CROSS Billy

From: CROSS Billy
Sent: 11 August 2005 15:59
To: 'esumner'
Subject: 4.UNCLASSIFIED-All Networks: RE: Adam Strain

Dr Sumner,

Thanks again for the early response. I will pursue this now.

Billy Cross

-----Original Message-----
From: esumner [mailto:[redacted]
Sent: 11 August 2005 15:57
To: CROSS Billy
Subject: Re: Adam Strain

I'm attaching Raychels draft report.
John Burton - home [redacted]

I'm back Dunday evening.
Ted Sumner

----- Original Message -----
From: Billy.Cross[redacted]
To: [redacted]
Sent: Thursday, August 11, 2005 3:36 PM
Subject: RE: Adam Strain

Dr Sumner,

Thanks again for the response.

I have spoken to the staff at the Public Inquiry. They have received your correspondence from Dr Burton re Adam. However since there is a delay in the hearings, your statements and the relevant correspondence is not being posted on the web and therefore is not in the public domain and I cannot gain access to it. Can I ask if you have a contact number or address for Dr Burton so that I can seek his consent to obtain a copy of the letter? It may be useful if I can have sight of the letter before we next meet as there may be issues raised in it on which we may wish your views.

Billy Cross

-----Original Message-----
From: esumner [mailto[redacted]
Sent: 11 August 2005 14:48
To: CROSS Billy
Subject: Re: Adam Strain

Thanks for the input - I'm away for the weekend but will finish it at the beginning of next week.
I also have the draft of a report for Raychel Ferguson which I will send
Ted Sumner

----- Original Message -----
From: Billy.Cross[redacted]
To: [redacted]
Sent: Thursday, August 11, 2005 12:37 PM
Subject: RE: Adam Strain

Dear Dr Sumner,

Thank you for the draft report.&nb sp; I regret my delay in responding but I am just back today from a few days leave. It appears to me the draft is in the right direction. I will consult with the senior investigating officer, a DCI Woods when the draft report on Raychel is available and will then reply as necessary.

It appears to me that we may need more explanation of the need for the electrolyte tests, as to be honest the average police offcier and jury mamber may not even be aware that an electrolyte test will give a sodium reading, but this can be clarified later as a matter of detail. We may also need an explanation of the significance and value of blood gas results.

Billy Cross

-----Original Message-----
From: esumner [redacted]
Sent: 03 August 2005 14:30
To: CROSS Billy
Subject: Adam Strain

Dear Mr Cross - I have to say I'm finding this very difficult. Could you please read my first draft and let me know whether I am in the right direction. I must stress that what I say is my own opinion and may not be in line with others.
Having read the whole thing again, I feel Dr Taylor, who was ultimately responsible for both fluid and metabokic management made several errors of judgement.
Please let me know what you think.
Ted Sumner

Any views expressed by the sender of this message are not necessarily those of the Police Service of Northern Ireland. This e-mail and any files transmitted with it are intended solely for the use of the individual or entity to whom they are addressed. If you have received this e-mail in error please notify the sender immediately by using the reply facility in your e-mail software. All e-mails are swept for the presence of viruses.

ADAM STRAIN

In the preparation of this report I have carefully perused the documentation presented to me by the Police Service of Northern Ireland.

I must stress that the comments I make and the answers to questions posed are only my opinion.

At the outset I largely stand by my report dated 22nd January 1996 which was read out in the Coroner's Court apart from the facts that I am corrected by Dr Taylor that the plasma he used intraoperatively was the type which contained sodium and that the transplanted kidney was found to be infarcted at post-mortem.

The verdict on the Inquest states the cause of death to be cerebral oedema due to Dilutional Hyponatraemia and impaired cerebral perfusion during renal transplant operation for chronic renal failure (congenital obstructive uropathy) My own opinion agreed with that as the findings were gross cerebral oedema and coning and other obvious causes of this such as hypoxia having been ruled out. Dr Taylor himself produces a paper which negates the argument that there might have been some obstruction to venous drainage from the head as a causative factor.

In any operation the anaesthetist is the person primarily responsible for the fluid management. I understand that there was a trainee present whose name is on the anaesthetic form (Page916) but is illegible in the copy I have looked at. The role of the surgeon in the fluid management is variable, but in my experience the surgeon usually checks from time to time that everything is under control.
I note that Dr Taylor says that the fluids were discussed with others in the theatre, but does not say whom.

Dr Taylor says that Adam was polyuric, but I could only find urine volumes after the operation. There is no note of urine output during the operation. There was a suprapubic catheter in place and at least for the early part of the procedure urine output could have been measured. I note that after the operation, urine volumes from the catheter were 49, 115 and 35 ml in the first 3 hours postoperatively. However, there is no way of telling whether these figures relate in any way to the volumes being passed pre- or intraoperatively.

I was taught and it was our practice that in polyuric phases, if the patient is not drinking, half the urine volume is replace by the intravenous route, but as normal saline each hour.

Dr Taylor relates his administration of dextrose-saline to this polyuria and suggests this urine volume was 150ml per hour which was required to be replaced.

