D227

Dr Edward Sumner MA, BM, BCh, FRCA

[redacted]

24 July 2005

Dear Dr Sumner

Re Adam Strain

Thank you for agreeing to review the circumstances surrounding Adam's death and to prepare a report for the PSNI.

Following ongoing concern over the death of Adam and other children in Northern Ireland police have decided to investigate the circumstances of Adam's death. This may involve interviewing doctors involved in his treatment. It appears to police at present that the fluid regime precipitated Adam's death and that Dr Taylor was primarily responsible for administering the fluids. Our request to you at this stage is to consider the roles of all the doctors involved at the time when Adam was receiving the fluids which led to his death and advise police if doctors other than Dr Taylor ought to have been aware that:

a. Adam was receiving too much fluid, and should any doctor other than Dr Taylor have taken action in this regard, and
b. no ongoing monitoring of Adam's electrolyte balances was occurring and should any doctor other than Dr Taylor have taken action in this regard.

If, as you read these papers, you have any other observations which you believe would be relevant to our investigations, we would be grateful if you would share these with us.

I have obtained a copy of your report to the Coroner, Mr Leckey, for the Inquest on Adam and it is therefore not necessary for you to repeat the contents of that report.

I provide the following documents for your consideration. They have been obtained from the Coroner's file and were numbered by police in the original copy file. While the numbering of the pages as provided to you is not consecutive, it is adequate for referral purposes and therefore I have not sought to alter it:

1. Statement of Dr Keane, p3.
2. Statement of Dr Savage, pp4-5.
3. Statement of Dr Taylor, p6-12.
4. Deposition of Constable Tester.
5. Deposition of Debra Strain, mother, pp14-16.
6. Deposition of Dr Armour, pp17-19.
7. Deposition of Dr Sumner, pp20-25.
8. Deposition of Dr Alexander, pp26-28.
9. Deposition of Dr Keane, pp29-30.

10. Deposition of Dr Savage, pp31-33.
11. Deposition of Dr Taylor, pp34-40.
12. Report of Dr Alexander, pp41-44.
13. Report of Dr Sumner, pp50-61.
14. Verdict on Inquest, p76-77.
15. Report of Autopsy, p78-85.
16. Charts re Adam Strain (from hospital notes?), pp115-128.
17. Letter from Debra Strain (28/5/96), p150.
18. Report on Equipment, p167-170.
19. Report of Professor Berry, p175-178.
20. Report of Dr Gibson, p180.
21. Letter of Debra Strain (6/2/96), pp193-194.
22. Coroner's note re meeting with Debra Strain, p212.

If there are any documents not provided which you believe may exist and would be of benefit to you, please inform me and I will endeavour to obtain them.

If you could advise me when your deliberations on these documents are complete and you are in a position to write a report, we could agree the most expeditious means to proceed. This death is the subject of a Public Inquiry, whose proceedings may be impeded by the police investigation, and therefore there is a burden on the PSNI to proceed expeditiously.

If I can be of any assistance please contact me at the telephone number or email address on this letter, or on [redacted]

Yours sincerely,

William R Cross
Detective Sergeant

[redacted]

BELFAST CITY HOSPITAL — LISBURN ROAD, BELFAST BT9 7AB

DEPARTMENT OF UROLOGY — TELEPHONE [redacted]
EXTS. [redacted]
FAX [redacted]

REGIONAL UROLOGY SERVICE

MR. P. KEANE - CONSULTANT UROLOGIST

11 December 1995

Mrs S Young
~~Complaints Officer~~
~~Floor~~
Tower Block

Statement of P.F Keane (Consultant Urologist).
c/o BCH, Dept of Urology

~~Dear Mrs Young~~

~~Re: Adam Strain - Deceased~~

~~Just a quick point initially, one of the surgical team was Mr Patrick Keane, Senior Registrar", I am a Consultant which would probably make a difference in this case and that ought to be cleared up with the Coroner first of all.~~

I was asked to transplant this 4 year old boy on Monday 27 November 1995. The operation started at 7.30 am and was technically very difficult because of previous surgery that this young boy had. However, despite the technical difficulties the kidney was successfully put into the child and perfused quite well initially and started to produce urine. At the end of the procedure it was obvious that the kidney was not perfusing as well as it had initially done but this is by no means unusual in renal transplantation. The whole operative procedure took about three hours.

I was informed later on that day that the child had severe cerebral oedema and that he was probably brain dead.

In summary, therefore, the operation was difficult but a successful result was achieved at the end of the procedure.

Yours sincerely

PF Keane
Consultant Urologist
/SH

PS/009-035.

DATE 21 DEC 1995	ACTION	INFO
CHAIRMAN		
CHIEF EXECUTIVE		
CLINICAL SERVICES		
CLINICAL DEVELOPMENT		
FINANCE		
OPERATIONAL SUPPORT		
CONTRACTING		
PERSONNEL		
NURSING		
OTHER DIRECTORS-		

3

The ROYAL HOSPITALS

THE ROYAL BELFAST HOSPITAL FOR SICK CHILDREN

~~TO WHOM IT MAY CONCERN~~

Statement of - Musgrave Ward.

~~RE: ADAM STRAIN~~

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

Adam Strain was a patient with chronic renal failure and polyuria. He developed problems with recurrent urinary infections in infancy and was under the care of Mr.Stephen Brown,Consultant Paediatric Surgeon. He required multiple urological operations for vesico ureteric reflux and a Fundal Plication to correct a hiatal hernia. As a result of infection and reflux his kidneys were damaged and deteriorated to the point where peritoneal dialysis was commenced in 1994. He was then placed on call for a renal transplant. He required multiple medications with Calcium Carbonate,Keflex,Iron,One-Alpha Vitamin D,Erythropoietin and Sodium Bicarbonate and night time gastrostomy tube feeding.

The medications and tube feeds were to ensure good nutrition and to prevent renal anaemia and bone disease. He was a well nourished,well grown boy with height near the 50th centile and weight at the 90th centile for his age. His most recent acute illness was with a gastrostomy exit site infection in July 1995.

On 26th November we had an offer of a kidney from the U.K.Transplant Service. He was admitted to Musgrave Ward RBHSC for pre-operative assessment. His serum electrolytes,haemoglobin and coagulation were satisfactory. H.B.10.5g/dl, Na 139, K 3.6,Urea 16.8,Ca.2.54,Albumin 40,Prothrombin time 12.3. His chest was clear on examination. B.P. 108/56. He was apyrexial. There were no signs of infection. His night gastrostomy feeds are normally 1.5l of Nutrizon. On anaesthetic advice this was changed to clear fluid which was stopped two hours pre op. This meant he had 900mls of Dioralyte overnight. His peritoneal dialysis was performed as usual - 750ml fluid volume 1.36% Dextrose solution. He was given 8 cycles before going to Theatre at 7a.m.

My contact with Theatre during the procedure indicated no major difficulties with cardiovascular status or anaesthesia. Surgery was complex but successful,organ transplantation achieved with acceptably matched kidney from a 16 year old donor.

~~DONOR~~		~~ADAM~~
Age	16 years	4 years
Blood group	A +ve	A+ve
CMV status	negative	negative
Tissue type	A 1,29	A 1,32
	B 8,44	B 14,44
	DR 7,3,	DR 7,8,

4

2 cont.....

Post-operatively Adam failed to breathe spontaneously. On examination he had dilated pupils and bilateral papilloedema.

A chest x-ray showed pulmonary oedema and an emergency CAT brain scan confirmed cerebral oedema and herniation and compression of the brain stem. Neurological testing by Dr.David Webb on the evening of 27/11/95 and the morning of the 28/11/95 confirmed brain death.

Deborah Strain,the mother,and the immediate family were informed of the complications and prognosis regularly throughout these events. Death was certified shortly after 9a.m.on 28th November. ~~Adam's mother offered his organs for donation and this was discussed with the Coroner who felt this not to be appropriate.~~ With the consent and in the presence of the family ventilatory support was withdrawn at 11.30a.m.while Adam was being nursed by his mother.

SIGNED.....Maurice Savage.....

MAURICE SAVAGE
Consultant Paediatric Nephrologist

DATE: 28th November 1995

c.c. Dr.G.Murnaghan Medical Administration RGH
c.c. Dr.B.Taylor Consultant Anaesthetist RBHSC

PATRON: *HRH The Duchess of Kent*

The Royal Victoria Hospital
The Royal Maternity Hospital
The Royal Belfast Hospital for Sick Children

THE ROYAL GROUP OF HOSPITALS AND DENTAL HOSPITAL
HEALTH AND SOCIAL SERVICES TRUST

180 Falls Road, Belfast BT12 6BE Northern Ireland
Telephone: 01232 240503

AS-PSNI

094-192-844

THE ROYAL HOSPITALS

THE ROYAL BELFAST HOSPITAL FOR SICK CHILDREN

30th November 1995

MEDICAL ADMINISTRATION
-1 DEC 1995

Dr. G. Murnaghan,
Director of Medical Administration,
King Edward Building,
RVH.

Statement of RH Taylor MB, FFARCS
Consultant Paediatric Anaesthetist
c/o RBHSC

Dear Dr. Murnaghan,

re: Adam Strain D.O.B. 4.8.91 - Hosp No. 364377

On the 27th November 1995 at 06.45 am 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 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

/ cont over ...

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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 a 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 09.30 am 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 fliuds 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 and iv fluids restricted to permit fluid to be drawn out of the oedematous spaces. Along with Dr Savage I spoke to Adams' 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 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.

Yours sincerely,

R.H. Taylor, MB, FFARCSI.,
Consultant Paediatric Anaesthetist.

c.c. Dr. M. Savage, Consultant Nephrologist, RBHSC
Dr. J. Gaston, Clinical Director ATICS,RVH

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~~Dear Dr Murnaghan~~

I wish to append my previous letter to you in regard to Adam Strain to take account of the post-mortem, which I attended, and other details. As previously mentioned I was very familiar with this child who presented complex management problems for renal transplantation.

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 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 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, Keflex & 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 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 on-going requirements (150 mls/hour). During the following 30-40 minutes his CVP increased 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, SaO_2) 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 "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 mild venous occlusion of the great veins.
There are two small increases in the systolic BP at around 10.00 am 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 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, 1000mls and 2 units Packed Cells). This is also confirmed by observing the haemoglobin concentration. The initial haemoglobin was 10.5 g/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 150 ml/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 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.

~~So what did happen~~?

can not explain what has happened

I ~~do not know~~. However I can explain several things that could not have happened.

~~Cause of death.~~ —
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.

~~Cardiac Arrest?~~

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 or "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 were 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).

~~Equipment?~~

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 (FiO_2), capnographs (CO_2), equipment log-books and printed records of actual monitoring measurements.

If 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 CO_2 and Oxygen monitors (SaO_2) 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.

~~Fluids?~~

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.

~~Brain "Insult"~~?

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

Yours sincerely,

Dr RH Taylor. MB, FFARCSI
Consultant Paediatric Anaesthetist.

CORONERS ACT (Northern Ireland), 1959

Deposition of Witness taken on TUESDAY the 18th 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 Stephen Richard Tester

of Grosvenor Road RUC Station *(Address)*

who being sworn upon his oath, saith

I am a Constable in the Royal Ulster Constabulary. At 9.30 am on Tuesday 28 November 1995 I was made aware of the death of Adam Strain, date of birth, 4 August 1991. I attended at the Royal Belfast Hospital for Sick Children where I was made aware of the circumstances surrounding the death of Adam Strain by Dr Maurice Savage, Consultant Paediatric Nephrologist. I ascertained that life had been pronounced extinct at about 0900 hours that morning by Dr David Webb, Neurologist. The body of Adam Strain was identified to me at 11.15 am by Dr Savage in the presence of his mother, Debra Strain. I then carried out certain enquiries into the circumstances leading up to Adam's death. At 2.00 pm on Wednesday, 29 November 1995 I identified the body of Adam Strain to Dr Alison Armour at the mortuary at the Royal Victoria Hospital.

S Tester

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P.T.O.

CORONERS ACT (Northern Ireland), 1959

Deposition of Witness taken on TUESDAY the 18th 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 DEBRA STRAIN

of [redacted] *(Address)*

who being sworn upon her oath, saith

Adam was born on the 4th August 1991 with dysplastic kidneys also obstruction and reflux of both ureters. He first started having surgery at three months old on the 22nd November 1991 when he had his first re-implantation of his ureters. This took place in the Ulster Hospital and on the 26th November he was then transferred to the R.B.H.S.C. because of complications. Between then and early January 1992, he had a further four re-implatations of his ureters, the end result being the left ureter had to be joined to the right and then attached to his bladder in a 'Y' shape. All this proved unsuccessful. In March 1992, because of severe oesophageal reflux he needed a fundo-plication. Also during this time and in the months and years following he had three gastrostomy tubes, two dialysis catheters and also central lines inserted. He started on peritoneal dialysis in September 1994 for thirteen hours a night, six nights a week. The last surgery that Adam had before his transplant was an orchidopexy and gastrostomy button in October 1995. He also needed to have various tubes removed and tests carried out which required anaesthesia for short periods of time, but unfortunately I cannot remember everyone of them. This takes us up to the 26th Nov 1995 when Adam was admitted to Musgrave Ward at 9pm for transplant. As he did not take anything by mouth and required 2100mls of fluid a day between midnight and 5am, he was fed approximately 900mls of water through his gastrostomy button to keep his fluid balance correct. He was taken to theatre shortly before 7am and at this point I was told surgery was expected to last between 2 & 3 hours. During the operation Adam's

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P.T.O.

doctors very kindly kept me in touch with what was going on. At 9.30am, Dr Savage told me that things were going well and that an epidural was in place. Also Mr Brown was assisting Mr Keane, but to be perfectly honest neither of these pleased me very much. In the remaining 2 and ½ hours of surgery I was told by Dr O'Connor that because Adam was quite heavy and because of adhesions caused by previous surgery, things were taking longer than expected. I was also told that Adam's bladder was enlarged and that after transplant, he would probably need to be catherized several times a day. The first time I saw Adam after surgery was at approximately 12.15pm and I was told he was just being slow to waken, but I knew straight away that there was something wrong as this had never happened to Adam before. I was then taken away to have a cup of tea and settle myself, but no one gave any indication at this point that there was anything wrong. I returned to ICU a short time later, but was not allowed in. I was then informed that there was something seriously wrong, but they could not tell me what. A short time later they took Adam for a CT Scan and about an hour later I was informed that there was very little hope. At 7pm the neurologist, Dr Webb, carried out his tests and agreed with the findings of Dr Savage and Dr Taylor. Later that night, I was made aware that Adam's postassium had risen and he needed to be dialysised. I attached him up to a dialysis machine which was brought round from Musgrave Ward. Dialysis proved unsuccessful as the fluid leaked from Adam's wound and it had to be switched off a short time later. At no time was I made aware of the problem with Adam's sodium level, I was just told Adam's condition was being treated aggressively and that everything was being done which I knew and I still believe to be true. Dr Webb returned next morning and carried out this tests again and at 12 o'clock midday Adam's respirator was switched off. As a parent and on behalf of the family circle who had Adam as the focal point of our lives for over four years, it was obviously a very emotional time. Dr Taylor, part of the medical team, described what had happened to Adam as "a one in a million thing." At this time and at the back of our minds still, this

TAKEN before me this 18th day of JUNE 19 96 ,

Coroner for the District of GREATER BELFAST

2/

CORONERS ACT (Northern Ireland), 1959

Deposition of Witness taken on TUESDAY the 18th 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 DEBRA STRAIN

of *(Address)*

who being sworn upon her oath, saith

was possibly not the way to describe what had happened to our little boy. I keep thinking and searching for an explanation. One question keeps coming to mind. It concerns Adam's sodium level mentioned in Dr Alexander's report. I would like to point out that it was commonly known that Adam had an ongoing problem with his sodium which he was being treated for and had been for the past four years. If this had any bearing on the outcome, I would like to know why more care was not taken with this, as surgery had to be prolonged for such a long period. I would just like to say that when you give a child life you never expect to have to watch that being taken away from them, but I did have to and that will be with me for the rest of my life. My son's full name was Adam Strain. He was born in Belfast on the 4th August 1991. My full name is Debra Strain and I am employed as an Accounts Clerk.

Mrs Higgins : I was unhappy about Mr Brown due to a previous surgical procedure. After surgery on the left occasion Adam looked very bloated. This was at 12.15 pm – I think the operation was over at about noon. Also he was not awake and on previous occasions he recovered from anaesthesia quickly. I produce 4 photographs showing Adam's bloated appearance before and after the operation. For his sodium problem he had been prescribed sodium bicarbonate and a 100 ml of saline into his food each day...

P.T.O.

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I did ~~my~~ look into his eyes after surgery. His health was greatly good. He was very well nourished and compared very favourably with the other children waiting for kidney transplants. On the last occasion I was with Graham to say any consultant on the morning of the operation, this had always happened promptly. The difficulty in inserting a line on the left side might be associated with scarring there from previous procedures.

