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 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

AS – CORONER

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 SUMNER

of

(Address)

who being sworn upon his oath, saith

Blood gas should have been taken as soon as Adam was on the operating table. He was a sick child but relative to other children on a renal transplant programme he was relatively healthy. I believe the mechanism 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 brain is more sensitive to oedema than other organs. The impaired blood flow ~~to~~ from the ~~head~~ 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 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.

AS – CORONER

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P.T.O.

Miss Huggins: One member of the anaesthesia team would see the parent in St. Omond 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 exercise + Adams 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. Tunnelling the lead may have occluded the external jugular vein. Drainage may have been impaired without one running 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 the third for infusion of drugs. It is not interrupted. Blood gases are measured by a machine or at the bed (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 operation determines the frequency of doing this. In a 6 hr operation — 4 only; 4 hrs — 3: if sodium falls below 128 that is hyponatraemia &

TAKEN before me this 18th day of June 1996,

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 GUNNER of

(Address)

who being sworn upon h[illegible] oath, saith

~~There~~ the 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 Ariff paper. Hyponatraemia is more difficult to diagnose ~~on~~ during anaesthesia — it can mask the signs. I believe that with only the wrong drainage fluid 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 Arrowsmith'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

sodium
indicated not enough red cells being given
Stability insufficient
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 are given 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.
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.

TAKEN before me this 18th day of June 1996

Coroner for the District of Greater Belfast

No. 20

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 SUMNER

of

(Address)

who being sworn upon his oath, saith

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

E Sumner.

TAKEN before me this 18th day of June 1996,

Coroner for the District of Greater Belfast

AS – CORONER

011-011-048

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.

Great Ormond Street Hospital for Children NHS Trust

and the Institute of Child Health

The child first and always

Great Ormond Street
London WC1N 3JH

Telephone: [redacted]

ES/DD

22 January 1996

Mr John L Leckey LL.M.
HM Coroner for Greater Belfast
Coroner's Office
Courthouse
Crumlin Road
Belfast
N Ireland
BT14 6AL

Dear Sir

re: Adam Strain, deceased.

Thank you for asking my opinion on this sad case. I enclose my report. The medical and nursing notes I will return under separate cover.

I also enclose a copy of my CV, together with my account for your kind attention in due course.

If you require more information or wish for any points to be clarified, please do not hesitate to ask.

Yours sincerely

E Sumner
Consultant Paediatric Anaesthetist

H.M. CORONER'S OFFICE
26 JAN 1996
GREATER BELFAST

DEPARTMENT OF ANAESTHESIA:
Direct line: [redacted] Fax: [redacted]

011-011-051

AS – CORONER

[redacted]

Adam Strain

Dr. E. Sumner

Anaesthetist

presents his Compliments and his Fees

for Professional Services.

Medico legal Report.

[redacted]

AS – CORONER

011-011-052

C3

MEDICAL REPORT

ON

ADAM STRAIN (DECEASED)

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

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

22 January 1996

Thank you for asking my opinion on this case. I have been a consultant paediatric anaesthetist at Great Ormond Street since 1973, with a particular interest in paediatric intensive care. I am the author of several textbooks on the subject and am the Editor-in-Chief of the journal, Paediatric Anaesthesia. For the preparation of this report I have carefully perused the recent medical and nursing notes, but realize, because of Adam's previous medical history there are several older bundles of notes.

Adam was born on 4.8.1991 with vesico-ureteric reflux causing repeated, damaging urinary tract infections. He had five operations for reflux ending up with one ureter connected to the other with only one draining into the bladder. He also had a fundoplication for gastro oesophageal reflux and marked vomiting. Nutrition was a problem and it became necessary to give him gastrostomy feeds. Eventually he refused all feeds and it is my understanding that he took nothing by mouth at all.

He gradually went into renal failure to the point that dialysis was commenced using the peritoneal route. Dialysis took place at night, but Adam also passed urine, presumably of a poor quality, and has been described as polyuric. However, he was generally progressing quite well having gastrostomy feeds of 3 x 200 ml Nutrizon during the day and 1500 ml at night, i.e. a total volume of 2100 ml per day. He was on the 50th centile for height but on the 95th for weight. In July 1995 he was admitted for a pyrexial illness which was extensively investigated and was

probably an infected gastrostomy site. On 14th July he was given a blood transfusion. At the time leading up to his renal transplant in November 1995, he was taking Keflex, Fersanel, vitamin D, bicarbonate and erythropoietin in addition to his feeds and dialysis regime.