Adam had been dialysed overnight and the measurement of electrolytes is mandatory after that. They had tried to obtain blood before Adam went to theatre but were unable to achieve venepuncture. In my experience it routine to measure electrolytes after dialysis and even more necessary if this has been done just prior to major surgery.

The dialysis sheet is page 810 and I note that the fluid balance for the peritoneal dialysis has not been filled in. This is a pity because it gives some idea of how fluid depleted, or otherwise Adam was likely to have been after the dialysis.
I do not agree with Dr Alexander (Page 28) when he says "with the benefit of hindsight..." They had tried to obtain blood before the operation so it was in their mind to measure the electrolytes. I do not understand why they did not do blood gases and electrolytes soon after arterial access had been obtained in theatre. The result at 0930 shows a picture of dilution, both of sodium and of haemoglobin, though blood loss not replaced with blood could have contributed to the low haemoglobin.
The management of the electrolytes and acid-base state is the primary responsibility of the anaesthetist in charge of the case.

To try to summarise:

My impression is that Dr Taylor acted in good faith but I believe he made some errors of judgement.

The fluid management in this situation is a very complex affair as the various requirements for replacement of deficits, ongoing hidden and obvious losses (evaporation and bleeding) and the amount being lost via urine all require minute-to-minute judgements based on clinical and biochemical findings.

If Adam died from dilutional hyponatraemia and its acute cerebral effects, then, in my opinion it must have been the volumes of intravenous dextrose-saline which contributed to this.

Dr Taylor had done initial calculations which he justifies, but in my opinion the administration of 500 ml dextrose-saline over a 30 minute period and a further 500ml aver the next 75 minutes was too much of this solution which is basically water, over too short a time and is in excess of his calculations.

I now understand that there was and indeed probably still is widespread ignorance concerning hyponatraemia, in spite of papers on the subject in prominent medical journals. The ignorance was that a dextrose-saline (0.18% saline) solution while being a reasonable choice for use strictly as a maintenance fluid cannot be used to replace abnormal losses such as bleeding or diarrhoea and vomiting in which the losses are sodium chloride.

What is harder to understand is the delay in measurement of the blood gases and electrolytes until 0930 and I have never seen a satisfactory explanation for this. Although a laboratory result would probably take one hour (possibly more at that time of day) instant information is available from blood gas results. The machine for measuring this is usually in theatre or at least nearby in the intensive care unit.
In my opinion this was an error of judgement.

CROSS Billy

From: esumner [redacted]
Sent: 15 August 2005 11:44
To: CROSS Billy
Subject: Re: Adam Strain

Thanks - I put in a paragraph.
Ted Sumner

----- Original Message -----
From: Billy.Cross[redacted]
To: [redacted]
Sent: Thursday, August 11, 2005 12:37 PM
Subject: RE: Adam Strain

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Ted Sumner

Version: 7.0.338 / Virus Database: 267.10.6/69 - Release Date: 11/08/2005

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Adam had been dialysed overnight and the measurement of electrolytes is mandatory after that. They had tried to obtain blood before Adam went to theatre but were unable to achieve venepuncture. In my experience it routine to measure electrolytes after dialysis and even more necessary if this has been done just prior to major surgery. Electrolytes which are usually measures automatically from a blood sample by a machine in the laboratory and include serum levels of sodium, potassium, chloride,

magnesium, bicarbonate, urea and creatinine (the latter two give an indication of renal function. The sodium, potassium and bicarbonate levels can also be measured during a blood gas analysis. These machines are either put in the operating room suite, intensive care unit or very close by for the use of the anaesthetists and the intensivists. Although blood gas analysis is primarily intended for the measurement of the blood (usually arterial) acidity (pH) oxygen and carbon dioxide levels, they also give a reading for sodium and potassium.
The dialysis sheet is page 810 and I note that the fluid balance for the peritoneal dialysis has not been filled in. This is a pity because it gives some idea of how fluid depleted, or otherwise Adam was likely to have been after the dialysis.

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of day) instant information is available from blood gas results. The machine for measuring this is usually in theatre or at least nearby in the intensive care unit. In my opinion this was an error of judgement.

CROSS Billy

From: CROSS Billy
Sent: 16 September 2005 09:56
To: [redacted]
Subject: 4.UNCLASSIFIED-All Networks: Adam Strain

Contacts: Sumner, Dr

Dear Dr Sumner

I have attached a hurriedly prepared outline of issues re Adam which we would like to discuss at the meeting next week. If you do not have time to consider the points I do not think we will be disadvantaged.

If time permits I may have a similar note for Raychel.

Billy

CROSS Billy

From: esumner [redacted]
Sent: 18 September 2005 13:08
To: CROSS Billy
Subject: Re: Adam Strain

There was no attachment!
Can you let me have it?
Thanks - Ted Sumner
----- Original Message -----
From: <Billy.Cross [redacted]
To: <esumner [redacted]; <es015f8200 [redacted]
Sent: Friday, September 16, 2005 9:55 AM
Subject: Adam Strain

Dear Dr Sumner

I have attached a hurriedly prepared outline of issues re Adam which we would like to discuss at the meeting next week. If you do not have time to consider the points I do not think we will be disadvantaged.