Peter Stark

TAKEN before me this 18th day of JUNE 19 96 ,

Coroner for the District of GREATER BELFAST

Miss Higgins: I was unhappy about Mr Brown due to a previous surgical procedure. After surgery on the last occasion Adam looked very bloated. This was at 12.15 pm - I think the operation was over at about noon. Also he was not awake and on previous occasions he recovered from anaesthesia quickly. I produce 4 photographs showing Adam's bloated appearance before and after the operation C1. For his sodium problem he had been prescribed sodium bicarbonate and a 100 ml of saline into his feed each day. I did not look into his eyes after surgery. His health was generally good. He was very well nourished and compared with favourably with the other children waiting for kidney transplants. On the last occasion I was not spoken to by any consultant on the morning of the operation. This had always happened previously. The difficulty in inserting a line on the left side might be associated with scarring there from previous procedures.

No. 20

CORONERS ACT (Northern Ireland), 1959

Deposition of Witness taken on TUESDAY the 18TH day of JUNE 1996, 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 DR ALISON ARMOUR

of INSTITUTE OF STATE PATHOLOGY *(Address)*

who being sworn upon her oath, saith

On the instructions of HM 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 on a body identified to me as that of Adam Strain. I now produce a copy of my report marked C1. This was massive cerebral oedema and I have never come across anything of a similar degree. The cause of it in this case is extremely rare and never encountered by me previously. On a worldwide basis it would be equally rare.

Mr. Bingham: It was a complex case because of Adam's underlying condition, his previous surgery and the technical difficulty of the operation. He experienced substantial blood loss during the operation & that made his haemodynamics very difficult to manage. Adam was not a healthy child – he was a sick little boy.

139 mmol/l is within the normal range.

So far as no significant oedema of any other organ my understanding is that fluid is absorbed into the brain in preference to any other organ. I distinguish between hyponatraemia & dilutional hyponatraemia. The latter is due to fluid given. Children are more susceptible to

TAKEN before me this 18th day of JUNE 1996

M.L. Leckey.

Coroner for the District of Greater Belfast

17

No. 2(

CORONERS ACT (Northern Ireland), 1959

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

Coroner for the District of

as follows to wit:—

The Deposition of DR ALISON ARMOUR

of (Address)

who being sworn upon her oath, saith

cerebral oedema and ~~the~~ adults and so far as dilutional hyponatraemia females are more susceptible than males. The paper I referred to refers to healthy children but it is still a good reference to this condition. There was impaired cerebral perfusion as there was a suture on the ~~left~~ side and a cottaker kip on the right. 1200 mls blood loss during the operation. I do not know what problems this would have caused for the anaesthetist.

Mrs Higgins: A critical point was the fluids used by the anaesthetist to replace blood loss. At the autopsy I had 10 sets of notes relating to Adam and the clinicians statements. The suture impaired the blood flow to the brain and the cottaker kip on the right may have had a role to play. The suture had been there for some time. Dr Taylor advised in ~~at~~ the autopsy of the calculation he made to replace blood loss. Haematocrit = packed cell volume. In this case the reading could indicate a dilutional state.

P.T.O.

Dd 087535 30M 3/78 PB N Ltd Gp 36

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This was massive cerebral oedema and I have never come across anything of a similar degree. The cause of it in this case is extremely rare and never encountered by me previously. On a worldwide basis it would be equally rare.

Mr Brangham: It was a complex case because of Adam's underlying condition, his previous surgery and the technical difficulty of the operation. He experienced substantial blood loss during the operation and that made his haemodynamics very difficult to manage. Adam was not a healthy child - he was a sick little boy. 139 mml/l is within the normal range. So far as no significant oedema of any other organ my understanding is that fluid is absorbed into the brain in preference to any other organ. I distinguish between hyponatraemia and dilutional hyponatraemia. The latter is due to fluids given. Children are more susceptible to cerebral oedema than adults and so far as dilutional hyponatraemia females are more susceptible than males. The paper I referred to refers to health children but it is still a good reference to this condition. There was impaired cerebral perfusion as there was a suture on the left side and a catheter tip on the right. 1200 mls blood loss during the operation. I do not know what problems this would have caused for the anaesthetist.

Miss Higgins: A critical point was the fluids used by the anaesthetist to replace blood loss. At the autopsy I had 10 sets of notes relating to Adam and the clinicians' statements. The suture impaired the blood flow to the brain and the catheter tip on the right may have had a role to play. The suture had been there for some time. Dr Taylor advised me at the autopsy of the calculation he made to replace blood loss. Haematocrit = packed cell volume. In this case the reading could indicate he was bleeding or in a dilutional state.

CORONERS ACT (Northern Ireland), 1959

Deposition of Witness taken on TUESDAY the 18TH day of JUNE 1996, 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 DR EDWARD SUMNER
of GREAT ORMOND STREET HOSPITAL, LONDON *(Address)*
who being sworn upon his oath, saith

I am a Consultant Paediatric Anaesthetist at the Great Ormond Street Hospital for Children NHS Trust. At the request of HM Coroner for Greater Belfast Mr J L Leckey LLM, I prepared a report on the circumstances of the death of Adam Strain which I now produce marked C3

E Sumner

TAKEN before me this 18th day of JUNE 1996

M L Leckey
Coroner for the District of Greater Belfast

CORONERS ACT (Northern Ireland), 1959

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

as follows to wit:—

The Deposition of DR EDWARD SUMNER

(Address)

of

who being sworn upon his oath, saith

Blood gas should have been taken as soon as Adam was on the operation table. He was a sick child but relatively stable for hypoxaemia in Adam would be the same as in any child. I personally have not come across a similar case — it is an extremely rare case. The brain is more sensitive to oedema than other organs. The impaired blood flow ~~to~~ from the head may have been contributory. I think it is impossible to say that Adam was more susceptible than a normal healthy child. Case management is extremely difficult. 123 a low reading which would require investigation.

Mr. Bingham: 123 — should not go any lower and something would have to be done about it. All fluids given contained sodium to a greater or lesser degree. With hindsight there was a problem with venous drainage which Dr Taylor could not have known about.

P.T.O.

21

Dd. 8375933 30M 10/81 PBN Ltd. Gp. 36

Huggins: One member of the anaesthetic would see the patient in St. Ormond before surgery to take a full history could include any problem with iron deficiency. Patients are very likeable and a good source of information, if this is a highly skilled exercise and clubbing would have made the difficult. Normally we go to the right but I cannot criticise what Dr Taylor had to get a line into the upper in baby, not the groin. Turning the may have occluded the external jugular Drainage may have been impaired without having it, though you might have noted that the drainage was normal. I always a patient's head to one side. Arterial is used for blood gases and electrolytes. One - life has three lumen for volume (blood, plasma), for continuous of CVP and the third for infusion It is not interrupted. Blood gases used by a machine over the last (the would be slow - an hour perhaps). In surgery I do blood gases at the ing, the middle and the end. In this case not taken at the beginning. Length determines the frequency of doing this, hr operation - 4 gas; 4 hrs - 3. If Hb below 128 that is hyponatraemia I

e me this 18th day of June 1996,

L. Kelly Coroner for the District of Greater Belfast

CORONERS ACT (Northern Ireland), 1959

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

as follows to wit:—

The Deposition of DR EDWARD GUMNER

of (Address)

who being sworn upon h[illegible] oath, saith

~~there~~ there will be an increased risk of fluid getting into the brain. Sodium is a crucial element of our body and is crucial for the maintenance of cells. Where that is absent water moves into the cells and they swell. At 123 some oedema of the tissues could be beginning. We would know of the Arieff paper. Hyponatraemia is more difficult to diagnose ~~on~~ during anaesthesia — it can mask the signs. I believe that with our normal drainage routine Adam may have survived provided the blood did not drop below 123. You can survive with a reading of 123 if it does not fall further. I agree with Dr Annear's definition of hyponatraemia & dilutional hyponatraemia. Adam was described as polyuric — passing a lot of urine. I do not know how much he passed during the operation. This information is not routinely kept. The fluids given did not contain sufficient sodium to counteract that being lost. The need to give adequate fluid does not override the importance of sufficient sodium. The haematocrit reading together with the low

P.T.O.

Dd. 8375453 30M 10/81 PBN Ltd. Gp. 36

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sodium
indicated not enough red cells being given
Hahnatrin sufficient
and ~~low~~ sodium. All the fluids given
after dialysis may have been given to
increase central venous pressure. It may have
had the effect of causing the dilution of the
sodium in the body. Fluid balance in paediatrics
is a very controversial area with a variety
of views. With kidney transplants we give more
fluids than in other operations. When the new
kidney is perfused it is vital that sufficient
fluids are available. I got the impression that
Dr. Taylor was not believing the CVP readings
he was getting. I believe they were possibly
~~correct~~ ~~but~~ but high. I think I would have
believed them. A high CVP can mean too
much fluid has been administered. In a
child it is very desirable. That in turn
increases blood volume. Once that was
apparent the rate of infusion of fluids
could be slowed or a different fluid given.
I, I would have transfused the child with red
blood cells. Last CVP reading was taken just
before 11.30. Monitoring was continued in ICU.
Swelling can occur very quickly — perhaps
within an hour. Sometimes with relatively
small amounts of fluid. I do not look out
for swelling intra-operatively due to the drapes.
It is not so easy to determine intra-operatively.
Swelling of the brain can be independent of
swelling of the face. They may be connected.

TAKEN before me this 18th day of June 1996

Coroner for the District of Greater Belfast

No. 2(

CORONERS ACT (Northern Ireland), 1959

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

The Deposition of DR EDWARD SUMNER
of (Address)
who being sworn upon h[illegible] oath, saith

The low sodium was indicative of the hyponatraemia. Below 128 is a hyponatraemic state. A mortality rate of 3 in 10,000 is unusual.

E Sumner

Blood gas should have been taken as soon as Adam was on the operation table. He was a sick child but relative to other children on a renal transplant programme. He was relatively healthy. I believe the mechanisms for hyponatraemia in Adam would be the same as in any child. I personally have not come across a similar case - it is an extremely rare case. The brian is more sensitive to oedema than other organs. The impaired blood flow from the brain may have been contributory. I think it is impossible to say that Adam was more susceptible than a normal healthy child. Case management is extremely difficult. 123 a low reading which would require investigation.

Mr Brangham: 123 - should not got any lower and something would have to be done about it. All fluids given contained sodium to a greater or lesser degree. With hindsight there was a problem with venous drainage which Dr Taylor could not have known about.

Miss Higgins: One member of the anaesthesia team would see the parent in Gt Ormond Street before surgery to take a full history. That could include any problem with sodium deficiency. Parents are very knowledgable and a good source of information. Putting lines in is a highly skilled business and Adam's chubbiness would have made that more difficult. Normally we go to the right first but I cannot criticise what Dr Taylor did. HE had to get a line into the upper part of the body, not the groin. Turning the head may have occluded the external jugular vein. Drainage may have been impaired without one knowing it, though you might have guessed that the drainage was normal. I always have the patient's head to one side. Arterial blood is used for blood gases and electrolytes. The venous line has three lumens for giving volume (blood, plasma) for continuous measurement of CVP and for infusion of drugs. It is not interrupted. Blood gases are measured by a machine or at the lab (the latter would be slow - an hour perhaps_. In complex surgery I do blood gases at the beginning, the middle and the end. In this case they were not taken at the beginning. Length of the operation determines the frequency of doing this. In a 6 hour operation - 4 sets; 4 hours - 3. If sodium falls below 128 that is hyponatraemia and there will be an increasing risk of fluid getting into the brain. Sodium is a crucial element of our body and is crucial for the maintenance of cells. Where that is absent water moves into the cells and they swell. At 123 some oedema of the tissues could be beginning. We would know of the Arieff paper. Hyponatraemia is more difficult to diagnose during anaesthesia - it can mask the signs. I believe that with out the venous drainage problem, Adam may have survived provided the level did not drop below 123. You can survive with a reading of 123 if it does not fall further. I agree with Dr Armour's definition of hyponatraemia and dilutional hyponatraemia. Adam was described as polyuric - passing a lot of urine. I do not know how much he passed during the operation. This information is not routinely kept. The fluids given did not contain sufficient sodium to counteract that being lost. The need to give adequate fluid does not override the importance of sufficient sodium. The haematocrit reading together with the low sodium indicated not enough red cells being given and relatively insufficient sodium. All the fluids given after dialysis may have been given to increase central venous pressure. It may have had the effect of causing the dilution of the sodium in the body. Fluid balance in paediatrics is a very controversial area with a variety of views. With kidney transplants one gives more fluids than in other operations. When the new kidney is perfused it is vital that sufficient fluids are available. I got the impression that Dr Taylor was not believing the CVP readings he was getting. I believe they were probably

correct but high. I think I would have believed them. A high CVO can mean too much fluid has been administered. In a child it is very distensible. That in turn increases blood volumes. Once that was apparent the rate of infusion of fluids could be slowed as a different fluid given. Also, I would have transfused the child with red blood cells. Last CVP reading was taken just before 11.30. Monitoring was continued in ICU. Swelling can occur very quickly - perhaps within an hour. Sometimes with relatively small amounts of fluid. I do not look out for swelling intra-operatively due to the drapes. It is not so easy to determine intra-operatively. Swelling of the brain can be independent of swelling of the face. They may be connected. The low sodium was indicative of the hyponatraemia. Below 128 is a hyponatraemic state. A mortality rate of 3 in 10,000 is unusual.

No. 20

CORONERS ACT (Northern Ireland), 1959

Deposition of Witness taken on TUESDAY the 18TH day of JUNE 1996, 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 DR JOHN ALEXANDER

of BELFAST CITY HOSPITAL *(Address)*

who being sworn upon his oath, saith

I am a Consultant Anaesthetist at the Belfast City Hospital. At the request of HM Coroner for Greater Belfast Mr J L Leckey LLM, I prepared a report on the circumstances of the death of Adam Strain which I now produce marked C4.

Mr. Brangham: There was a fluid deficit between 5am and 9a.m. That would be a normal precaution for any child coming to surgery. During surgery it would have been impossible for the anaesthetist to measure urinary output. The blood loss was 2/3rs of his volume which was very severe. The fact that the haemoglobin was normal at the end of the procedure would indicate that blood loss had been replaced. A reading of 14 mm of pressure was abnormally high. That would have made me think there was something wrong with the transducer. If it had started low and gone up that is the response that would have been wanted. I am not convinced that tying off the internal jugular vein effected drainage from the vein. If it had been effected there was no way the anaesthetist would have known. I would not entirely concur with Dr Brenner's view that a compromised renal function is not a factor in the onset of hypernatraemia.

TAKEN before me this 18th day of JUNE 1996

Coroner for the District of Greater Belfast

26

No. 2

CORONERS ACT (Northern Ireland), 1959

Deposition of Witness taken on the da
of 19 , at inquest touching the death o
, before me
Coroner for the District of
as follows to wit:—

The Deposition of DR JOHN ALEXANDER
of
(Address,
who being sworn upon h[illegible] oath, saith

Miss Higgins: The USA practice regarding infusion is not followed here. Adam's case is not an identical scenario to that in Arch paper. There the children had evidence of hypoxia and there is no evidence of that in Adam's case. With the benefit of hindsight saline loads in children with a compromised function should be monitored. That would not have been the former practice. I agree that a reading of 123 suggests that something should be done but it would not have been particularly alarmed. I would have been concerned with the Haemoglobin reading. That would have been an indication for giving blood transfusion. I would have taken a further blood gas and haemoglobin reading. If I thought a hangover was giving a faulty reading I would get another one. I think it was unlikely that a 1000 ml infusion of saline would raise the urine volume to 17 ml. I do not know what volume would achieve that I do not believe that the problem could be recognised until after the operation. I would be

P.T.O.

AS - PSNI 094-192-870

very concerned if the sodium level dropped to 120 or below. I do not know if Adam's death could be averted. Every drop below 123 increases the risk. I would agree that in Arnoff's paper and in Adam's case there was a high infusion of fluids.

J Alexander

TAKEN before me this 18th day of June 1996.

Mark Lanney Coroner for the District of Greater Belfast

Mr Brangham: There was a fluid deficit between 5.00 am and 7.00 am. That would be a normal precaution for any child coming to surgery. During surgery it would have been impossible for the anaesthetist to measure urinary output. The blood loss was 2/3's of his volume which was very serious. The fact that the haemoglobin was normal at the end of the procedure would indicate that blood loss had been replaced. A reading of 17mm re pressure was abnormally high. That would have made me think there was something wring with the transducer. If it had started low and gone up that is the response that would have been wanted. I am not convinced that tying off the internal jugular vein effected drainage from the vein. If it had been effected there was no way the anaesthetist would have known. I would not entirely concur with Dr Sumner's view that a compromised renal function is not a factor in the onset of hyponatraemia.

Miss Higgins: The USA practice regarding infusion is not followed here. Adam's case is not an identical scenario to that in Arieff's paper. There the children had evidence of hypoxia and there is no evidence of that in Adam's case. With the benefit of hindsight sodium levels in children with a compromised renal function should be monitored. That would not have been the former practice. I agree that a reading of 123 suggests that something should be done but I would not have been particularly alarmed. I would have been concerned with the Haematocrit reading. That would have been an indication for giving blood transfusions. I would have taken a further blood gas and haematocrit readings. If I thought a transducer was giving a faulty reading I would get another one. I think it was unlikely that a 1000 ml infusion of saline would raise the venous pressure to 17mm. I do not know what volume would achieve that. I do not believe that the problem could be recognized until after the operations. I would be very concerned if the sodium level dropped to 120 or below. I do not know if Adam's death could be averted. Every drop below 123 increases the risk. I would agree that in Arieff's paper and in Adam's case there was a high infusion of fluids.

