1N 3JH

Appointed September 1973

Director of the Department of Anaesthetics : 1987 - 1992

Director: Cardiac Intensive Care Unit : 1988 - 1993

Honorary Senior Lecturer : University of London

I have six operating lists each week with cardiac and general paediatric surgery and I am involved in all aspects of paediatric anaesthesia. Approximately 12,000 anaesthetics per year are administered at Great Ormond Street Hospital for Children. My appointment also includes sessions in the Cardiac ICU with more

than 600 patients per year requiring care. The respiratory support service for the whole hospital developed under my supervision.

I am involved in the training of 24 new residents each year, plus at least 12 seconded residents.

PREVIOUS APPOINTMENTS:

House Officer Medicine and Surgery	University College Hospital London	Jan 1967-Feb 1968
S.H.O. Anaesthetics	University College Hospital London	Mar 1968-Feb 1969
Registrar Anaesthetics	University College Hospital London	Mar 1969-Jan 1971
Senior Registrar Anaesthetics	St Thomas' Hospital London	Nov 1971-Sep 1973
	Including 6 monthly rotations to National Hospital for Nervous Diseases, London, and National Heart Hospital, London.	

After my consultant appointment, I was seconded on a part-time basis to the Nuffield Research Department, Royal College of Surgeons, London, for research experience with Mass Spectrometry for the year 1974.

TEACHING EXPERIENCE:

Teaching in the Institute of Child Health, London:

Annual Advanced Course in Paediatrics

Annual Course in Paediatric Intensive Care

Twice Annual Final Fellowship Course (part of the London Hospitals' Course)

Annual St Bartholomew's Hospital Final Fellowship Course.

British Council Course, Paediatric Cardiac Surgery - Teaching Respiratory Support - 1978, 79, 80, 81, 83, 84, 85, 86, 87, 90 and 92.

Courses in Paediatric Anaesthesia to Danish Anaesthetists and Norwegian Anaesthetists - 1980, 1982, 1990 and 1995.

Presented papers at the Association of Paediatric Anaesthetists:

London 1974 : Mass Spectrometry - Clinical Applications.

Newcastle 1976 : Wilson-Mikity Syndrome

Edinburgh 1982 : Congenital Diaphragmatic Hernia

Dublin 1985 : Management of Phrenic Palsy in the Infant.

London 1988 : Analgesia for Newborns

Royal College of Anaesthetists Final Fellowship Course 1974, 78, 80, 84, 88, 89 - 1995.

Continuing Medical Education Day - 1991, 1993, 1994

1982 European Congress, London.

Paper - Congenital Diaphragmatic Hernia
Poster - Caudal Analgesia

Royal Society of Medicine

1982 - Congenital Diaphragmatic Hernia
1982 - Paediatric Anaesthesia Symposium

1980 Stockholm, Sweden : Paediatric mechanical ventilation.

1980-1984 Liege, Belgium - 5 visits. Paediatric cardiac anaesthesia.

1982 Bonn, Germany - Cardiac anaesthesia.

Visiting Professor, Sydney, Australia, Royal Alexandra Children's Hospital - December 1981.

Lecturer to the British Council Sponsored Workshop in Neonatal Surgery and Intensive Care: Delhi - March 1983 : Jaipur - 1984.

Prague 1983 - Congenital Diaphragmatic Hernia.

Royal College of Surgeons, London: Symposia:

1983 Paediatric Anaesthesia
1984 Neonatal Emergencies
1985 Paediatric Anaesthesia
1987 Paediatric Anaesthesia in the District Hospital

Paris 1983 - Congenital Diaphragmatic Hernia.

Manila, Phillipines 1984 - Total Intravenous Anaesthesia in Paediatrics. World Congress.

Lectures at Intensive Care Meetings:

Birmingham 1984
Rotterdam 1984
Tubingen 1984

Lectured at Thoracic Anaesthetic Meeting, London 1983, 1984.

Open Heart Surgery Congress - Bombay 1985.

Pulmonary Hypertensive Crisis - Paediatric Intensive Care. Brussels 1985.

British Council Lecturer in Paediatric Anaesthesia - Kathmandu, Nepal : 1986, 1987.

Association of Anaesthetists, London:

1986 - Neonatal Analgesia
1987 - Cyclopropane

Neonatal Anaesthesia - Gothenburg, Sweden : 1986

Neonatal Anaesthesia - Oslo, Norway : 1986.

Paediatric Anaesthesia, Basel, Switzerland : 1987, 1994.

Lectures:

Barbican - ICU Update
September 1991

: Respiratory Support in Paediatrics

Oporto
September 1991
: Pulmonary hypertension
: Cardiothoracic anaesthesia for children.

Coimbra
May 1992, May 1994
: Neonatal topics
: Cardio-respiratory physiology
: Renal physiology
: Fluid management
: Pain management

3rd World Congress of Paediatric Anaesthesia
Amsterdam
June 1992
: Transplantation: Are children different?

Munich
July 1992
: Neonatal anaesthesia

Royal College of Anaesthetists
Fellowship Course
1992
: Respiratory support in paediatrics

1992 : Knights of Malta Lecturer, University of Bologna (Italy)

1993/4 : Invited speaker, Munich, Mannheim, Amsterdam, Brussels, Bergamo, Florence, Jerusalem.