If time permits I may have a similar note for Raychel.

Billy

CROSS Billy

From: CROSS Billy
Sent: 19 September 2005 09:11
To: [redacted]
Subject: 4.UNCLASSIFIED-All Networks: Adam

Contacts: Sumner, Dr

Dr Sumner.doc

Dr Sumner,

Hope this works.

Billy

Dr Sumner,

RE Adam Strain.

Here are some questions to which we would like a response. Some will require a very brief answer. The page references refer to the page numbers in the documents which I left with you, but some of the questions may refer to papers not left with you. I will be bringing a full set and we can examine them at the meeting. It is my view that you could answer everything here without preparation and therefore if you do not have time to consider these in advance I do not think that will disadvantage our discussions.

GENERAL

1. Can you provide a CV?

2. Please provide a unified report. At present we have your report to the Coroner and your draft report to the PSNI. It would be greatly beneficial to the consideration of the evidence if you could combine your final report to the PSNI into your report to the Coroner so that we present a single piece of evidence.

ELECTROLYTES

3. Was the existing Na problem a reason to be particularly careful with electrolytes and indicate a greater need for checking their levels? P 205, 33.

4. Was it practical to carry out electrolyte tests? P40.

5. 'There was no reason to believe there would have been a change in electrolytes.' Is that true? Was the opposite not true? P40.

6. Can you explain 'the fluids I gave were isotonic' since 0.18% solution is elsewhere referred to as hypotonic? P40. How quickly would dextrose be metabolised?

7. Would a blood loss of 2/3 make fluid management difficult? Is adjusting for such a situation a routine part of an anaesthetists work, or is it rare? P26

8. Is it right to say 'with the benefit of hindsight...should be monitored'? p27.

9. It is suggested there is a distinction in this case and those referred to by Arieff because in his cases hypoxia was present. Is that relevant? P27.

10. Is it true to say the Na level could be predicted by a 'simple calculation'? p42. Is there evidence anyone did that calculation and so could have predicted the hyponatraemia?

11. 'Blood gas should have...' p24. Why/How important is that?

12. How often would you personally have started an operation like this without electrolyte measurements?

URINE

13. Dr Keane says that urine is never measured during an operation (p30) and Dr Alexander says it is impossible to (p26). What is your opinion on these comments since you have highlighted the lack of such a record.

14. Prof. Berry states that the pre-operation electrolytes were OK (p176). I assume he means the tests from the night before? How far back does pre-op go?

OPERATION

15. Was it foolish to attempt to cannulate as described at p57?

16. How difficult technically was this operation compared to another paediatric kidney transplant? Depositions have highlighted the technical difficulties yet you say it was 'slightly' more difficult (p53). Would it not be the case that most recipients of a donor kidney would have had prior operations leaving adhesions? Did the surgeon make it unnecessarily more difficult by deciding to attach the renal arteries to the iliac rather than the more normal dorsal aorta?

17. Prof Berry refers to almost complete infarction of the donor kidney (p177). What does that mean? He states it happened at or before the transplant – is it possible to time the damage as accurately, is it possible that could have happened and not been seen at the operation? If it was the case at the operation and was seen, what should have been done? If that were true, would it alter your opinion of the cause of death?

18. Are you aware if anyone checked the fate and function of the other donor kidney. P177.

19. Can we check if the 'fluid regime employed successfully with Adam previously' is true? P40.

20. Should a better note have been made re the epidural, where, what extra details should be recorded, why? P55.

21. Explain PD. P55 (peritoneal dialysis?).

22. Explain 'physiological 3rd space'. P56.

23. Was it reasonable to say that Adam's decline and death had 'nothing to do with anaesthetics'? p220.

24. Is there a value in comparing past operation notes with the relevant operation notes?

25. What is the relevance of the 'enlarged bladder'? Would that have been caused during the transplant? P1.

26. Is it right to say that a final haemoglobin level near to that found before the operation confirms good intraoperative management of haemoglobin? P26.

27. Are the views of Dr Gibson reasonable? P180.

28. Where Dr Taylor's records meticulous? P82. Did he not omit recording urine/electrolytes/epidural/CVP?

29. Did anything appear on the anaesthetic monitor? P227.

30. Is it reasonable to read the anaesthetic records and not detect the problem? P227.

31. Is there evidence of hypoxia (p227 eight lines up from bottom)? Dr Alexander says there is not (p27). Also p85.

32. 'Problem with venous drainage'. P24. What was the problem? Did it have a bearing on the cause of death or on the degree of negligence?

33. Explain 'the space is not noted'? p101 (bottom of page).

34. Can you show where on the anaesthetic forms the CVP should have been recorded? P102.

35. Can you demonstrate from the charts the rise in BP. P108.

36. Can you show from the traces the signs of coning? P108.

CVP

37. Why were there no CVP figures and where did Dr Taylor record them? Does a copy of the letter referred to exist? P52, 102.