CORONERS ACT (Northern Ireland), 1959

Deposition of Witness taken on TUESDAY the 18th 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 ~~DOCTOR~~ MR. D F KEANE (Consultant Urologist)

of c/o BCH, Dept of Urology *(Address)*

who being sworn upon h oath, saith

I was asked to transplant this 4 year old boy on Monday 27 November 1995. The operation started at 7.30am and was technically very difficult because of previous surgery that this young boy had. However, despite the technical difficulties the kidney was successfully put into the child and perfused quite well initially and started to produce urine. At the end of the procedure it was obvious that the kidney was not perfusing as well as it had initially done, but this is by no means unusual in renal transplantation. The whole operative procedure took about three hours. I was informed later on that day that the child had severe cerebral oedema and that he was probably brain dead. In summary, therefore, the operation was difficult, but a successful result was achieved at the end of the procedure. Monitoring of urine during a transplant procedure is never done. Mrs Higgins: The operation would have started between 7.15 and 8.00 a.m. I do not believe that surgery of this nature should be undertaken if possible at 2/3 or 4 a.m. In this case the kidney being transplanted had been removed within a normal time before surgery. It was sometime after the end of surgery that the problem with Adam was noticed. The blood loss of 1200 cc was in all blood but contained fluid as well. I was not aware of Arriff's paper. In the light of Adam's experience the factors in that ~~Report~~ Paper

P.T.O.

would be carefully considered in future surgery of a similar nature.

Patrick J Kelly

TAKEN before me this 18th day of JUNE 19 96,

Coroner for the District of GREATER BELFAST

Monitoring of urine during a transplant procedure is never done.

Miss Higgins:- The operation would have started between 7.15 and 8.00 am. I do not believe that surgery of that nature should be undertaken at 2/3 or 4 am if possible. In this case the kidney being transplanted had been removed within a normal time before surgery. It was sometime after the end of surgery that the problem with Adam was noticed. The blood loss of 1200 cc was not all blood but contained fluid as well. I was not aware of Arieff's paper. In the light of Adam's experience the factors in that paper would be carefully considered in future surgery of a similar nature.

CORONERS ACT (Northern Ireland), 1959

Deposition of Witness taken on FRIDAY the 21st 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 MAURICE SAVAGE

of c/o R.B.H.S.C. *(Address)*

who being sworn upon his oath, saith

Adam Strain was a patient with chronic renal failure and polyuria. He developed problems with recurrent urinary infections in infancy and was under the care of Mr Stephen Brown, Consultant Paediatric Surgeon. He required multiple urological operations for vesico ureteric reflux and a Fundal Plication to correct a hiatal hernia. As a result of infection and reflux his kidneys were damaged and deteriorated to the point where peritoneal dislysis was commenced in 1994. He was then placed on call for a renal transplant. He required multiple medications with Calcium Carbonate, Keflex, Iron, One-Alpha Vitamin D, Erythropoietin and Sodium Bicarbonate and night time gastrostomy tube feeding. The medications and tube feeds were to ensure good nutrition and to prevent renal anaemia and bone disease. He was a well nourished, well grown boy with height near the 50th centile and weight at the 90th centile for his age. His most recent acute illness was with a gastrostomy exit site infection in July 1995. On 26th November we had an offer of a kidney from the UK Transplant Service. He was admitted to Musgrave Ward RBHSC for pre-operative assessment. His serum electrolytes, haemoglobin and coagulation were satisfactory. H.B.10.5g/dl, Na 139, K 3.6, Urea 16.8, Ca.2.54, Albumin 40, Prothrombin time 12.3. His chest was clear on examination. B.P. 108/56. He was apyrexial. There were no signs of infection. His night gastrostomy feeds are normally 1.5l of Nutrizon. On anaesthetic advice this was changed to clear fluid which was stopped two hours pre op. This meant he had 900mls of ~~Dioralyte~~ N/S Saline Dextrose overnight. His peritoneal dialysis was performed as usual - 750ml fluid volume 1.36% Dextrose solution

P.T.O.

He was given 8 cycles before going to Theatre at 7am. My contact with Theatre during the procedure indicated no major difficulties with cardiovascular status or anaesthesia. Surgery was complex, but successful, organ transplantation achieved with acceptably matched kidney from a 16 year old donor. Post-operatively Adam failed to breathe spontaneously. On examination he had dilated pupils and bilateral papilloedema. A chest x-ray showed pulmonary oedema and an emergency CAT brain scan confirmed cerebral oedema and herniation and compression of the brain stem. Neurological testing by Dr David Webb on the evening of 27/11/95 and the morning of the 28/11/95 confirmed brain death. Deborah Strain, the mother and the immediate family were informed of the complications and prognosis regularly throughout these events. Death was certified shortly after 9am on 28th November. With the consent and in the presence of the family ventilatory support was withdrawn at 11.30am while Adam was being nursed by his mother. He did need sodium in his feeds but his sodium was well controlled. His mother's care of him was meticulous and his health was due to her meticulous care. I believe the speedy changed electrolytes is very significant in that the body copes with it less well.

Higgins: After 1994 Adam was under the care of Mr. Sorkin. He had a potential for a low sodium which was being managed. Adam never had their symptoms which disappeared because of his illness. The majority of children with renal failure have similar problems concerning electrolyte levels. ~~They would be monitored as it is UK standard practice to measure them during surgery.~~ Since Adam's death they would be measured more frequently. I have discovered that in the UK there have been 9 other deaths

TAKEN before me this 21 day of JUNE 19 96 ,

Coroner for the District of GREATER BELFAST

No. :

CORONERS ACT (Northern Ireland), 1959

Deposition of Witness taken on FRIDAY the 21 da
of JUNE 1996. , at inquest touching the death
, before me MR J L LECKE
Coroner for the District of GREATER BELFA
as follows to wit:—

The Deposition of MAURICE SAVAGE
of
(Address
who being sworn upon his oath, saith

from an apparently similar cause though the
have not been published. Any level below
135° is hypernatraemia but there is a lower
figure at which it becomes dangerous. A level
below 120 needs urgent action. At 128 action
needs to be taken to redress the balance.
However, the patient could be perfectly well.
Electrolytes could not be checked first thing in
the morning as venous access could not be
obtained. Standard practice would be to
keep electrolyte levels near the start of surgery
but it is essentially a matter for clinical
judgment. I was not aware of the 9.32 reading.
I believe a child in renal failure is at
greater risk of developing sodium imbalance. I accept
the cause of death given by the pathologist.
Mr. Bingham is / had known Adam since he was
a baby. He had to have the operation to live
any length of time and to have a normal life.
We discussed the operation in detail with his
mother the day before. Also, I discussed it with
Dr. Taylor. The operation had been put back to
the following morning. His overnight feeding was

discussed in detail and he would have been aware what the normal regime would have been. 900 ml area or we had to switch from tube feeding to intra-venous feeding two hours before the operation. I was satisfied the anaesthetic staff had all the relevant information. The information about the 9 otter deaths was held to be verbally taken — it was not published. All the fluids given to Adam during the operation contained sodium. One cannot pick a figure for determining hyponatraemia — it is a matter for clinical judgment which will be influenced by the speed of change. The lab would take about an hour to do an electrolyte analysis. Miss Higgins: With the benefit of hindsight his sodium became too low. A lab analysis is more accurate than the blood/gas machine. I personally never use that machine as I have no reason to do so.

J Maurice Savage

TAKEN before me this 21st day of June 1996.

Coroner for the District of Greater Belfast

He did need sodium in his feeds but his sodium was well controlled. His mother's care of him was meticulous and his health was due to her meticulous care. I believe the speedy change of electrolytes is very significant in that the body copes with it less well.

Miss Higgins:- After 1994 Adam was under the care of Mr Boston. He had a potential for a low sodium which was being managed. Adam never had thirst symptoms which disappeared because of his illness. The majority of children with renal failure have similar problems concerning electrolyte levels. Since Adam's death these would be measured more frequently. I have discovered that in the UK there have been 9 other deaths from an apparently similar cause though these have not been published. Any level below 135 is hyponatraemia but there is a lower figure at which it becomes dangerous. A level below 120 needs urgent action. At 128 action need to be taken to redress the balance. However, the patient could be perfectly well. Electrolytes could not be checked first thing in the morning as venous access could not be obtained. Standard practice would be to test electrolyte levels near the start of surgery but it is essentially a matter for clinical judgment. I was not aware of the 9.32 reading. I believe a child in renal failure is at greater risk of developing sodium imbalance. I accept the cause of death given by the pathologist.

Mr Brangham: I had known Adam since he was a baby. He had to have the operation to live any length of time and to have a normal life. We discussed the operation in detail with his mother the day before. Also, I discussed it with Dr Taylor. The operation had been put back to the following morning. His overnight feeding was discussed in detail and he would have been aware what the normal regime would have been. 900 ml arose as we had to switch from tube feeding to intra-venous feeding two hours before the operation. I was satisfied the anaesthetic staff had all the relevant information. The information about the 9 other deaths was told to me verbally later - it was not published. All the fluids given to Adam during the operation contained sodium. One cannot pick a figure for determining hyponatraemia - it is a matter for clinical judgment which will be influenced by the speed of change. The lab would take about an hour to do an electrolyte analysis.

Miss Higgins: With the benefit of hindsight is sodium became too low. A lab analysis is more accurate than the blood/gas machine. I personally never use that machine as I have no reason to do so.

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

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 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 21 day of JUNE 19 96,

Mark Cowley. Coroner for the District of GREATER BELFAST

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:–

2/

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,

P35.

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 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 21 day of JUNE 19 96 ,

Coroner for the District of GREATER BELFAST

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 *(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

'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 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 is my practice and teaching that fluids must be carefully calculated in realtion 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

TAKEN before me this 21 day of JUNE 19 96 ,

Coroner for the District of GREATER BELFAST

CORONERS ACT (Northern Ireland), 1959

Deposition of Witness taken on FRIDAY the 21 day of JUNE 19 96, at inquest touching the death of 4/ 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

(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.

equipment log-books and printed records of actual monitoring measurments. If 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 quantitites 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 21 day of JUNE 19 96

Coroner for the District of GREATER BELFAST

CORONERS ACT (Northern Ireland), 1959

Deposition of Witness taken on FRIDAY the 21 day

of JUNE 19 96, at inquest touching the death of

5/ 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 (Address)

who being sworn upon h[illegible] 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, had 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 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 back. 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.

38

managed successfully. There was no reason to believe there would have been a change in electrolytes between 11 p.m. and 6.45 a.m. Nothing in that period happened to change that. Adam was the only child with polycystic renal failure I have encountered 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. There was Friday duct work leading to a re-assessment of the fluids given. This made us think we had underestimated fluid & we gave a fluid bolus at 9.32. I checked CVP at 9.30 a.m. (about 7.30 a.m.) or soon as I had inserted the line. 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 .a.m. We were considering having 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 rt subclavian vein — I do not know if it had any effect on drainage. I cannot explain the mercury reading of 17 but

TAKEN before me this 21 day of JUNE 19 96,

Coroner for the District of GREATER BELFAST

No. 2

CORONERS ACT (Northern Ireland), 1959

Deposition of Witness taken on FRIDAY the 21 da
of JUNE 19 96, at inquest touching the death c
, before me MR J L LECKEY
Coroner for the District of GREATER BELFA

as follows to wit:—

The Deposition of R.H. TAYLOR

of

(*Address*)

who being sworn upon his oath, saith

I agree with the views expressed by Dr Alexander.

Withdrawn 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 blood/gas 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 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 everything had gone to plan when the clamps were released surgery would have completed soon afterwards. The fluids I gave were isotonic — the same potential as plasma which should

AS - PSNI

094-192-891

have munched Hope that Adam formerly
recorded. I produce as further statement CS.

TAKEN before me this 28th day of June 1996,
Coroner for the District of Greater Belfast

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.

This report has been prepared by me, Dr John Alexander, on the instructions of Mr John L Leckey LL.M., HM Coroner for the District of Greater Belfast. I have studied the relevant case notes and anaesthetic record.

Re: **ADAM STRAIN, DECEASED**

This little boy suffered from congenital vesico-ureteric reflux and dysplastic kidneys and had had multiple surgical operations in the past, many under general anaesthesia, and all apparently uneventful as far as the anaesthesia was concerned. At the time of admission on the 26th November 1995 he was in renal failure with a high volume of dilute urine from his own (native) kidneys. His renal failure was being treated by Continuous Ambulatory Peritoneal Dialysis (CAPD) and feeding difficulties had been overcome by fashioning a feeding gastrostomy.

He was 4 years 3 months of age, weighed 21 kg, and was well nourished. Relevant blood tests that evening were haemoglobin 10.5 g/dl, packed cell volume 0.32, sodium 139 millimoles per litre (mmol/l), potassium 3.6 mmol/l, albumin 40 mmol/l, urea 16.8 mmol/l and creatinine 702 μmol/l. The latter two results are very high, an expression of his renal failure, the remainder within normal limits. He was given 952 ml 'clear fluid', presumably water, overnight, into his gastrostomy, and this was stopped at 0500 on the 27th. The child was taken to the operating theatre at 0700 for a renal transplant.

Anaesthesia was induced at 0700 in the standard manner and the child intubated and artificially ventilated. Venous access was secured, a triple-lumen central venous pressure catheter inserted into the right subclavian vein and a fine catheter into the right radial artery to continuously monitor arterial blood pressure. The child's estimated blood volume was 1600 ml, estimated fluid deficit 300 ml and calculated intraoperative maintenance 200 ml/hour. Infact a great deal more fluid was infused, which included 1500 ml of one fifth isotonic saline in 4% dextrose, 500 ml Hartmanns solution, and eventually 800 ml of Human Plasma Protein Fraction and 2 units of packed red cells to replace a blood loss during the operation of about 1200 ml.

The operation proved to be technically difficult and took 4 hours to complete. During that time the heart rate decreased from 140 to 90 beats per minute, the systolic blood pressure increased from 90 to 120 mmHg and arterial blood saturation with oxygen remained consistently at 99 - 100 %. There were no dramatic changes and no evidence of either hypoxia or hypotension, as documented by Dr Taylor's meticulous records, and confirmed by the computerised print-out obtained at the end of the operation. Central venous pressure remained very high throughout the procedure; this may have been partly due to a technical problem with the pressure transducer but was also partly deliberate, since releasing the clamps on a transplanted near-adult sized kidney in a child can divert most of the cardiac output into the new organ with a dramatic fall in blood pressure; a high venous pressure will encourage a high cardiac output and avoid this problem.

A 21 kg child has an extracellular fluid volume of about 5 litres. This is made up of the blood volume inside the intravascular space (red cells and plasma) and the interstitial fluid which lies outside the vascular space and also outside the cells. Infused fluids will distribute themselves through the intravascular and interstitial spaces. A simple calculation reveals that if 1500 ml 1/5 isotonic or 'normal' saline is infused into a child of this size, plasma (or serum) sodium will fall to about 120 mmol/l. Since it takes some time for infused fluids to leave the vascular compartment,

serum (or plasma) sodium is likely to be even lower than this and the situation may be made worse by increased levels of antiduretic hormone produced during anaesthesia which will cause water retention by the kidneys. There is very little firm information available concerning dilutional hyponatraemia (low serum sodium) in children, either in standard textbooks or in the recent literature, although the condition is well recognised in neonates and in adults who have certain operations which result in an excess of water entering the circulation. Arieff and colleagues published a paper entitled "Hyopnatraemia and death or permanent brain damage in healthy children" (BMJ 1992; 304: 1218-22) which is informative. These workers described how, after hypotonic fluid administration, serum sodium can fall to levels around 115 mmol/l and lead to vague non-specific symptoms and then an explosive onset of respiratory arrest, cerebral oedema and coma. They also discuss the reasons why a child's brain has less room than an adult's to expand inside a rigid skull and suggest that developing brain cells are less able to protect themselves. One might speculate as to whether a child suffering from chronic renal failure could have increased vulnerability. In the discussion Arieff et al states: "These cases show that generally healthy children with symptomatic hyponatraemia (101-123 mmol/l) can abruptly develop respiratory arrest and either die or develop permanent brain damage". Of the 16 cases they described, 10 died and the others suffered permanent brain damage. A copy of this paper is attached.

At the end of the procedure, Adam was apnoeic and had widely dilated pupils. He was transferred to the intensive care unit. Serum sodium was 119 mmol/l and did not rise above 125 mmol/l in the next 20 hours. CT scan of the brain showed cerebral oedema and lung oedema was also evident. Tests for brain stem function were negative and active therapy was discontinued on the morning of the 28th November.

SUMMARY The complex metabolic and fluid requirements of this child having major surgery led to the administration of a large volume of hypotonic (0.18%) saline which produced a dilutional hyponatraemia and subsequent cerebral oedema. The operation was difficult and prolonged and the problem could not be recognised until the surgery was completed. At no time during the procedure was there any suggestion of hypoxia nor is there the slightest indication of a malfunction in the anaesthesia apparatus. Dr Taylor is to be commended on the detailed notes and records he kept throughout the anaesthetic.