He was not hypertensive as his blood pressure on 18.10.95 was 106/61 when he had his orchidopexy and on 26th November, when admitted for the transplant the following day, the BP was 108/56.

The renal transplant took place on 27.11.1995 beginning at 07.00, the anaesthetist being Dr Taylor and the surgeons Mr Keane and Mr Brown. Adam weighed approximately 20 kg, had a haemoglobin of 10.5 g/dl with reasonable electrolytes (urea 16.8, but sodium 139) at 11 pm on 26/11. Overnight he was given 900 ml Diorolyte (4% dextrose, .18% saline) via the gastrostomy, instead of his feed, but nothing for the two hours leading up to anaesthesia. P.D. was as usual. I can find no note of how much urine per hour he was passing nor of any electrolyte results just prior to anaesthesia.

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

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

There was considerable blood loss - in excess of 1100 ml as the operation was slightly more difficult than usual because of all the previous surgery. The systolic blood pressure started at 85 - 90 mm Hg and gradually rose, according to the charting, to 120, whereas the pulse rate started high (145/min) presumably because of the IV atropine and gradually fell, dipping to 80/min around 09.30. There are no entries in the space available on the anaesthesia record for central venous pressure measurements. Body temperature was well maintained.

Administered fluids were, dextrose-saline (4% and .18%) 1000 ml from 07.00 - 08.30 and a further 500 ml thereafter, 500 ml Hartman's solution, 1000 ml albumin and 500 ml of packed cells. A blood gas result taken at 09.32 showed mild hypoventilation with $PaCO_2$ 44 mm Hg (normal 40), very low sodium of 123 mmol/l (normal 135 - 145) and a very low haematocrit of 18% (normal 35 -

40%). I could find no note of an earlier result. There is no note of urine output during the case - there is note of a suprapubic catheter, but I do not know whether this was in use in the theatre.

At the end of the procedure, around 11.00 am, Adam was given neostigmine and glycopyrrolate to reverse the neuromuscular blockade, but he did not breathe and was found to have fixed dilated pupils and bilateral papilloedema with haemorrhages. He had obviously suffered a major cerebral insult. On the ICU he was hypertensive, requiring nifediprine to control this. He was described as 'puffy' and he had some pulmonary oedema. He was appropriately treated with mannitol and hyperventilation in an attempt to shrink the brain, but a CT scan showed severe cerebral oedema with obliteration of the ventricles and the neurologists confirmed that his signs were compatible with brain stem death, i.e. he had coned. Electrolyte results from 27/11 (not timed) showed a sodium of 119 mmol/l. A chest x-ray showed that the triple-lumen central venous line was going up into the neck vessel. Adam died the following day.

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

I would like to make the following comments:

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

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

3. Nowhere could I find a note of how much urine Adam was passing even though he was described as 'polyuric'. However, he was in a stable state for several weeks, growing and gaining weight. He was given 2100 ml per day of feed, i.e. approx 100 ml/kg/day - 4 ml/kg/hour - in addition to this there would be some water of oxidation of the nutrients in the diet. In a stable state intake equals output and his output in urine, sweat, respiration must equal 2100 ml, in addition to this there would be some volume taken off by the PD. As he was passing urine, the PD would be mainly for electrolyte exchange -K+, urea, etc., but could be in the order of 1-200 ml per day in total. I do not think his urine output could therefore be more than 1500 ml per day, i.e. 75 ml/kg/day - 3.5

ml/kg/hour on average.

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

In cases of renal transplant it is usual to be generous with fluids to maintain a CVP of 10 - 12 to optimize perfusion of the new kidney and to establish its urine-producing function. I think Dr Taylor overestimated the deficit somewhat, but was reasonable in suggesting 150 ml/hour for maintenance, but in fact he gave 500 ml D/S in just 30 minutes (07.00 - 07.30) and a further 500 ml over the next hour of a hypotonic solution - on top of the 900 ml that Adam had been given overnight. A further 500 ml

over 2½ hours is also greater than his calculations. Up to 09.30 he was given 800 ml plasma and 500 ml Hartman's solution for replacement of blood loss. I am assuming that the bleeding was steady, with the odd bigger loss and if Hartman's is used for blood volume replacement, twice the volume as loss is required, Adam was thus given volume replacement by 09.30 of 1050 ml for a total blood loss over four hours of 1100+ ml. It should be noted that plasma is also low in sodium.