38. What is the significance of the raised CVP? What action ought to have been taken? What action was taken? P59.

39. How can a CVP of 28-30mmHg be achieved other than by excess fluid?

40. How can body position alter the CVP and what degree of change might one expect?

41. How does the anaesthetist predict such CVP changes and adjust treatment or readings?

42. If there is a suspicion that the transducer is giving a false reading is it possible to check if it is malfunctioning at that time?

43. Why were no tests done on the CVP equipment/transducer? P167.

44. Is it right to assume at first that the transducer was faulty? P26.

45. The Siemens Monitor measures blood pressure. Is this what measures CVP? P167.

46. What is your opinion re the extent of the testing of the equipment? P167.

47. What is your opinion on an initial CVP of 17mmHg? Is that high before any fluids? P79.

48. Explain 'titrated against BP and CVP'. P34a.

49. Explain your view of using 17mmHg as a baseline. P3

CONCLUSIONS

50. What is the meaning of 'on the balance of probabilities'? What other possible causes could there have been? How certain is this cause of death i.e. cerebral oedema caused by dilutional hyponatraemia? Did any medical opinion dissent from that conclusion? P60.

51. Had you seen Prof. Berry's report before preparing your report for the Coroner? Do you think you should have seen it?

52. Dr Alexander says he believes that renal failure may affect hyponatraemia and disagrees with you on that point. P28. Is he wrong and why?

53. 'Case management is extremely difficult' p24. How difficult is it to get it right? Will that amount to a reasonable excuse?

CROSS Billy

From: esumner[redacted]
Sent: 19 September 2005 10:18
To: CROSS Billy
Subject: Re: Adam

Thanks! Ted Sumner
----- Original Message -----
From: <Billy.Cross@[redacted]
To: <[redacted]
Sent: Monday, September 19, 2005 9:10 AM
Subject: Adam

<<Dr Sumner.doc>>

Dr Sumner,

Hope this works.

lly

CURRICULUM VITAE

OF

EDWARD SUMNER, MA BM BCh FRCA

June 2005

NAME: Edward Sumner

ADDRESS: [redacted]

DATE OF BIRTH: [redacted]

MARITAL STATUS: [redacted]

DEGREES HELD:

MA, BM, BCH	Oxford 1966
FRCA	London 1971

ACADEMIC DISTINCTIONS:

First Class Honours Degree
Animal Physiology
Oxford 1963

Nuffield Prize
Primary Fellowship
London 1969

DATE OF MEDICAL REGISTRATION: March 1968

PRESENT APPOINTMENT:

Consultant Paediatric Anaesthetist **(retired)** - Great Ormond Street Hospital for Children NHS Trust

Great Ormond Street
London WC1N 3JH

Appointed September 1973 – Retired June 2001

Director of the Department of Anaesthetics 1987 - 1992

Director: Cardiac Intensive Care Unit 1988 - 1993

Honorary Senior Lecturer : University of London

I had six operating lists each week with cardiac and general paediatric surgery and I am involved in all aspects of paediatric anaesthesia. Approximately 12,000 anaesthetics per year are administered at Great Ormond Street Hospital for Children. The respiratory support service for the whole hospital developed under my supervision.

I was involved in the training of 40 new residents each year, plus at least 12 seconded residents.

PREVIOUS APPOINTMENTS:

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

After my consultant appointment, I was seconded on a part-time basis to the Nuffield Research Department, Royal College of Surgeons, London, for research experience with Mass Spectrometry and its use for inhaled and expired gases (a prototype breathalyser) for the year 1974.

OFFICES HELD I have been elected to the Executive of the Association of Paediatric Anaesthetists of Great Britain and Ireland, Representing England and am chairman of the Scientific Subcommittee.
President Association of Paediatric Anaesthetists of Great Britain and Ireland
President Elect Federation of European Associations of Paediatric Anaesthesia

TEACHING EXPERIENCE:

I have very extensive lecturing and teaching experience all over the world as an invited speaker

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

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

Jackson Rees Lecturer, Erasmus University, Rotterdam October

PUBLICATIONS:

PAPERS:

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

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

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

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Pain
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J Challens, E Facer, E Sumner. Epidural anaesthesia and Dysautonomia (Riley Day Syndrome) *Paed Anaesth* 1999

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Anaesthesia. In Surgery for congenital heart disease. Eds Stark, de Leval, Tsang. Saunders 2004

BOOKS:

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

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

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

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

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

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

Paediatric Anaesthesia. Sumner E and Hatch DJ 1999 London, Arnold

In preparation:

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

Other Publications:

"Blood". An Essay. *The Blood Show*. Five Mile Gallery, Underwood Street, London N1

"Gesundheit" An Essay. *The Sneeze*. University of Central England, Birmingham

EDITORIAL DUTIES:

Editor-in-Chief: Pediatric Anesthesia. Blackwell Publishing. An International Journal.

RESEARCH INTERESTS:

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

Popularised - epidural analgesia in infants and children.