26/6/96 - Form 21 to Bangor Reg

Form 22

CORONERS ACT (NORTHERN IRELAND) 1959

VERDICT ON INQUEST

On an inquest taken for our Sovereign Lady the Queen, at COURTHOUSE CRUMLIN ROAD in the County Court Division of BELFAST on TUESDAY the 18TH day of JUNE 1996, [and by adjournment on FRIDAY the 21ST day of JUNE 1996] before me MR J L LECKEY HM Coroner for the district of GREATER BELFAST touching the death of ADAM STRAIN to inquire how, when and where the said ADAM STRAIN came to his death, the following matters were found:

1. Name and surname of deceased: ADAM STRAIN

2. Sex: MALE

3. Date of Death: 28TH NOVEMBER 1995

4. Place of Death: ROYAL BELFAST HOSPITAL FOR SICK CHILDREN

5. Usual address (if different from place of death): 20 FIRMOUNT CRESCENT, HOLYWOOD

6. Marital status: SINGLE

7. Date and place of birth: 4TH AUGUST 1991 BELFAST

8. Occupation: SON OF DEBRA STRAIN - ACCOUNTS CLERK

9. Maiden surname: N/A

10. 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)

Findings:

The onset of cerebral oedema was caused by the acute onset of hyponatraemia from the excess administration of fluids containing only very small amounts of sodium and this was exacerbated by blood loss and possibly the overnight dialysis and the obstruction of the venous drainage to the head.

Date: 21ST JUNE 1996

Signed:
Coroner for GREATER BELFAST

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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

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.

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

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.

Larynx: There was ulceration of the mucosa, in keeping with intubation.

Liver: There was no evidence of cyst formation within the portal tract. There were scattered foci of clear cell change.

Kidney: There was widespread scarring and cyst formation, interstitial fibrosis and chronic inflammation. There was widespread glomerulo-sclerosis and the arterioles were thickened.

Transplanted Kidney: There was complete infarction.

Spleen: There was congestion of the red pulp.

Lymph Node: Normal.

(The above slides were seen by Professor J. Berry, Consultant Paediatric Pathologist).

Brain: There was massive cerebral oedema of the cortex and white matter. There was no evidence of terminal hypoxia. There was no evidence of myelinolysis.

Spinal Cord: No specific pathological features were noted.

(The brain, spinal cord and histological slides were seen by Dr. M. Mirakhur, Consultant Neuropathologist)

COMMENTARY:

This little boy with a past medical history of polyuric renal failure, numerous hospital admissions and operations was admitted to hospital one evening for a renal transplant operation. He was fed via a gastrostomy and ate nothing by mouth. Usually he would receive 1,500 mls. a night and 900 mls. during the day. That night investigations included blood pressure 108/56, sodium 139 mmol/l and haemoglobin 10.5 g/dl. Overnight he was given 900 mls. dioralyte (4% dextrose 0.18% saline) and peritoneal dialysis was performed as usual. He went to theatre the next morning.

General anaesthesia was induced. Intravenous access was difficult and four attempts were made to pass a central venous pressure catheter before it was successfully passed into the right subclavian vein. A lumbar epidural was also sited with .25% bupivacaine and fentanyl. An initial CVP reading was taken at 17 mm.Hg. and intravenous fluids were given of 3 x 500 ml. bags of dextrose saline (4% and .18%). The operation itself was technically difficult due to the previous surgical procedures and there was an increased blood loss calculated to be approximately 1,200 mls. This was replaced by intravenous fluids of 500 mls. of Hartman's, 1,000 mls. HPPF and 500 mls. of packed cells. At 9.32 a.m. a blood gas analysis revealed a sodium of 123 mmol/l (normal 135-145) and a low haematocrit. During the operation the CVP increased to 20-21 mm.Hg., the haemoglobin fell to 6.1 g/dl., the systolic blood pressure rose to 150 mm.Hg. and the pulse gradually fell but rose steadily from 10.15 a.m. onwards. When the procedure was completed and the neuromuscular block was reversed this little boy did not wake up. A CT scan of the brain revealed gross cerebral oedema. Brain stem function tests were carried out and he was declared dead a little over 26 hours from the start of the operation.

The autopsy revealed gross cerebral oedema. The fixed weight of the brain at postmortem was 1,680 gms., the average weight for a boy of this age being 1,300 gms and the average weight of a man's brain being 1,450 gms. It was the effects of this massive swelling of the brain which caused his death. There was no significant oedema of any other organ.

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.

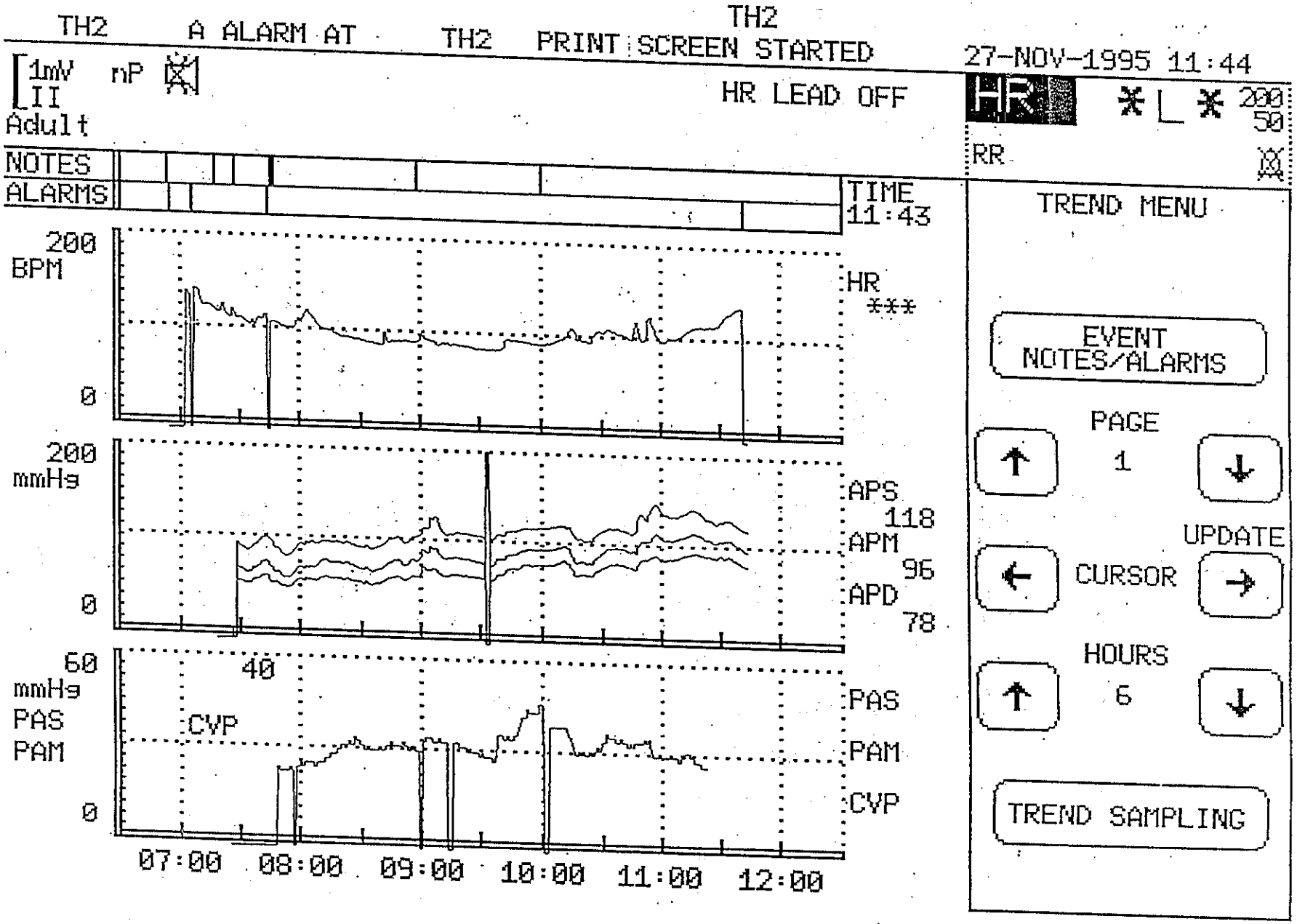
The autopsy also revealed changes in the kidneys, in keeping with chronic renal failure and total infarction of the transplanted kidney. These played no part in the fatal outcome.

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

Adam Strum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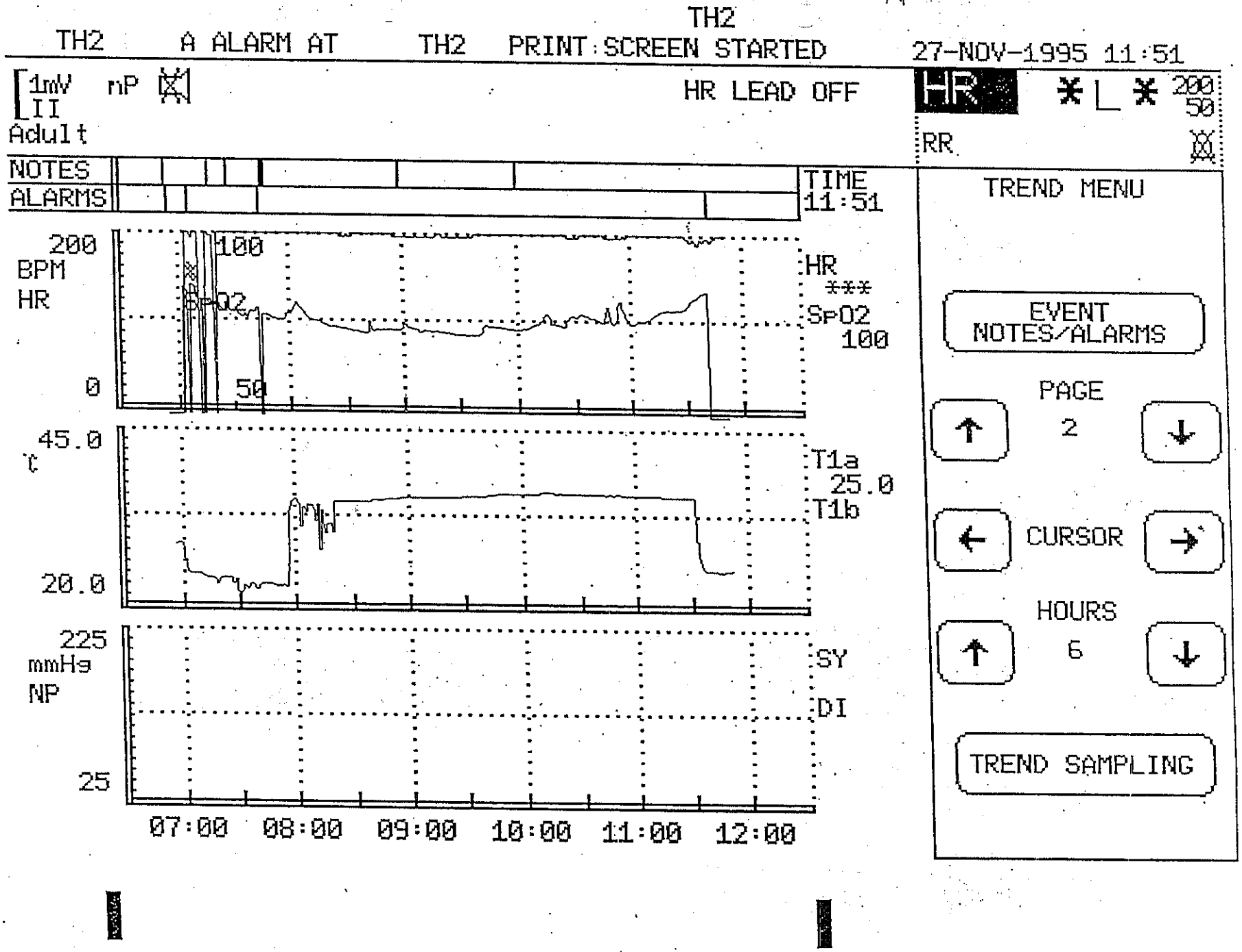
Adam Strum.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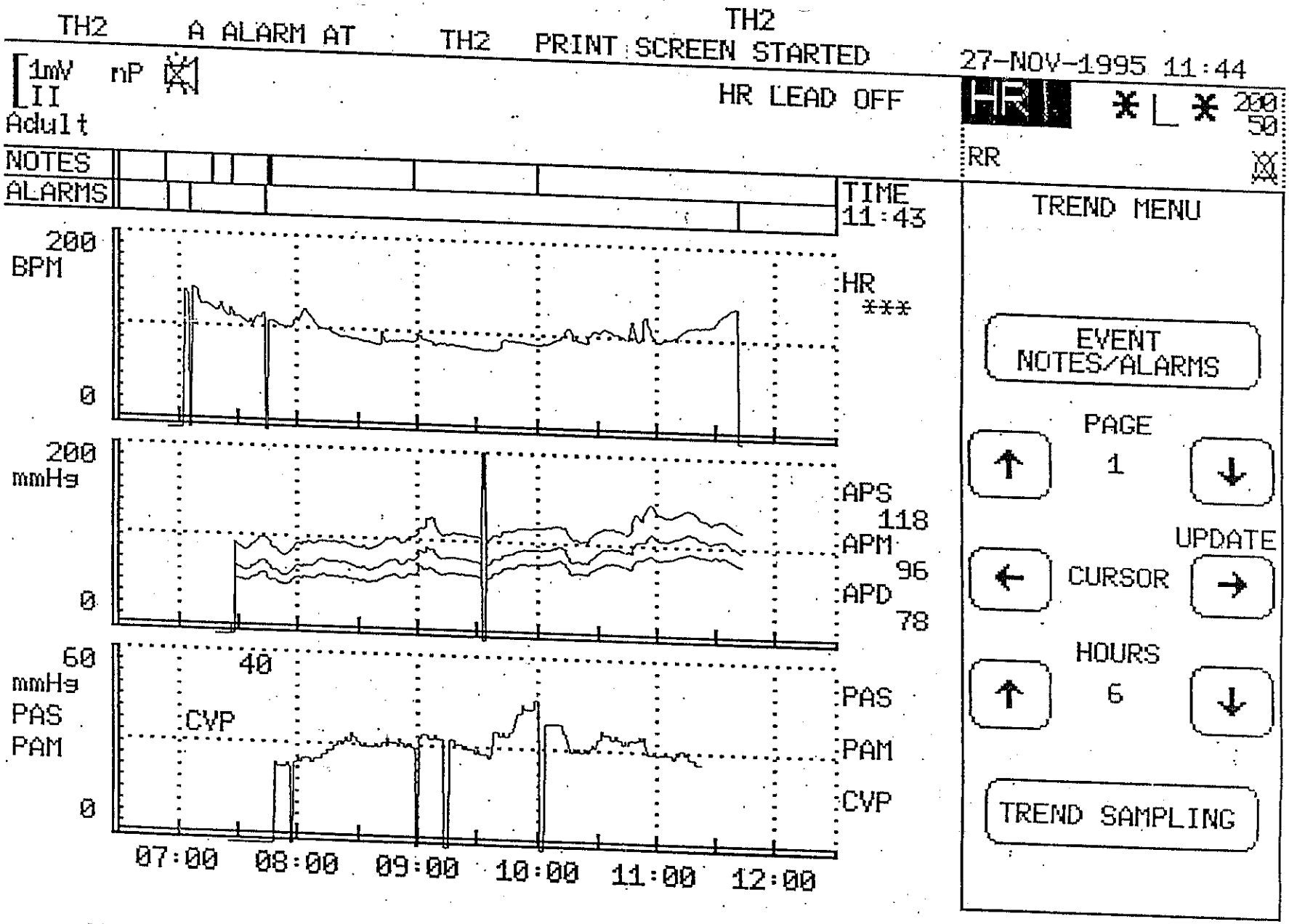
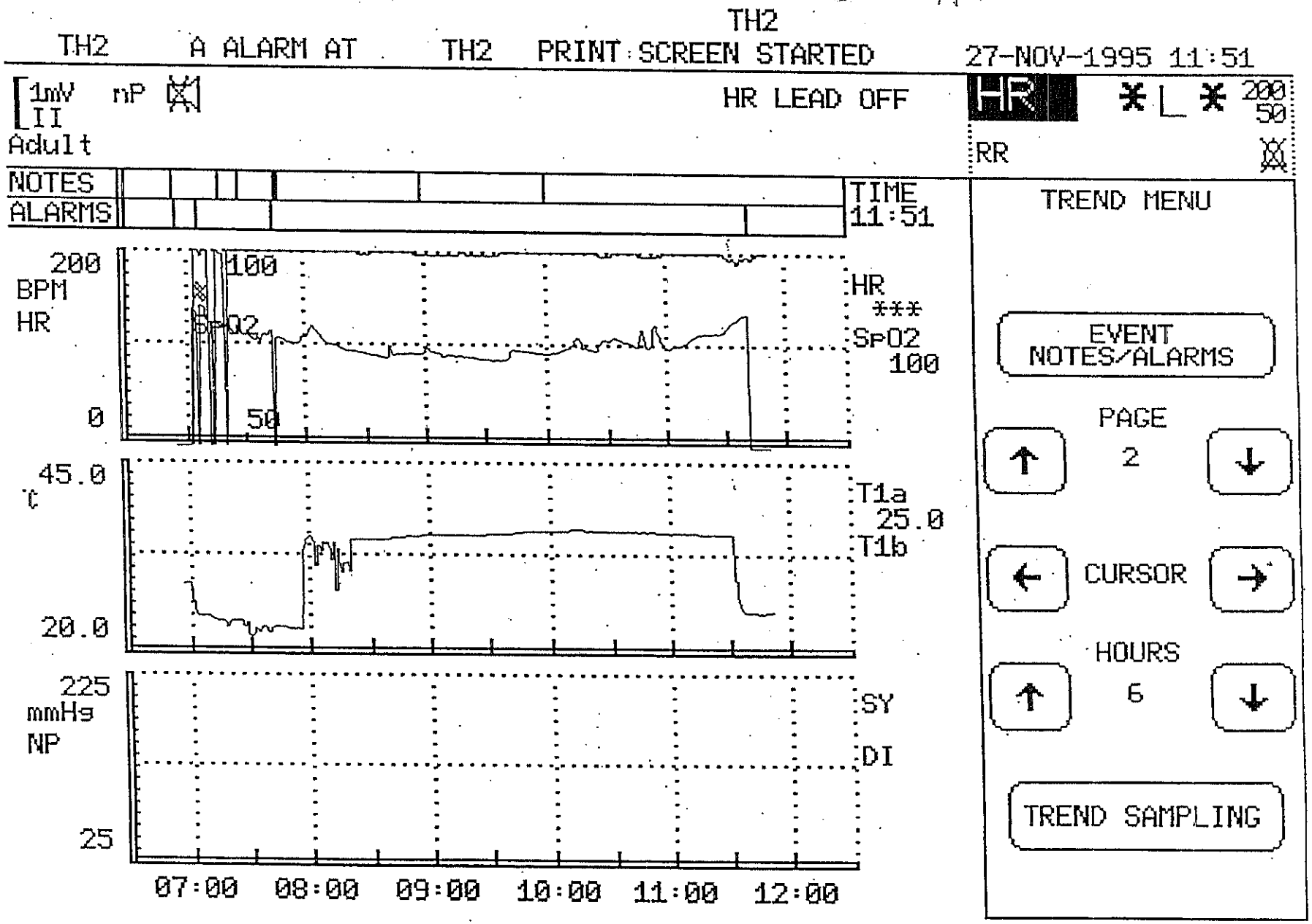
Adam Strum