PUBLICATIONS:

PAPERS:

Quinsy tonsillectomy: A safe procedure. Sumner E (1973) Anaesthesia 28: 558.

Porphyria in relation to surgery and anaesthesia. Sumner E (1975) Annals of the Royal College of Surgeons, England, 56: 81.

The use of tolazoline in congenital diaphragmatic hernia. Sumner E and Frank DJ (1981) Archives of Disease in Childhood 56:350.

Congenital diaphragmatic hernia: improved prognosis. An experience of 62 cases over 2 years. marshall A and Sumner E (1982) Journal of the Royal Society of Medicine 75: 607.

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Tracheal perforation in newborns. Macleod B and Sumner E (19876) Anaesthesia 41, 67.

Prune Belly Syndrome - anaesthetic hazards. Vallis C, Henderson A and Sumner E (1987) Anaesthesia 42: 54.

Fatal intraoperative tumor embolus in a child with hepatoblastoma. Dormon F, Sumner E and Spitz L (1985) Anesthesiology 63: 692.

Halothane hepatitis in a baby. Whitburn R and Sumner E (1986) Anaesthesia 41: 611.

The use of opioids in neonates. A retrospective study of 933 cases. Purcell-Jones G, Dorman F and Sumner E (1987) Anaesthesia, 42: 1316.

The use of opioids in neonates. A survey. Dorman F, Purcell-Jones G and Sumner E (1988) Pain (in press)

Macleod B and Sumner E (1987) Neonatal tracheal perforation. Anaesthesia 41: 67-70.

Creagh-Barry P and Sumner E (1992) Neuroblastoma and anaesthesia. Paediatric Anaesthesia 2: 147-153.

Sumner E (1993) Gas exchange in children. Paediatric Anaesthesia 3: 1-3.

Sumner E (1994) Paediatric Anaesthesia. paediatric Anaesthesia 4: 1-2.

CHAPTERS:

Anaesthesia for the older child. Kaufman L and Sumner E (1980) In General Anaesthesia, Ed Gray TC, Nunn JF and Utting JE. 4th Edition. London, Butterworth.

The paediatric patient. Sumner E and Patrick EK (1980) In Preparation for Anaesthesia. Ed Stevens AJ. Tunbridge Wells, Pitman Medical.

Paediatric anaesthesia and intensive care. Sumner E. In Anaesthesia Reviews. Ed Kaufman L. London, Churchill Livingstone.

One 1982
Two 1983
Four 1987

Paediatric Anaesthesia. Sumner E (1984) In Practice of Anaesthesia. Ed Wylie D and Churchill-Davidson CD. London, Lloyd-Luke.

Artificial ventilation of children. Sumner E. In Diagnosis and Management of Paediatric Respiratory Disease. Ed Dinwiddie R. London, Churchill Livingstone. 1989.

Respiratory care in paediatrics. Sumner E (1984) In Anaesthesia and Patient Care. Ed Anis and Salim, Pakistan.

Paediatric Anaesthesia. Sumner E (1988) In Operative Surgery-Paediatric Surgery. Ed Spitz L. London, Butterworth.

Preparation for Anaesthesia: the paediatric patient. Sumner E and Facer EK (1986) Ed Stevens J. Preparation for Anaesthesia. Clinics in Anaesthesiology. London, Saunders. Vol 4.

Unusual Paediatric Conditions. Sumner E and Facer EK (1986) Ed Stevens J. Preparation for Anaesthesia. Clinics in Anaesthesiology. Vol 4.

Postoperative care in surgery for congenital heart disease. Eds Stark and de Leval. Philadelphia, Saunders. 1994.

BOOKS:

Medical Problems and the Anaesthetist. Kaufman L and Sumner E (1980) London, Arnold.

Neonatal Anaesthesia. Hatch DJ and Sumner E (1981) London, Arnold.

Paediatric Anaesthesia. Ed Sumner E and Hatch DJ Clinics in Anaesthesiology ()1985 vol 3)

Neonatal Anaesthesia and Perioperative Care. Hatch DJ and Sumner E (1986) 2nd Edition. London, Arnold.

A Textbook of Paediatric Anaesthetic Practice. Sumner E and Hatch DJ. London, Bailliere-Tindall.

The Surgical Neonate: Anaesthesia and Intensive Care. London, Arnold. Hatch DJ, Sumner E and Hellman J (1995)

In preparation:

The Respiratory System. Sumner E. In Clinical Paediatric Anatomy. Ed Dickson JSR. Oxford, Blackwells. In press.

EDITORIAL DUTIES:

Editor-in-Chief: Paediatric Anaesthesia. Blackwell Science. An International Journal.

LEARNED SOCIETIES:

Association of Anaesthetists of the United Kingdom.

Association of Paediatric Anaesthetists of the United Kingdom.

European Neonatal and Paediatric Intensive Care Society.

European Society of Anaesthesiology.

RESEARCH INTERESTS:

I started the first Paediatric Acute Pain Service in the UK in 1990.

Popularised - epidural analgesia in infants and children.

- axillary artery cannulation in infants.

Projects include - the pulmonary circulation

- gastro oesophageal reflux.

MEDICO-LEGAL WORK:

On the Panel of the Association for Victims of Medical Accidents.