- axillary artery cannulation in infants.

Projects have included - the pulmonary circulation

- gastro oesophageal reflux.

MEDICO-LEGAL WORK:

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

Expert:

Bristol Royal Infirmary Public Inquiry 1999/2000

Belfast: Inquiry into Hyponatraemia-related deaths – present time

CROSS Billy

From: CROSS Billy
Sent: 29 September 2005 09:05
To: WOODS Thomas; NICHOLL Tara
Subject: : FW: Adam

Rachel Ferguson police final d...

For information.

Billy

-----Original Message-----
From: esumner [mailto:[redacted]
Sent: 26 September 2005 12:21
To: CROSS Billy
Subject: Re: Adam

Dear Mr Cross - I did this over the weekend. I have combined both reports into a single document and have tried to incorporate all the points we discussed.
Please let me know what you think.
All the best - Ted Sumner
----- Original Message -----
From: [redacted]
To: [redacted]
Sent: Monday, September 19, 2005 9:10 AM
Subject: Adam

<<Dr Sumner.doc>>

Dr Sumner,

Hope this works.

Billy

CROSS Billy

From: esumner[redacted]
Sent: 05 October 2005 11:03
To: CROSS Billy
Subject: Adam

This is a new draft of the combined, single version of my report.
I must stress that the surgery was done correctly - the transplanted kidney is always put onto the iliac vessels in the pelvis.
I'd be grateful for your opinion.
Thanks - Ted Sumner

Medicolegal Report

On

Adam Strain (deceased)

Prepared for: Police Service of Northern Ireland
Fermanagh District Command Unit
48 Queen Street
Enniskillen BT74 7JR

By: Edward Sumner MA BM BCh FRCA

September 2005

Thanks you for asking my opinion on this case. I am a retired consultant paediatric anaesthetist from Great Ormond Street where I worked from 1973 with a particular interest in paediatric intensive care. I am the author of several textbooks on the subject and am the editor-in-chief of the journal, Pediatric Anesthesia.

For the preparation of this report I have carefully perused the recent medical and nursing notes, but realise, because of Adam's previous medical history there are several older bundles of notes. I have also read the statements and depositions of those involved presented to me by the Police Service of Northern Ireland. The report of the autopsy by DR Alison Armour (pages78-85) is excellent and informative.

The verdict at the Inquest states the cause of death to be cerebral oedema due to Dilutional Hyponatraemia and impaired cerebral perfusion during renal transplant operation for chronic renal failure (congenital obstructive uropathy)

I must stress that the comments I make and the answers to questions posed are only my opinion.

Adam was born on 4.8.1991 with vesico-ureteric reflux causing repeated, damaging urinary tract infections. He had five operations for reflux ending up with one ureter connected to the other with only one draining into the bladder. He also had a fundoplication for gastro-oesophageal reflux and marked vomiting. Nutrition was a problem and it became necessary to give him gastrostomy feeds. Eventually he refused all feeds and it is my understanding that he took nothing by mouth.
He gradually went into renal failure to the point that dialysis was commenced using the peritoneal route. Dialysis took place at night, but Adam also passed urine, presumably of a poor quality and has been described as polyuric. However he was generally progressing quite well having gastrostomy feeds of 3 X 200ml Nutrizon during the day and 1500 ml at night, ie. A total volume of 2100ml per day. He was on the 50th centile for height but on the 95th for weight. In July 1995 he was admitted for a pyrexial illness which was extensively investigated and was probably an infected gastrostomy site. On 14th July he was given a blood transfusion. At the time leading up to his renal transplant in November 1995, he was taking Keflex, Fersanel, Vitamin D, bicarbonate and erythropoietin in addition to his feeds and dialysis regime.

He was not hypertensive as his blood pressure on 18.10.1995 was 106/61 when he had his orchidopexy and on 26th November when admitted for the transplant the following day, the BP was 108/56

The renal transplant took place on 27.11.1995 beginning at 0700, the anaesthetists were consultant Dr Taylor and trainee Dr Montague and the surgeons Mr Keane and Mr Brown. Adam weighed approximately 20kg, had a haemoglobin of 10.5 g/dl with reasonable electrolytes (urea 16.8, but sodium 139) at 11pm on 26.11. Overnight he was given 900ml Diorolyte (4% dextrose, 0.18% saline) via the gastrostomy, instead of his feed, but nothing for the two hours leading up to anaesthesia. Peritoneal dialysis

(PD) was as usual. I can find no note of how much urine per hour he was passing nor of any electrolytes results just prior to anaesthesia. They had tried to obtain blood before the operation in the morning to measure electrolytes but had been unable to do so.
The dialysis sheet is page 810 and I note that the fluid balance for the peritoneal dialysis has not been filled in. This is a pity because it gives some idea of how fluid depleted, or otherwise Adam was likely to have been after the dialysis.

The anaesthetic technique was appropriate for a renal transplant and involved mechanical ventilation, atracurium and an epidural, though the space this was inserted is not noted.
Dr Taylor estimated the blood volume as 1600 ml (80 ml/kg), an estimated fluid deficit of 300ml and calculated an intraoperative fluid maintenance of 200ml/hr.