AS - PSNI

Adam Simm.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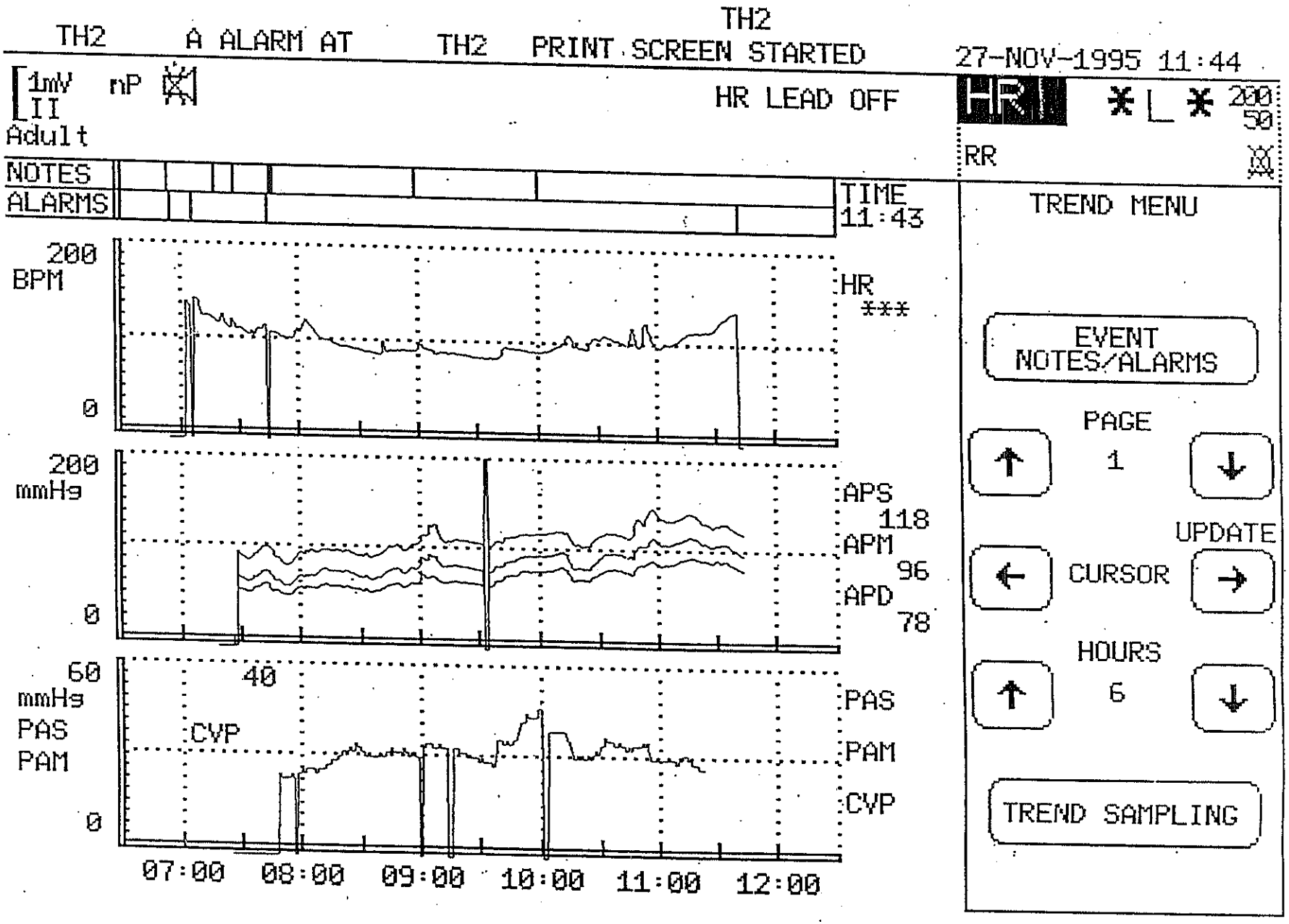
Adam Stram



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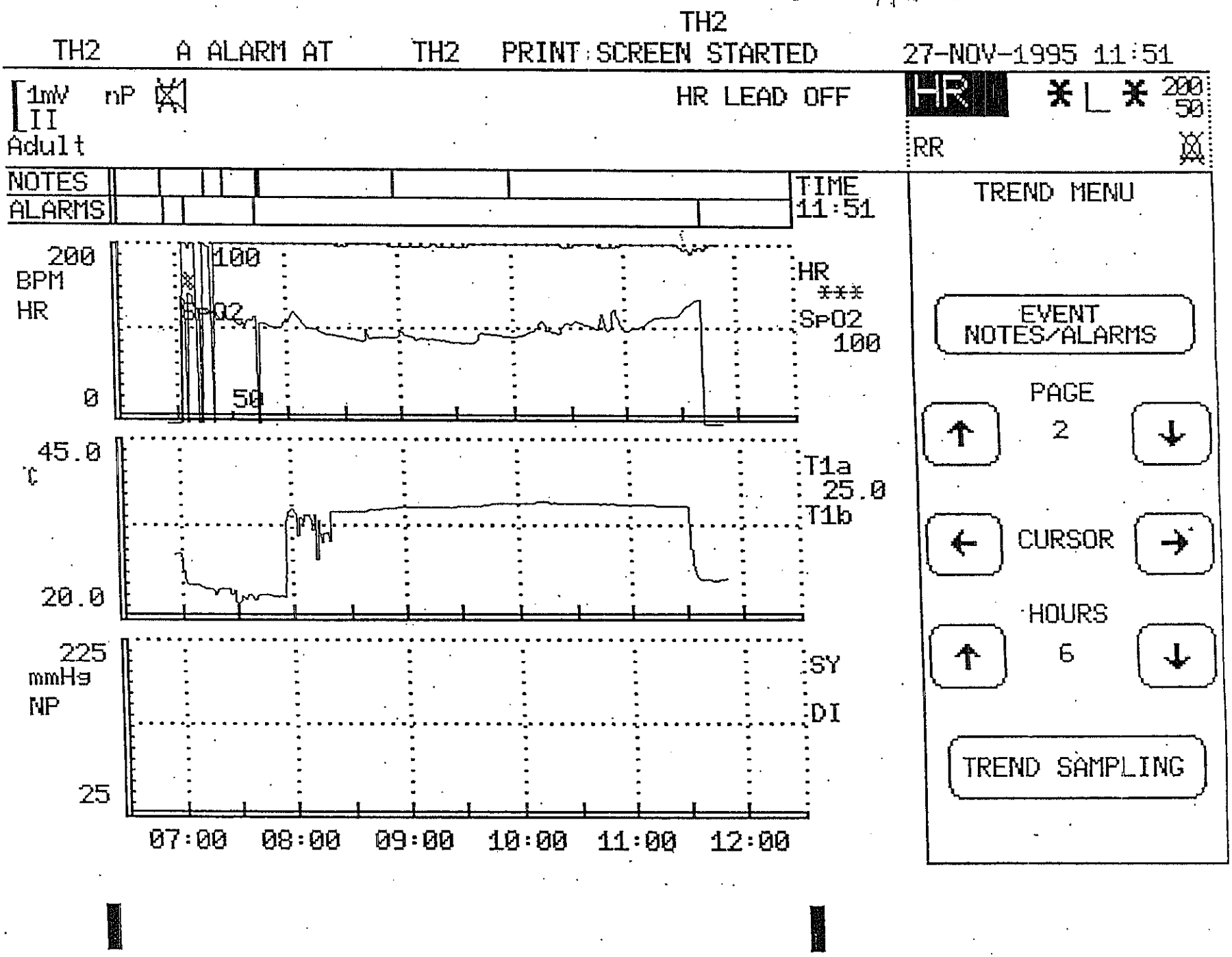
Adam S. ...



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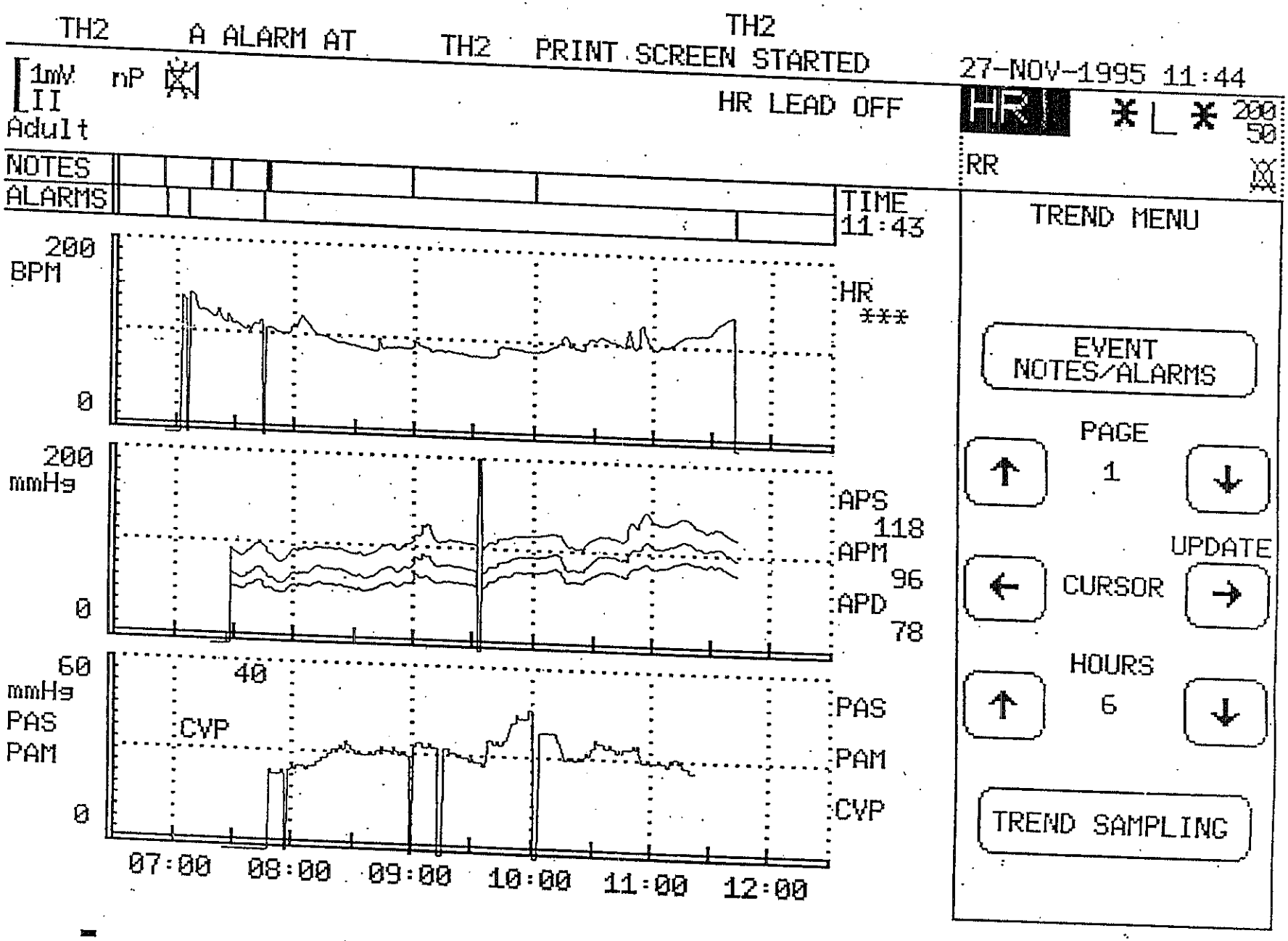

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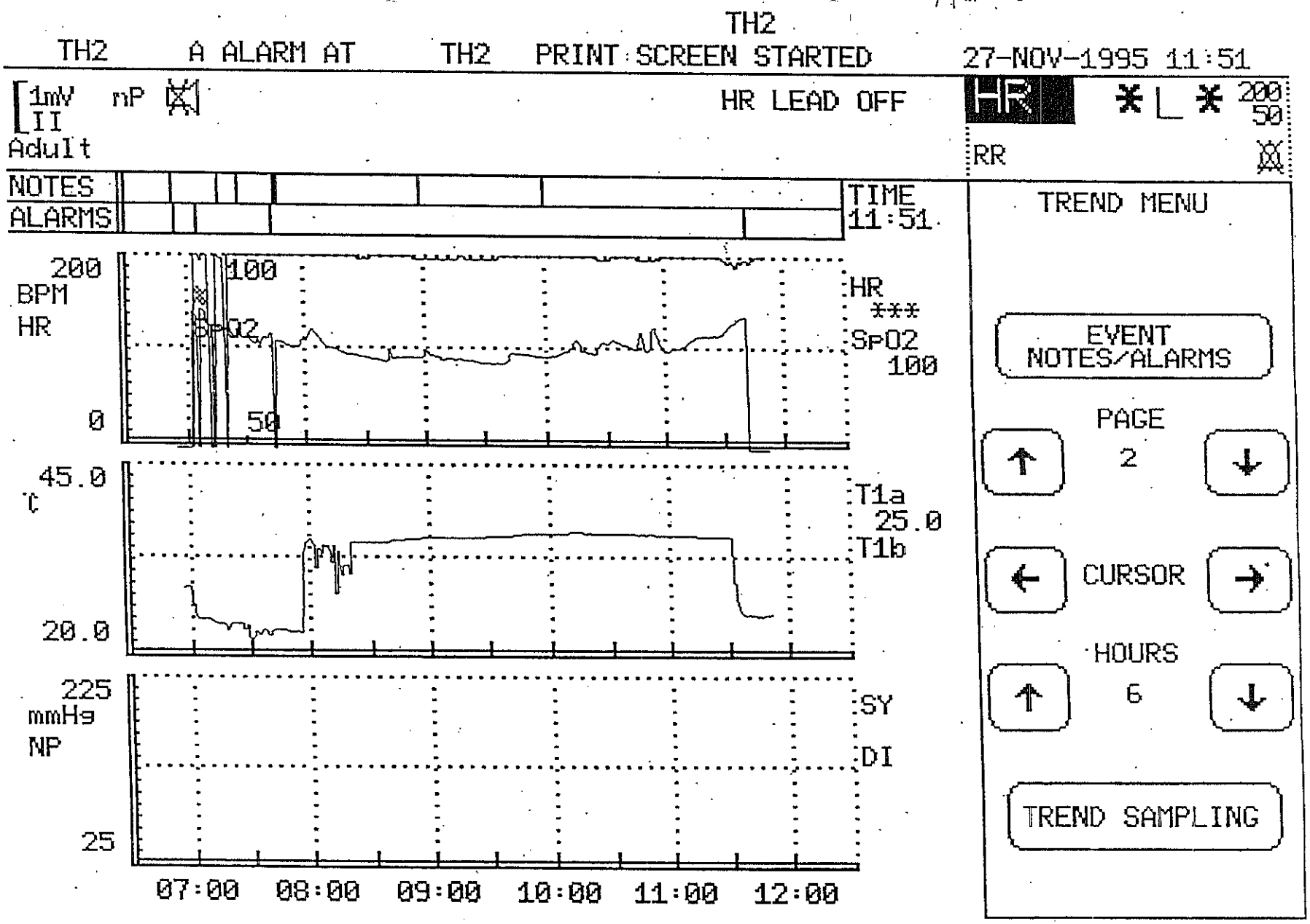

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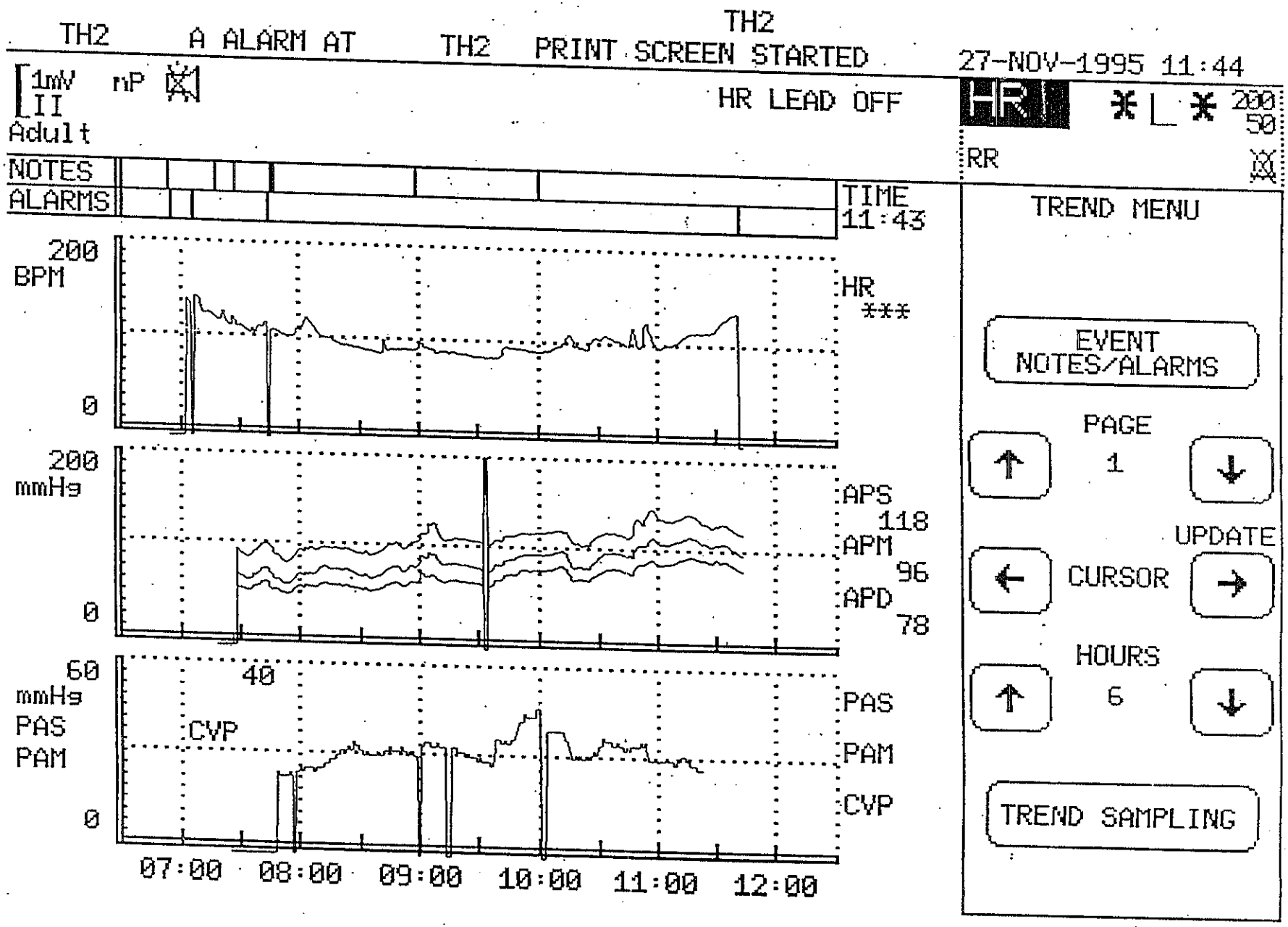

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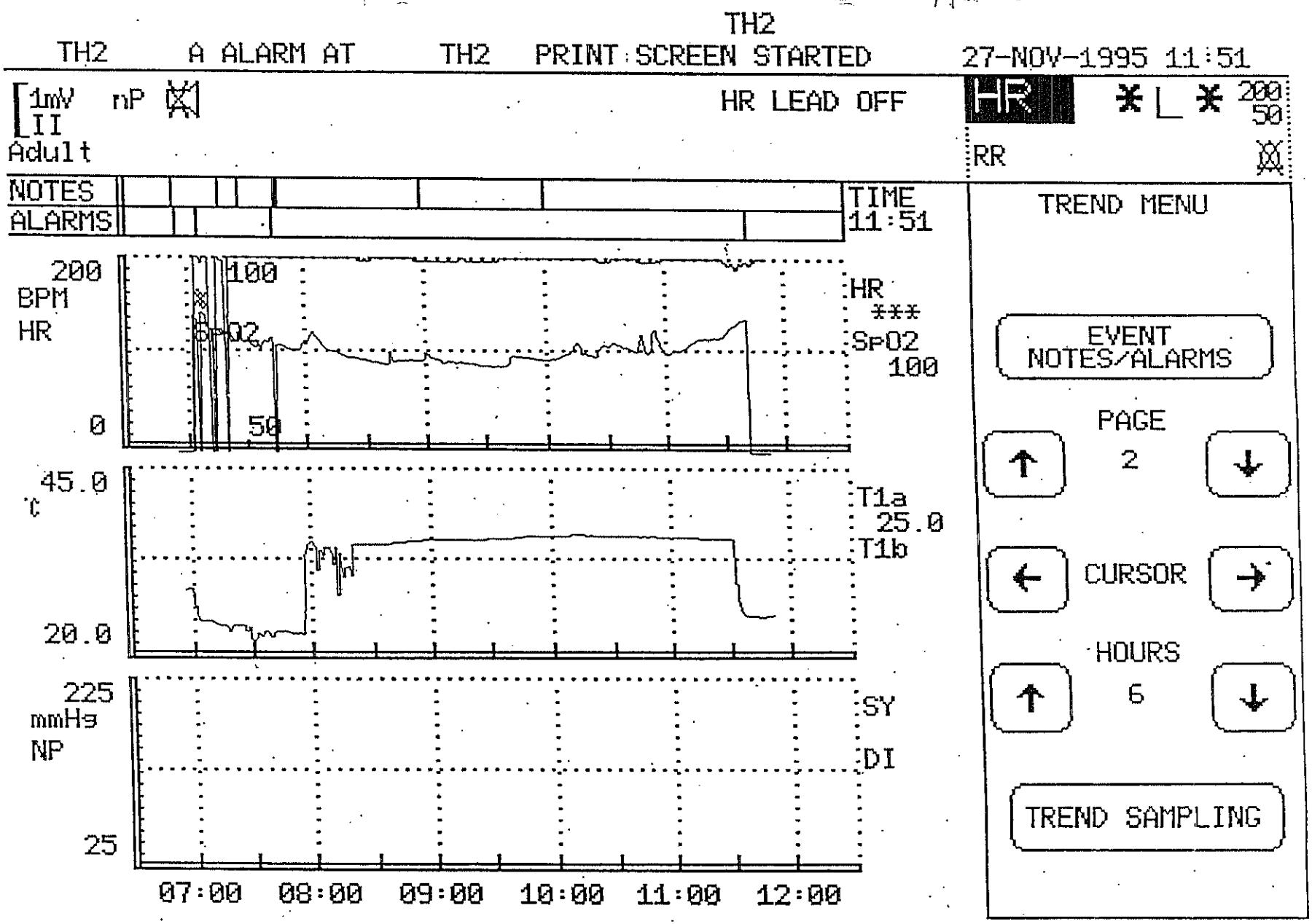

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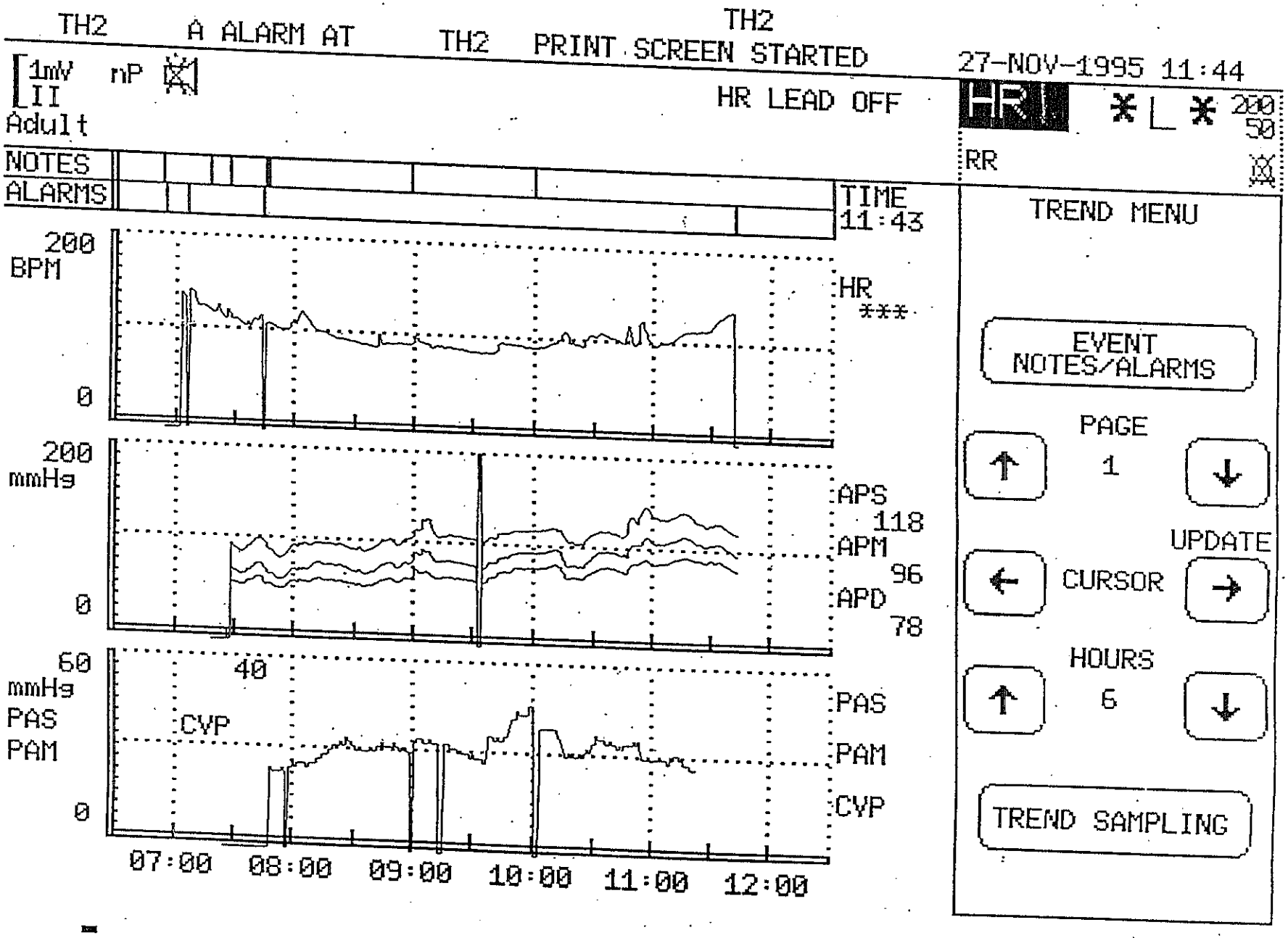
Adam Strain



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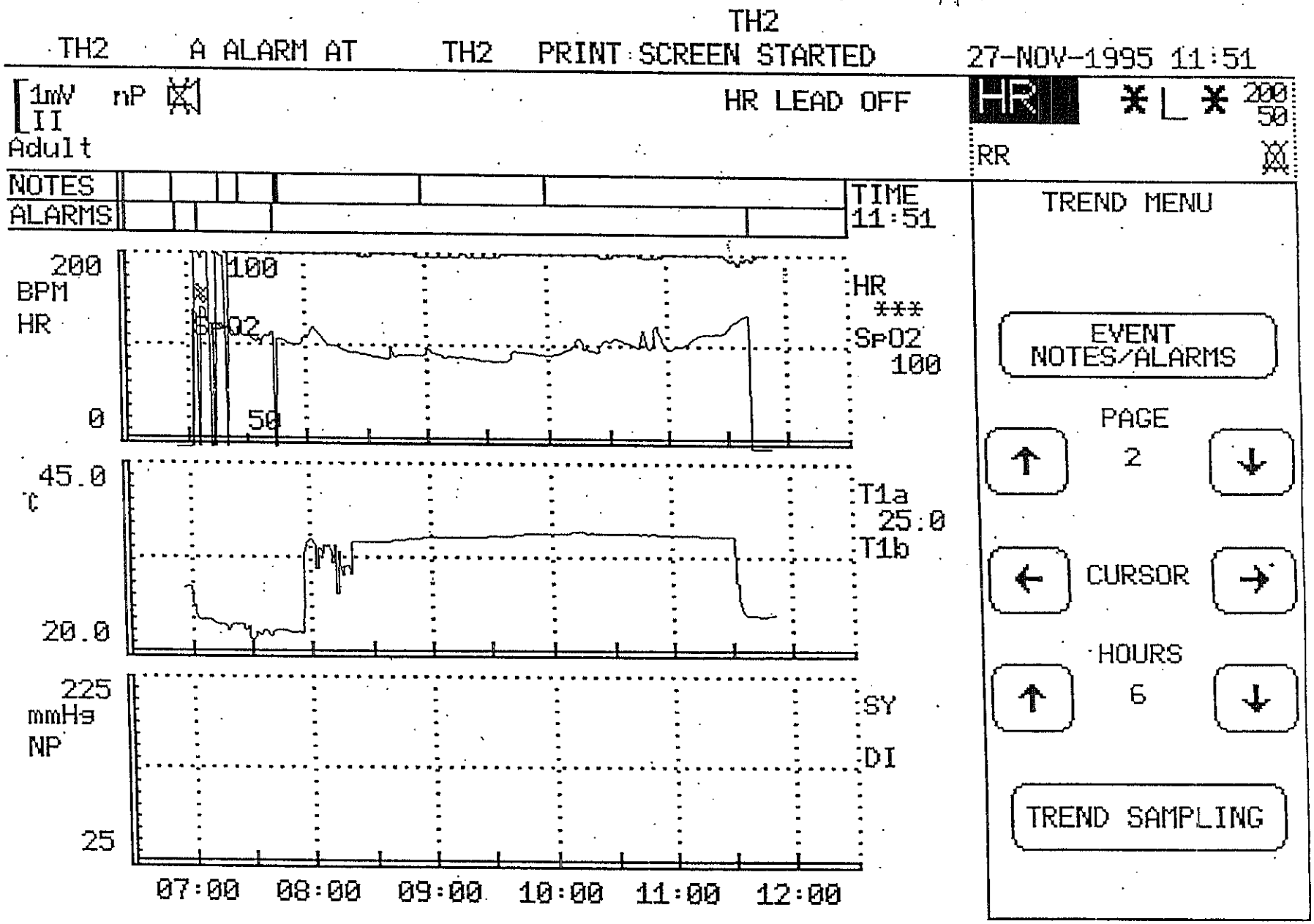
Adam Simm.