Central venous access was not easy to achieve. There were three attempts at the left Subclavian vein, one in the left internal jugular, but successful access was achieved in the right subclavian vein using a triple-lumen catheter. There were also cannulas in a vein on the left hand and in the right radial artery. Apart from anaesthesia drugs, also administered intravenously were the antibiotic Augmentin 500mg, methylprednisolone 200mg, Asathioprin 25mg (anti-rejection) and a low, renal vasodilating dose of dopamine by continuous infusion of 5mcg/kg/min, though there is no record of this on the anaesthetic form.

There was considerable blood loss – in excess of 1100ml (two thirds of the estimated blood volume)as the operation was more difficult than usual because of all the previous surgery. The systolic blood pressure started at 85 –90 mmHg and gradually rose, according to the charting, to 120, whereas the pulse rate started high (145/min) presumably because of the IV atropine and gradually fell, dipping to 80/min around 0930 but later rose again from1000. There are no entries in the space available on the anaesthesia record for central venous pressure measurements.

Body temperature was well maintained.

Administered fluids were, dextrose-saline (4% and 0.18%) 1000ml from 0700-0830 and a further 500ml thereafter, 500ml Hartman's solution, 1000 .ml albumin and 500ml of packed red blood cells. A blood gas result taken at 0932 showed mild hypoventilation with PaCO2 44 mmHg (normal 40), very low sodium of 123 mmol/l (normal (135-145) and a very low haematocrit of 18% (normal 35-40%). I could find no note of an earlier result. There is no note of urine output during the case – there is note of a suprapubic catheter, but I do not know whether this was in use in the theatre.

The arterial supply for the kidney came from the iliac artery which is routine for this procedure. The vascular clamps were removed at 1030 and initially the kidney was well perfused with blood and started to produce urine, but by the end of the procedure was not so well perfused which is why dopamine was commenced. In the intensive

care unit postoperatively the kidney was described as "bluish" when the wound was closed

At the end of the procedure, around 1100, Adam was given neostigmine and glycopyrrolate to reverse the neuromuscular blockade, but he did not breathe and was found to have fixed dilated pupils and bilateral papilloedema with haemorrhages. He had obviously suffered a major cerebral insult. On the ICU he was hypertensive, requiring nifedipine to control this. He was described as "puffy" and he had some pulmonary oedema. He was appropriately treated with mannitol and hyperventilation in an attempt to shrink the brain, but a CT scan showed severe cerebral oedema with obliteration of the ventricles and the neurologists confirmed that his signs were compatible with brain stem death ie. he had coned.

Electrolyte results from 27.11 not timed, but in the early postoperative period showed a sodium of 119 mmol/l. A chest Xray showed that the triple-lumen central venous line was going up into the neck vessel. Adam died the following day.

The findings at autopsy included gross cerebral oedema but no substantial pulmonary oedema or oedema of any other organ. It was noted that the left internal jugular vein was tied off where it becomes the innominate vein. The transplanted kidney was infarcted

I would like to make the following comments:

I do not think that the epidural had any part to play. Dr Taylor does not say which level of the spine was used nor how much 0.25% marcain he gave, but there is nothing to suggest an untoward incident with this technique.

Adam was normotensive throughout his life and certainly did not require drugs to control his blood pressure until after the transplant. In that case a systolic BP of 85-90 during anaesthesia is well within the normal range for a child having had an epidural and should not require a fluid load to raise the blood pressure at that stage, particularly as it would be some time before the new kidney was inserted when a "full" blood pressure is beneficial to perfuse the new kidney.

Nowhere could I find a note of how much urine Adam was passing, even though he was described as "polyuric". However, he was in a stable state for several weeks, growing and gaining weight. He was given 2100 ml per day of feed (approx 100ml/kg/day - 4ml/kg/hour) – in addition to this there would be some water of oxidation of the nutrients in the diet. In a stable state intake equals output and his

output in urine, sweat, respiration must equal approximately 2100 ml; in addition to this there would be some volume taken off by the PD. As he was passing urine, the PD would be mainly for electrolyte exchange, K^+, urea, etc but could be in the order of 1-200 ml per day in total. I do not think his urine output could therefore be more than 1500 ml per day – ie. 75ml/kg/day - 3.5 ml/kg/hour on average.

Preoperatively, instead of his feed he was given 900ml Dioralyte (dextrose-saline solution) until two hours before anaesthesia. If we take his average intake as 4ml/kg/hour, then two hours without fluids would give a deficit of 160 ml. Intraoperative maintenance fluids for abdominal surgery are usually calculated at 10 ml/kg for the first hour, then 6-8 ml/kg for subsequent hours. The initial bolus contains extra fluids to make up any deficits from preoperative starvation and then fluid is given for normal maintenance (4ml/kg/hour) plus some extra to replenish evaporation from exposed surfaces and fluid shifts into the physiological third space. It was also thought necessary to give some dextrose to prevent hypoglycaemia, but increasingly dextrose solutions are not used as hyperglycaemia is readily produced. It is better to give isotonic solutions such as Hartman's or lactated-Ringer's solution.