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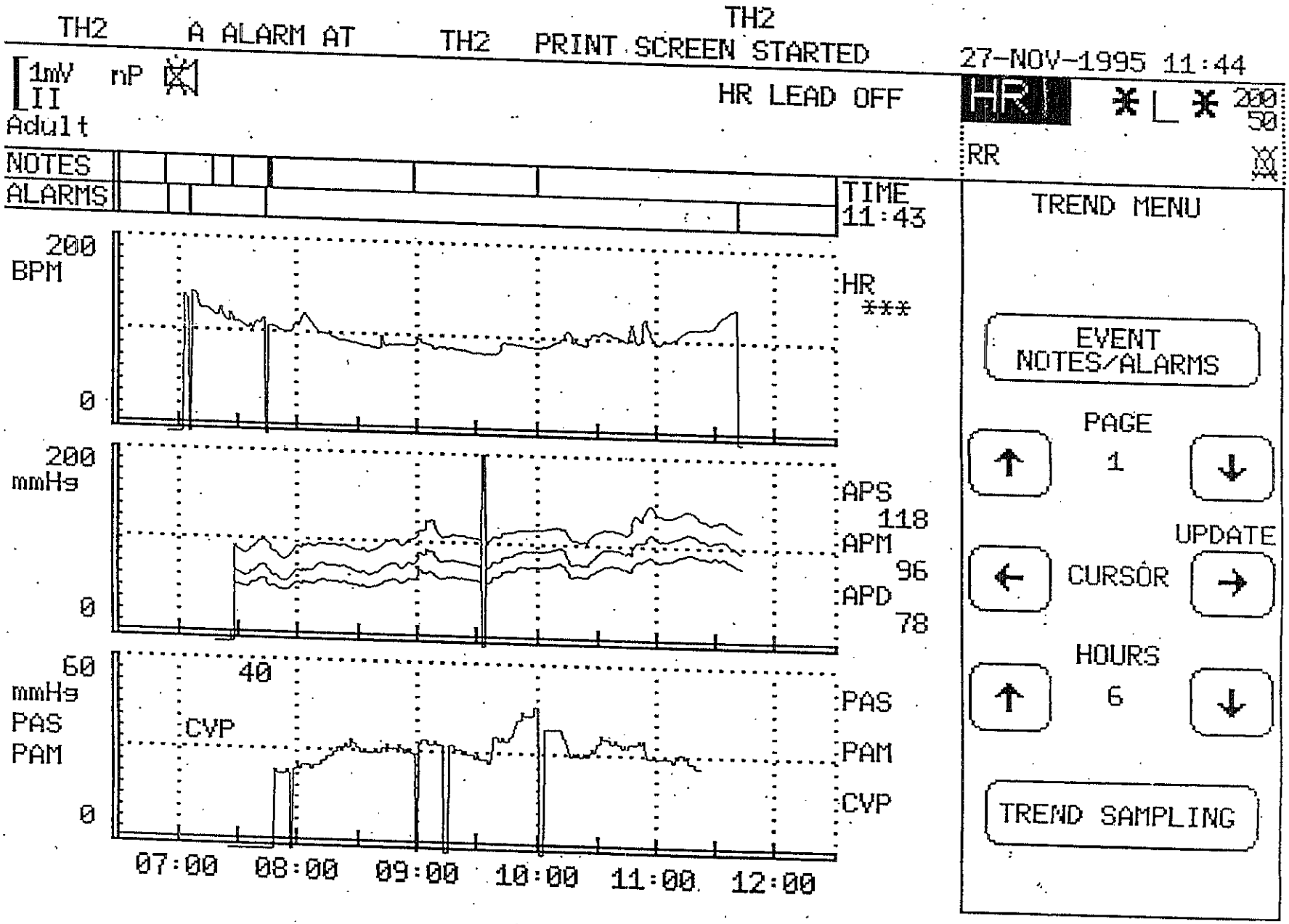

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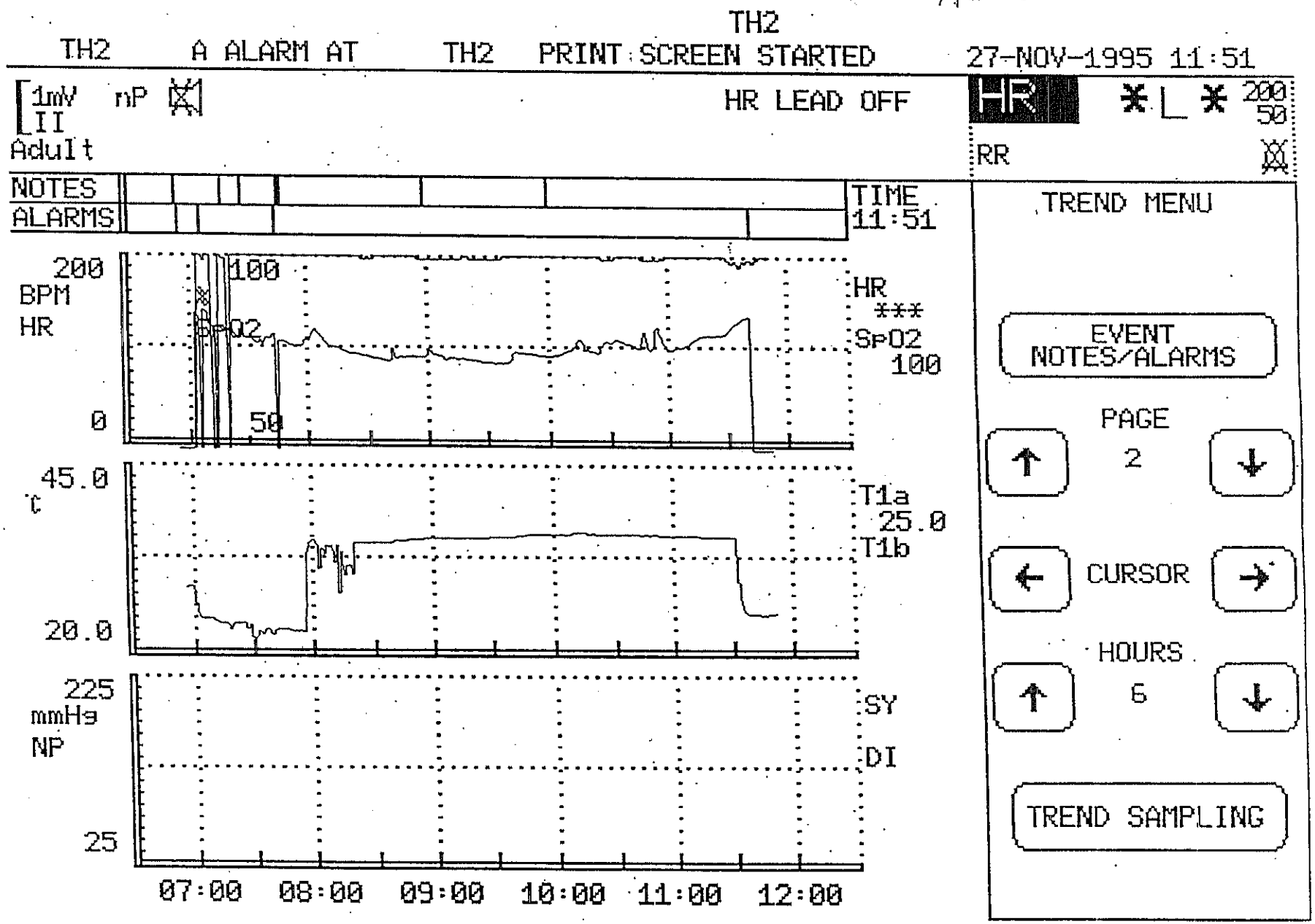

AS - PSNI

094-192-921

28th May 1996

Mr J.L.Leckey LL.M
H.M. Coroner
Coroner's Office
Courthouse
Crumlin Road
Belfast
BT14 6AL

Debra Strain
[redacted]

Dear Mr Leckey,

Thankyou for sending me a copy of Dr Armours' report which although I found upsetting, was helpful. Unfortunately there was one point which was not quite right (see attached). Adam was only fed 600mls during the day not 900mls, as stated by Dr Armour. From what I have been told a major factor which caused Adam to suffer Dilutional Hyponatraemia was Fluid Overloading.

I thought it best to inform you that he was fed 2100mls in total per day, which was less than he received in his five hours of surgery.

I hope this information will be of some use to you.

Your sincerely

Debra Strain

enc.

H.M. CORONER'S OFFICE
29 MAY 1996
GREATER BELFAST

REPORT ON EQUIPMENT USED DURING UNTOWARD INCIDENTS IN THE OPERATING THEATRES, RBHSC

Mr B. McLaughlin, Medical Technical Officer 4 and Mr J. Wilson, Medical Technical Officer 5 examined the anaesthetic, temperature control and monitoring equipment used in the theatre under investigation.

The investigation was carried out between 0900 hours to 1130 hours on Saturday 2 December 1995.

The equipment examined consisted of the following,
Lamtec Anaesthetic Machine, Model 990-905, Serial No. 8704905089
Penlon Nuffield Ventilator, Serial No. 0387-06 fitted with either the NV200 valve, Serial No 33694 or the Paediatric valve, Serial No 432004.

Siemens Patient Monitor, Model 1281, Serial No.
(This monitor is currently out for repair - a new display screen is being fitted and a loan monitor is in use.)
Datex Ultima, Model ULT V-21-01, Serial No 31523.
Hudson Oxygen Analyser.

The Siemens Monitor measures vital signs including ECG, Blood Pressure, Temperature, Heart Rate and Respiration.

The Datex measures End Tidal Carbon Dioxide (ETCO2) and oxygen concentrations (FIO2) in the breathing circuit.

To assist in maintaining the patient's temperature an Aqua-K-Thermia Unit is used. A water blanket in placed below the patient and the circulating water kept at a suitable temperature to maintain body temperature. The patient's temperature in monitored on the Siemens monitor using a reusable general temperature probe.

All service reports pertaining to the equipment were examined and no indication of malfunction found in the documentation. The parts replaced are standard under preventative maintenance and functional checks. The service reports for the period under investigation are Ulster Anaesthetics Job No.DD833 and DD834 (Nuffield) and Anaesthetic Services 7524,7232, and 6992 (Lamtec).

A copy of the service report for the Siemens monitor is expected this week but verbal indications are that nothing untoward was discovered during its overhaul.

The Datex monitor is not on service contract but the calibration was checked and found to be satisfactory.

The Aqua-K-Thermia Unit is not on contract and as it is over 10 tens old does require regular maintenance and must be considered for replacement. It was difficult to assess its performance over a short period, but at the time of the investigation it appeared to work satisfactorily.

All monitor alarms worked and gave no cause for concern.

The Lamtec and Nuffield were set-up and connected to the test lung fitted with a Wright's Respirator and a Hudson Oxygen Analyser. Once a steady state was achieved the patient circuit was disconnected and the low pressure alarm became active within 20 seconds (as specification).

The steady state was again achieved and the oxygen pipeline supply disconnected causing the Alarm Whistle to be activated (as specification).

The standby oxygen cylinder fitted to the Lamtec was opened and the oxygen supply restored (as specification).

All cylinders were removed from the Lamtec, one nitrous oxide (N2O), two medical air, one Carbon Dioxide (CO2), one oxygen (O2). The Pin Index System was checked for security. Five pins were discovered to be loose and could be removed. One on N2O, both on the CO2 and both on the O2. This effectively removes an essential safety feature from the machine and allowed the investigators to fit the CO2 cylinder in the O2 yoke and supply CO2 via the O2 flowmeter.

At this stage the O2 supply was still from the hospital pipeline system, that the valve system on the Lamtec should maintain. Instead the supply from the cylinder replaced the pipeline O2 supply and the percentage oxygen in the breathing circuit fell from 50% to 11%. All anaesthetic machine and ventilator alarms were bypassed. The Datex monitor did function correctly and the high CO2 and low O2 alarms were activated.

It must be clearly stated that this could only be achieved by gross misconduct and failure to use the monitoring equipment.

The pins were re-inserted and the Lamtec put back to a safe working condition and again checked by a second person to ensure

correctness of gas delivery. The purity of oxygen was checked and also found to be satisfactory.

Examination of theatre practice would indicate that the cylinders are checked daily by the medical technical officer (MTO) on duty and the cylinders are only changed by the MTO. The Lamtec log book was examined and found to be signed daily prior to the commencement of the days list by the MTO after all safety and function checks were carried out satisfactorily. The Anaesthetist using the machine is also expected to sign the log before commencing the list but this does not happen on most occasions. A reason for this omission should be requested.

The anaesthetic machine is approximately 10 years old and has been regularly serviced by Anaesthetic Services. The last visit was on 12 September 1995. It is difficult to believe that 5 pins have come loose in 3 yokes in such a short time. This must be considered as a major omission on the part of the service company and requires investigation.

It is also essential that all cylinder yokes are replaced or repaired as a matter of urgency. A check of all pin index equipment within the Trust should be carried out forthwith to ensure the safety of such systems. This will include oxygen cylinders in use at ward level.

Finally it must be emphasised that the protocols and monitoring procedures set up within the RBHSC's Theatres, for more than 2 years, would have discovered if a reversal of cylinders had occurred. If these procedures had been ignored the following actions had to occur;

1. MTO did not check the anaesthetic machine
2. Anaesthetist did not check the anaesthetic machine.
3. The fresh gas supply was not checked.
4. The Datex monitor was not used.
5. Poor tissue oxygenation was ignored by the Surgeon.
6. The pulse oximeter was not used.

The procedure for constructing arterial lines was examined and found to be satisfactory and in accordance with other areas within the Trust

In conclusion the equipment was found to be in satisfactory condition. The current practices covering anaesthetic and monitoring equipment are safe and satisfactory.

ADAM STRAIN (DECEASED)

MEDICOLEGAL REPORT

REPORT OF PROFESSOR P J BERRY
23.3.96

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I am Peter Jeremy Berry of [redacted] My qualifications are BA, MB, BChir, FRCP, FRCPath. I am professor of Paediatric Pathology in the University of Bristol, and have been a Consultant Paediatric Pathologist for more than 12 years.

At the request of HM Coroner for Greater Belfast, Mr J L Lecky, LLM I have examined copies of the case notes of Adam Strain referring to his last admission, the report of Dr M Savage (Consultant Paediatric Nephrologist) reports of Dr R H Taylor (Consultant Paediatric Anaesthetist), and the report on equipment used during Adam Strain's transplant operation. I have also examined 15 stained microscope slides taken at the time of Adam Strain's post-mortem examination.

Background:

Adam Strain was 4 years old at the time of his death. He had a history of chronic renal failure and polyuria with recurrent urinary tract infections in infancy. He had undergone multiple urological operations for vesico-ureteric reflux and a fundal plicaton for hiatus hernia. His renal function had deteriorated to a point where peritoneal dialysis was required in 1994. His nutrition was maintained by night time gastrostomy tube feeding and he was taking multiple medications.

As a result of his treatment, and despite his underlying condition he was well grown and normally nourished.

On the 26th November 1995 he was admitted to the Royal Belfast Hospital for Sick Children to receive a kidney transplant. His pre-operative blood tests including electrolytes, haemoglobin, and coagulation studies were satisfactory.

I will not comment on his pre-operative preparation and intraoperative fluid management which are beyond my expertise. However, no major difficulties were encountered during the operation during which his cardiovascular status and oxygenation remained satisfactory. The surgery was complex, but a satisfactory transplant was carried out with an acceptably matched kidney from a 16 year old donor.

Quite unexpectedly Adam Strain failed to breath spontaneously after his operation and he was found to have dilated pupils and bilateral papilloedoma. There was pulmonary oedema by chest x-ray, and an emergency CAT scan showed cerebral oedema with tonsillar herniation. Tests of brain function were carried out on two occasions and confirmed brain death. Ventilatory support was withdrawn at 11.30 am on 28.11.95 on the second post-operative day.

The reports of Dr R H Taylor, Consultant Paediatric Anaesthetist suggest that the problem was pulmonary and cerebral oedema, although the cause was not apparent.

A report on the equipment in use, while indicating deficiencies showed no cause for equipment failure.

The hand-written report of the surgery in the clinical notes indicates no life threatening operative complication and the kidney perfused reasonably. At 12.05 pm the central venous pressure is recorded as + 30 cm. On examination both pupils were fixed and dilated. Both optic discs were indistinct with retinal haemorrhages. Adam was described as "puffy"

Contd.....

Continuation Sheet 1

I have not been shown a copy of the provisional post-mortem findings. However the brain weighed 1320 grams. I understand that the brain and spinal chord are awaiting a neuropathological opinion. The heart was donated for valve transplantation.

Microscope slides

Kidney: Sections show a scarred kidney with numerous cysts, interstitial fibrosis and chronic inflammation, tubular atrophy, glomerulosclerosis, prominence of the juxta glomerular apparatus, hyperplastic tubules with circumferential mesenchyme, a single focus of hyaline cartilage, Tamm-Horsfall protein and thickened arterioles. The number of glomerular generations is reduced. Many of the cysts appear to be medullary.

Spleen: There is intense congestion of the red pulp.

Lungs: There is capillary congestion, occasional clusters of lymphoid cells, and a moderate number of intra-alveolar macrophages. Oedema is not conspicuous, and there is no evidence of embolism. A section of larynx shows superficial ulceration associated with intubation, and mild mucus retention in mucous glands.

Liver: Normal lobular architecture is accentuated by post-mortem change or possible mild extension of fine fibrous trabeculae from portal tracts. There are curious foci of clear cell change in hepatocytes scattered throughout the liver substance. I do not know the significance of these nor can I relate them to any underlying disease process. Portal tracts do not show the changes seen in hereditary renal cystic diseases.

Lymphnode: No significant abnormality.

Transplant Kidney: The kidney shows almost complete infarction.

Comment:

From my examination of the histological sections I can confirm that this child had severe renal disease supporting the clinical decision to undertake renal transplantation. I note the clinical history of reflux and recurrent urinary tract infection. Whilst the histological appearance is entirely consistent with cystic renal displasia, the medullary cysts, intense interstitial fibrosis, and the history of polyuria raise the possibility of medullary cystic disease. (This is not relevant to the child's death, but may be important in counselling and can be resolved from the clinical history).

The transplant kidney was infarcted (dead). The extent of the change suggested that this occurred at or before the time of transplantation. This could be resolved by enquiries about the fate and function of the donor's other kidney after transplantation.

Contd/....177

Continuation Sheet 2

The histological material available to me does not include brain, heart, pituitary, adrenal gland, intestine or skeletal muscle. These tissues might have a bearing on the cause of post-operative death, although are unlikely to do so in the circumstances of this case. Sections of bone and parathyroid glands are part of the post-mortem examination of patients with renal failure.

From the material available to me I have been unable to determine an anatomical cause or underlying disease to account for this child's failure to recover from his transplant operation.