In cases of renal transplant it is usual to be generous with fluids to maintain a CVP of 10-12 to optimise perfusion of the new kidney and to establish its urine-producing function. I think Dr Taylor over-estimated the deficit somewhat, but was reasonable in suggesting 150ml/hour for maintenance, but in fact he gave 500 ml D/S in just 30 minutes (0700-0730) and a further 500 ml over the next hour of this solution – on top of the 900 ml that Adam had been given overnight. A further 500ml over 2 ½ hours is also greater than his calculations.

Up to 0930 he was given 800 ml plasma and 500 ml Hartman's solution for replacement of blood loss. I am assuming that the bleeding was steady, with the odd bigger loss and if Hartman's is used for blood replacement, twice the volume as loss is required. Adam was thus given volume replacement by 0930 of 1050 ml for a total blood loss over four hours of 1100+ ml. Thus the bulk of the fluid administered had been done so one hour before the vascular clamps were removed

I think it was unwise not to have electrolyte values taken before going to theatre and after the PD had been completed. It might be that the serum sodium was already low at that stage. The delay in measurement of the blood gases and electrolytes until 0930 is hard to understand and I have never seen a satisfactory explanation for this.

Although a laboratory result would probably take one hour (possibly more at that time of day) instant information on sodium and potassium values is available from blood gas results. Although blood gas analysis is primarily intended for the measurement of the blood (usually arterial) acidity (pH) oxygen and carbon dioxide levels, it does also give readings for sodium and potassium with a fair degree of accuracy.

The machine for measuring this is usually in theatre or at least nearby in the intensive care unit.
I do not agree with Dr Alexander (Page 28) when he says "with the benefit of hindsight..." They had tried to obtain blood before the operation so it was in their mind to measure the electrolytes, which is the correct thing to do.

In my opinion this was an error of judgement.

By 0930 when the first blood gas was taken Adam was already severely hyponatraemic and diluted (haematocrit 18%) from a combination of excess crystalloid and blood loss. Arterial access had been gained early in the case and it seems logical to analyse the arterial blood for gases and electrolytes as soon as the patient is put on the operating table. Blood loss not replaced with blood could also have contributed to the low haemoglobin.

There is no note of urine output during the case.

It is not surprising in retrospect, that it proved impossible to cannulate the left internal jugular vein and left subclavian since the internal jugular had been tied off. There must have been scars on the skin from a previous surgical approach to the vein. I do not believe it is a sign of dehydration if there is difficulty in cannulating a central vein, unless other signs of dehydration, such as cold peripheries are present.

Cannulation of the right subclavian was achieved, but on subsequent chest Xray the tip was found to be lying in a neck vein, rather than in the right atrium of the heart. Unfortunately this is not an uncommon occurrence especially when the venous anatomy is deranged from multiple previous usage. My own philosophy is that while it is possible to freely aspirate blood, it can be used on a temporary basis, but should be changed at the earliest convenient opportunity. It is not routine practice to Xray for these lines when they are put in, in the anaesthetic room prior to surgery. It is possible that the venous drainage from the head was not completely normal. Dr Taylor did not chart CVP measurements and the information comes from the monitor print-out.

There were obvious problems with CVP readings. It is advisable to attach the pressure transducers to the side of the operating table so that when this is raised and lowered as it so often is during surgery, the zero is not changed.

If the transducer is correctly put to zero, there is free flow of blood in and out of the central line, cardiac and respiratory patterns to the waveform then, in my opinion, the reading is correct. Dr Taylor said that "from the pressure reading I concluded that the tip of the line was not in close relation to the heart" I am not sure how he could say that. I believe that the pressure of 17 mmHg was likely to have been the actual reading at the tip of the catheter. This is a high reading and the rise to 20-21 mmHg is very high and actually quite difficult to achieve in a child because the venous system (including the liver) is very distensible.
With hindsight, knowing that the tip of the catheter was up in the neck and with the knowledge that the left internal jugular vein had been tied off, these high figures could imply there was some degree of obstruction to venous drainage from the head.

This was possibly caused by having the head turned to on side as is usual in theatre, as the CVP came down from 18, the last reading in theatre to 10-12 in the ICU with the head in the neutral position.
Again with the advantage of hindsight, the initial high reading of CVP was probably correct and could have been the result of the initial fluid bolus

If gross obstruction to the venous flow had been present the head alone would have been suffused and swollen as suggested by Dr Taylor in his letter. Adam was described as "puffy" by the ICU staff, but it is not clear whether this refers just to the head. Adam's mother described him as bloated which implies to me, whole body swelling.

It is very interesting to have the monitoring data printed out from the machine. I assume that for the systemic blood pressure with a range of 200mmHg, the half-way line is 100 mmHg. The trace shows much more clearly than Dr Taylor's anaesthesia record the consistent rise in BP from around 0930, soon after the blood gas was drawn, peaking at 150 mmHg. The pulse rate also rose steadily from 1015 onwards. Again, with hindsight these changes could represent the cardiovascular changes of a coning patient under anaesthesia. The arterial trace shows that the line was not interrupted for sampling until just after 0930.