P J BERRY

To Whom it may concern

I visited the operating theatre suite of the Childrens Hospital on 02/12/95 at the request of Drs G Murnaghan and J Gaston to discuss with Dr R Taylor three patients whose post-mortem examinations had been brought to the attention of the Coroner.

I was accompanied by Mr J Wilson and Mr B McLaughlin Senior Medical Technical Officers on the site who carried out checks into the ventilators and other equipment in the theatre.

The technical checks demonstrated a high degree of vigilance in this area, found nothing at fault in relation to the cases in question but identified a problem relating to pin indexing which the whole hospital will now address.

The three cases in question were all very complex in different aspects

Case 1

[redacted]

Case 2

[redacted]

Case 3

A four year old child with polyuric renal failure was brought to theatre for renal transplant and a very carefully thought out and well monitored anaesthetic was delivered with great care to fluid management -- in a child whose normal urine output was 100mls per hour. This child was well known to the anaesthetist as he had anaesthetised the youngster very many times in its short life. Full records of all monitored parameters are available on this case and show that no untoward episode took place and that a very stable anaesthetic was given. At the end of the operation the child was found to have fixed and dilated pupils and a C.T. scan showed it to have gross cerebral oedema.

Although all these cases were tragic in their consequences and outcome, all three were cases of significant complexity with a substantiall increased risk of morbidity and mortality. All cases were performed in the same operating room -- that being the room used in the suite for all major surgical procedures. Each case was performed by a different surgeon and each anaesthetic conducted by a different anaesthetist -- all of Consultant standing. All the cases were extensively monitored, including the use of pulse oximetry.

The Protocols for monitoring, anaesthetic set-up and drug administration in this area are among the best on the Royal Hospitals site and I can see no reason to link these very sad cases into any pattern.

Signed Fiona Gibson

Fiona Gibson MD FFARCSI
Consultant Anaesthetist

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Debra Strain
[redacted]

6th February 1996

Mr J Leckey LL.M.
H.M. Coroner
Coroner's Office
Courthouse
Crumlin Road
BELFAST
BT14 6AL

H.M. CORONER'S OFFICE
-8 FEB 1996
GREATER BELFAST

Dear Mr Leckey,

Thankyou for sending me Dr Sumners report. I am sorry that I cancelled my appointment with you but I thought as you are so busy and I was afraid of taking up too much of your time, it would be better to write to you with my concerns.

I very much appreciate you asking Dr Sumners expert opinion as it is a comfort to me to know how much you have done to find answers. Although I discussed the report with Dr Savage there are obviously some things that have been said concerning what happened on that terrible day that I cannot understand and are not as I remember them. The first thing is that I have been told that a possible reason Adam did not have his Electrolytes checked before going to theatre could have been that two doctors had tried for an hour between 5 and 6 a.m. to find a vein to put a cannula into Adam without success, so therefore getting any blood would not have been possible. This I know happened because I was there comforting Adam and when the second doctor gave up she told me Dr Taylor would be coming in at around 6.20 a.m. and would come to Musgrave Ward to see Adam and he would put a cannula in at that time. As I have pointed before at no time did Adam see Dr Taylor before the transplant which I did think unusual. Another thing is that I was told by Dr M O'Connor who was keeping me in touch with what was happening with Adam in theatre that morning, that surgery ended at 11.50 a.m. not 11.00 a.m. as Dr Sumner said in his report. This may only be a small point but I can't help wondering at exactly what time surgery ended? Also Dr Sumner states that the I.C.U. staff described Adam as 'puffy' when he went to the Unit, I would like to point out that Adam was not just 'puffy' he was extremely bloated that was why I was so distressed when I first saw him at 12.15 p.m. and in all honesty he did not even look like my little boy which is what I said at the time. This can be seen from a photograph taken by a nurse 24 hours later on the day Adam passed away when he was actually less 'puffy' than straight after transplant. The straight forward explanation of the report that I was given, was that "Adam was quite simply fluid overloaded", but Dr Taylors's opinion was that he gave the right amount of fluid on the day this is one of the most upsetting things that I have heard because I just cannot comprehend how such a mistake can be made.

cont/d....

I sincerely hope you do not mind me voicing my concerns and I hope you can understand how distressing all this is for me and how much I need to know for my own peace of mind that I am also doing everything I possibly can for my son.

I am sorry that I do not know legally what questions I can ask you but if it is allowed could you please let me know if you are going to call Dr Sumner as a witness at the inquest?

Once again thankyou for all you are doing.

Yours sincerely,

Debra Strain

DEBRA STRAIN

ADAM STRAIN

4TH JANUARY 1996

Miss Strain and her brother-in-law called to see me at 2.00 pm and spent about an hour with me. I went over all the investigations that were carried out and I promised to send Miss Strain a copy of each report as it arrived with me. I said that I would ask Dr Savage from RBHSC if he would be willing to explain each to Miss Strain. I said that if all the reports were in by the end of January it may be possible to hold the Inquest before Easter. I said that in medical inquests of this nature a major problem was finding a date that suited all the medical witnesses.

Miss Strain advised me that Dr Armour was not correct in stating that Adam had had 5 fundoplications. He had 5 reimplantations of the ureters between 2nd November 1991 and January 1992 and one fundoplication on 16th March 1992.

Miss Strain told me that she had been present at the operation from 7am onwards. She said that she was annoyed that Dr Taylor had given an epidural and also that the surgeon was Mr Brown. Mr Brown had been involved with the previous surgery in connection with the ureters and this had not been successful. With regard to the epidural, one had been given at the time of a previous procedure and had not been successful. During surgery she had been told that the bladder was not emptying properly and it might be necessary for her to catheterise Adam twice daily. Miss Strain had told me that there were 10 files of medical notes and she wanted to know if these had been made available to the various experts. I said I would check this out with Dr Armour.

Subsequently I spoke to Dr Savage and he agreed to explain the reports to Miss Strain provided that Dr Murnaghan was happy and there were no medico-legal reasons to suggest otherwise.

Subsequently I spoke to Dr Armour. She had not sent copies of all 10 files to all the experts due to the huge number of records involved. I suggested that she should write to each saying that these files were in existence and would be available via Dr Murnaghan. She agreed to. Also I told her what Miss Strain had said about the 5 fundoplications. She noted this but did not feel it made any difference.

Q.(1) ~~WHY~~ HOW DID ADAM DIE?

A:— CEREBRAL OEDEMA — SWOLLEN BRAIN.
POST MORTEM EXAMINATION FINDING.
THIS WE KNOW!

Q.(2) WHY DID ADAM'S BRAIN SWELL?

A:— HYPONATREMIA — ie LOW SODIUM — ie LOW SALT.

SODIUM 123 m m/LITRE (NORMAL 135 → 145)
AT 9.32 AM ON MORNING OF ADAMS SURGERY

SODIUM 119 m m/LITRE IN INTENSIVE CARE UNIT.

Q.(3) WHY WAS THE SODIUM LOW?

A:— HIS BLOOD WAS DILUTED BY FLUID LOW IN SODIUM
500 c/c X 3 = 1.5 L. of 4% DEXTROSE, ⅕ NORMAL SODIUM.

Q.(4) WAS IT REASONABLE TO GIVE THIS TYPE OR VOLUME OF FLUID TO ADAM?

A:— 1ST LET US CONSIDER TYPE OF FLUID

ADAM WEIGHED APPROX 20Kg — UP TO 25% OF HIS WEIGHT
IS EXTRACELLULAR FLUID ie 5 LITERS.

5 LITERS @ 140m m/L = 700mm
1.5 LITERS @ 24 m m/L = 36 m m > TOTAL 736 m m.

IT IS LIKELY THAT ADAM PASSED 70 c/c OF URINE PER HOUR.
AT 9.30 ADAMS EXTRACELLULAR FLUID WOULD BE 6.5L - 0.2L = 6.
72L ÷ 6.2 = 119 m m/LITER

Q 4 CONTD

THIS ASSUMES THAT THE WATER IN THE FLUID GIVEN TO ADAM IMMEDIATELY OR ALMOST IMMEDIATELY LEFT THE BLOOD STREAM (VASCULAR SPACE) AND ENTERED THE EXTRA VASCULAR (TISSUES) SPACE.

NOW LET US CONSIDER THE VOLUME OF FLUID.

ADAM USUALLY RECIEVED 1500cc AT NIGHT + 300cc X 2 DURING DAY
TOTAL 2100 cc's/DAY
IE 90 cc's/HOUR

DURING THE NIGHT PRIOR TO SURGERY ADAM RECIEVED 950ccs
950 cc's ÷ 90 = 10+
IE ENOUGH FLUID FOR 10+ HOURS

IT IS HIGHLY LIKELY THAT ADAM WAS ADEQUATELY HYDRATED WHEN HE ARRIVED IN THE OPERATING ROOM.

DURING SURGERY ADAM SHOULD RECIEVE 6→8cc/Kg/HOUR
ADAM WEIGHED 20 Kg IE 120 → 160 cc's/HOUR

THIS IS MORE THAN ADAM'S USUAL 90 cc's/HOUR AND TAKES ACCOUNT OF "3RD SPACE" LOSS
IE SWELLING CAUSED BY SURGICAL TRAUMA TO THE TISSU

ADAM WOULD ALSO REQUIRE TO HAVE ANY SIGNIFICAN BLOOD LOSS REPLACED.

REPLACEMENT 2u PACKED CELLS = 450 cc's + PLASMA 800cc's + HARTMANS 500cc = 1750
−1100.
650 cc

BLOOD LOST (ESTIMATE)

NET CONTRIBUTION TO FLUID BALANCE

Q4 SECOND PART CONTD.

ADAM RECIEVED 500 cc's x 3 ½ N.NACL = 1500 cc's
+ CONTRIBUTION FROM BLOOD REPLACEMENT 650 cc's
2150 cc's

ADAM REQUIRED (SEE EARLIER)
150 cc's / HOUR FOR 4 HOURS = − 600 cc
1550 cc's

ADAM RECIEVED 1,500 cc's TOO MUCH FLUID.

Q5. HOW DOES A DOCTOR TELL THAT A PATIENT IS PROPERLY HYDRATED?

A:- (i) URINE OUTPUT — NOT APPLICABLE IN ADAMS CASE

(ii) GENERAL APPEARANCE — eg SWOLLEN TISSUE

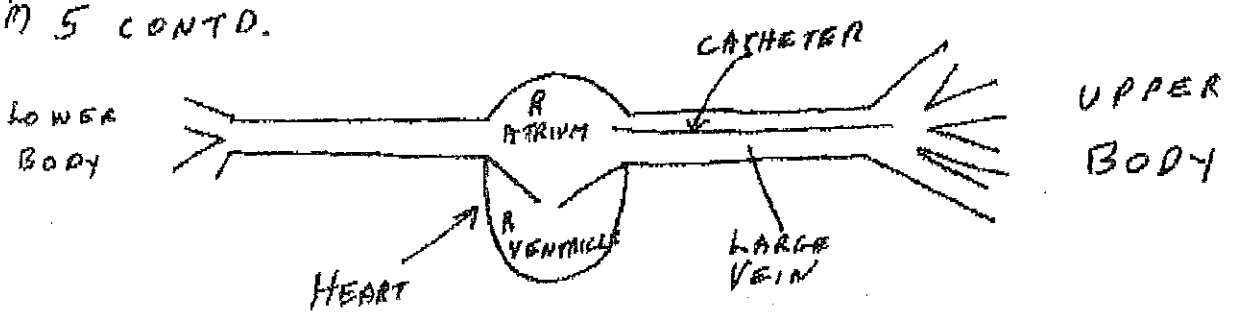
(iii) CENTRAL VENOUS PRESSURE

BACKGROUND:- THE CENTRAL VENOUS SYSTEM IS COMPOSED OF THE LARGE VEINS IN THE BODY, AND UNLIKE THE PERIPHERAL (SMALL) VEINS THERE ARE NO VALVES IN THE SYSTEM. THIS MEANS THAT PRESSURE IS TRANSMITTED EVENLY THROUGHOUT THE SYSTEM.

THE PRESSURE READING IS TAKEN AT THE END OF A CATHETER (TUBE) PLACED IN RIGHT ATRIUM (... THE RIGHT CHAMBER OF THE HEART).

PAGE (4)

M 5 CONTD.



THE USUAL POINT OF ENTRY TO THIS SYSTEM IS VIA THE NECK VEINS OR THE SUBCLAVIAN (BELOW CLAVICLE = COLLAR BONE) VEINS

IN ADAMS CASE ~~[illegible]~~ ONE OF THE NECK VEINS HAD BEEN PREVIOUSLY TIED OFF.

DR TAYLOR WAS SUCCESSFUL IN GAINING ACCESS TO THE CENTRAL VENOUS SYSTEM AT THE 3RD SITE HE TRIED.

EACH ATTEMPT CARRIES WITH IT RISKS, INCLUDING DAMAGE TO THE LUNGS WHICH ARE CLOSE TO THE VEINS

OBVIOUSLY DR TAYLOR THOUGHT THAT IT WAS IMPORTANT TO PLACE THIS CATHETER (TUBE) OR HE WOULD NOT HAVE EXPOSED ADAM TO THESE DANGERS.

MOST ANESTHETISTS WOULD AGREE! [I DO!]

THERE ARE 2 MAIN REASONS TO PLACE A TUBE IN THIS SYSTEM

(i) ACCESS TO THE BLOOD STREAM (ADAM ALREADY HAD A TUBE IN ONE OF HIS VEINS

(ii) TO MEASURE THE PRESSURE IN THIS SYSTEM.

Q5 CONTD.

THE NORMAL PRESSURE IN THIS SYSTEM IS :-

0 → 8 CMS. WATER [SEE ATTACHMENT (1)]

THE PRESSURE IN ADAM'S SYSTEM WAS :-

17 mm Hg (MERCURY) OR 22 CMS WATER.

OBVIOUSLY ADAM WAS MORE THAN ADEQUATELY HYDRATED

NOTE :- THIS WAS A KIDNEY TRANSPLANT OPERATION.

IT WAS IMPORTANT TO KEEP ADAM WELL HYDRATED

A CENTRAL VENOUS PRESSURE OF 10 → 14 CMS WATER

IS CONSIDERED OPTIMAL.

THIS 10 – 14 CMS H_2O FIGURE WOULD NORMALLY

INCLUDE AN ALLOWANCE FOR THE INCREASE

CAUSED BY MECHANICAL VENTILATION.

QUESTION (6)

WHY DID DR TAYLOR IGNORE THIS READING

HE HAD EXPOSED ADAM TO THE RISKS OF PLACING THE CATHETER — 3 SEPERATE SITES.

ONLY DR TAYLOR CAN ANSWER THIS QUESTION.

MY PERSONAL VIEW IS THAT IT IS MANDATORY TO INVESTIGATE THE CAUSE OF A C.V.P OF 22 CMS H_2O AND TO TREAT THE PATIENT IN A MANNER APPROPRIATE TO THE CAUSE (POSSIBLY DOCTOR INDUCED)

A PRUDENT COURSE OF TREATMENT WOULD INCLUDE :-

(1) RESTRICT FLUIDS IE ONLY ENOUGH TO KEEP LINES OPEN

(2) CHECK EQUIPMENT

(3) PULL CATHETER BACK TO A POSITION WHERE IT COULD NOT POSSIBLY BE IN THE VENTRICLE — A PLACE WHERE IT IS REASONABLE TO EXPECT PRESSURES OF 22 CMS H_2O BUT ALSO A PLACE WHERE THE TIP OF THE CATHETER CAN CAUSE CARDIAC IRREGULARITIES

(4) CHEST X RAY — TO CHECK PLACEMENT.

I BELIEVE THAT IT IS GOOD PRACTICE TO ALWAYS TAKE A CHEST X RAY AFTER PLACING A CENTRAL LINE FOR 2 REASONS:- (i) PLACEMENT. (ii) RULE OUT [illegible] LUNG DAMAGE WHICH

HOWEVER AS A COST SAVING MEASURE THIS IS NOT ALWAYS DONE, ESPECIALLY WHEN THE CHEST CAVITY WILL BE OPENED, AS IN HEART OR LUNG SURGERY.

WITH A DIFFICULT PLACEMENT & A HIGH READING THE COST BENIFIT RATIO OBVIOUSLY CALLED FOR A CHEST X RAY.

DR SUMNER IN PARAGRAPH 5 OF HIS REPORT STATES THAT 17mm IS A HIGH READING & THAT 20 - 21 IS VERY HIGH AND ACTUALLY DIFFICULT TO ACHIEVE.

IN MY SEARCH OF THE LITERATURE I WAS ABLE TO FIND ONLY 1 REPORT OF SUCH PRESSURES - SEE ATTACHMENT 2.
[ASTRONAUTS ON SHUTTLE TAKE OF]

DR TAYLOR CONTINUED TO RAPIDLY INFUSE FLUID IGNORING THE WARNING SIGN OF HIGH C.V.P READINGS FOR ABOUT 2 HOURS.

DR TAYLOR'S COMMENTS ON FLUID DEFICIT ARE WRONG

" " " ON GLUCOSE LEVELS ARE IRRELEVENT.

TO CALL A C.V.P READING OF 17mm MERCURY A BASELINE IS INCOMPREHENSIBLE OR RECKLESS