Blood transfusion is usually given to patients who are losing in excess of 15-20% of the blood volume (250-300 ml in Adam's case) Until that point is reached volume is replaced using plasma and/or Hartman's. I think they were rather late in starting the blood transfusion as the haematocrit at 0930 had fallen to 18% (normal 40). Overall, however, the haemoglobin was well managed as the result at the end of the case was 10 g/dl, the level at which it had started.

Dr Taylor suggests that the cerebral oedema is difficult to explain because both thiopentone and methylpredisolone had been given, albeit for other reasons. While methylprednisolone is often given as a cerebral protector, for example for patients going on cardiopulmonary bypass, there are no hard data to support its efficacy. It is 10 years at least since thiopentone was used as a cerebral protector and in much higher doses than those for induction of anaesthesia., Success with animal work was not borne out in the human clinical situation. Modern evidence suggests that barbiturates may even be detrimental.

There is no evidence that the surgical aspects of the renal transplant were not routine. The vascular anastomoses were to the iliac vessels which is normal for this procedure and although the perfusion of the kidney when they closed was not normal, this is often the case and it can be expected to improve with time. It is unthinkable that the wound would have been closed if the kidney had infarcted at that stage.

To summarise, Adam's gross cerebral oedema was caused by the acute onset of hyponatraemia form the excess administration of fluids containing only very small amounts of sodium. This state could have been exacerbated by the blood loss and possibly the overnight dialysis.

A further exacerbating cause could have been an obstruction to the venous drainage of the head but there is no way of knowing whether this was a contributing factor to the brain swelling.

My impression is that Dr Taylor acted in good faith but I believe he made some errors of judgement.

The fluid management in this situation is complex as the various requirements for replacement of deficits, ongoing hidden and obvious losses (evaporation and bleeding) and the amount being lost via urine all require minute-to-minute judgements based on clinical and biochemical findings.

As Adam died from dilutional hyponatraemia and its acute cerebral effects, then, in my opinion it must have been the volumes of intravenous dextrose-saline which contributed to this.

Dr Taylor had done initial calculations which he justifies, but in my opinion they were over-estimations but the administration of 500 ml dextrose-saline over a 30 minute period and a further 500ml over the next 75 minutes was too much of this solution, over too short a time and is far in excess of his calculations. Dextrose-saline strictly speaking is "isotonic" in the bag but basically becomes water in the body as the sugar is rapidly metabolised

I now understand that there was and indeed probably still is widespread ignorance concerning hyponatraemia, in spite of papers on the subject in prominent medical journals from the 1980s. The ignorance was that a dextrose-saline (0.18% saline) solution while being a reasonable choice for use strictly as a maintenance fluid, cannot be used to replace abnormal losses such as bleeding or diarrhoea and vomiting in which the losses are primarily sodium chloride.

CROSS Billy

From: esumner [redacted]
Sent: 13 February 2006 11:20
To: CROSS Billy
Subject: Re: Meetings on 22-23 February etc.

Yes - just confirm nearer the time - Ted Sumner
----- Original Message -----
From: <Billy.Cross[redacted]
To: [redacted]
Sent: Monday, February 13, 2006 7:53 AM
Subject: RE: Meetings on 22-23 February etc.

Dr Sumner

Yes, we are staying at the Hotel Russell in Russell Square, same location as consultation on 23rd. Would 1400 hrs on 22nd or any time thereafter suit?

lly Cross

-----Original Message-----
rom: esumner [mailto:[redacted]
Sent: 12 February 2006 12:51
To: CROSS Billy
Subject: Re: Meetings on 22-23 February etc.

Thanks - do you think we could meet at your hotel if you tell me where it is?
Ted Sumner
----- Original Message -----
From: <Billy.Cross[redacted]
To: [redacted]
Sent: Friday, February 10, 2006 11:07 PM
Subject: Meetings on 22-23 February etc.

Dear Dr Sumner,

an I finalise now our arrangements for the meeting with myself and DC PJ naghan on the afternoon of 22 February? Our flight lands at Gatwick at 1020 so I expect to be checked in at the Hotel by 1230. We would be free to eet thereafter at a time and location which you advise.

The consultation at 0900 on the 23 February will take place at the Hotel Russell, Russell Square.

I have some documents for your consideration before our meeting on 22 February. I will put them in the post on Monday morning. There will be a covering letter which raises a few questions proposed by an analyst who has studied the medical notes and your reports. We can discuss your response on 22 February.

Thanking you,

Billy Cross

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presence of viruses.

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No virus found in this incoming message.
Checked by AVG Free Edition.
Version: 7.1.375 / Virus Database: 267.15.5/256 - Release Date: 10/02/2006

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Checked by AVG Free Edition.
Version: 7.1.375 / Virus Database: 267.15.6/258 - Release Date: 13/02/2006