4. I think it was unwise not to have electrolyte values taken before going to theatre and after the PD had been completed. It might be that the serum sodium was already low at that stage. It is also strange that the first blood gas was not taken until 09.32 when Adam was already severely hyponatraemic and diluted (haematocrit 18) from a combination of excess crystalloid and blood loss. Arterial access had been gained early in the case and it seems logical to analyze the blood for gases and electrolytes as soon as the patient is put on the table. There is no note of urine output during the case.

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

other signs of dehydration, such as cold peripheries are present. Cannulation of the right subclavian was achieved, but on subsequent chest x-ray the tip was found to be lying in a neck vein, rather than in the right atrium of the heart. Unfortunately, this is not an uncommon occurrence especially when the venous anatomy is deranged from multiple previous usage. My own philosophy is that while it is possible to freely aspirate blood, it can be used on a temporary basis, but should be changed at the earliest opportunity. It is not routine practice to x-ray for these lines when they are put in in the anaesthetic room prior to surgery. It is possible that the venous drainage from the head was not completely normal. Dr Taylor did not chart any CVP measurements and all the information on this I have from his letter. There were obvious problems with CVP readings. It is advisable to attach the pressure transducers to the side of the operating table so that when this is raised and lowered as it so often is during surgery, the zero is not changed. If the transducer is correctly put at zero, there is free flow of blood in and out of the central line, cardiac and respiratory patterns to the waveform then, in my opinion, the reading is correct. I do not agree with Dr Taylor that 'from the pressure reading I concluded that the tip of the line was not in close relation to the heart.' I believe that the pressure of 17 mm was the actual reading at the tip of the catheter. This is a high reading and the rise to 20 - 21 mm Hg is very high and actually difficult to achieve in a

child because the venous system (including the liver) is incredibly distensible. With hindsight, knowing that the tip of the catheter was up in the neck, these high figures for venous pressure imply there was some degree of obstruction to venous drainage from the head and with the knowledge that the left internal jugular vein had been tied off. This was possibly caused by having the head turned to one side as is usual in theatre, as the CVP came down to 10 - 12 in the ICU with the head in the neutral position. If gross obstruction to the venous flow had been present the head would have been suffused and swollen as suggested by Dr Taylor in his letter. However, Adam was described as 'puffy' by the ICU staff.

6. It is very interesting to have the monitoring data printed out from the machine. I assume that for the systemic blood pressure with a range of 200 mm Hg, the half-way line is 100 mm Hg. The trace shows much more clearly than Dr Taylor's anaesthetic record the consistent rise in BP from around 09.30, i.e. soon after the blood gas was drawn, peaking at 150 mm Hg. The pulse rate also rose steadily from 10.15 onwards. Again, with hindsight these could represent the cardiovascular changes of a coning patient under anaesthesia. The arterial trace shows that the line was not interrupted for sampling until just after 09.30.

7. Blood transfusion is usually given to patients who are losing in excess of 15 - 20% of the blood volume (i.e. 250

- 300 ml in Adam's case). Until that point is reached volume is replaced using plasma and/or Hartman's. I think they were rather late in starting the blood transfusion as the haematocrit at 09.30 had fallen to 18% (normal 40). Overall, however, the haemoglobin was well managed as the result at the end of the case was 10 g/dl.

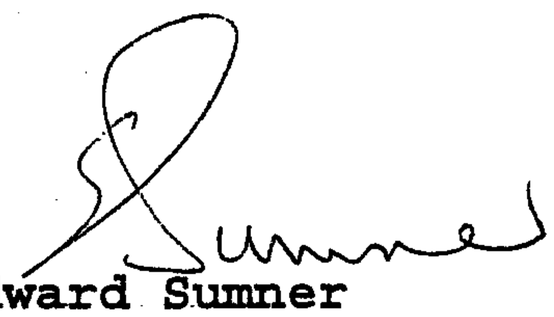
8. Dr Taylor suggests that cerebral oedema is difficult to explain because both thiopentone and methyl prednisone had been given albeit for other reasons. While methyl prednisolone is often given as a cerebral protector, for example for patients going on cardiopulmonary bypass, there is no hard data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

To summarize, I believe that on the balance of probabilities Adam's gross cerebral oedema was caused by the acute onset of hyponatraemia (see reference) from the excess administration of fluids containing only very small amounts of sodium (dextrose-saline and plasma). This state was exacerbated by the blood loss and possibly by the overnight dialysis.

A further exacerbating cause may have been the obstruction to the venous drainage of the head. If drugs such as antibiotics were

administered through a venous line in a partially obstructed neck vein then it is possible that they could cause some cerebral damage as well.

Ref: Arieff AI, Ayus JC, Fraser CL. Hyponatraemia and death or permanent brain damage in healthy children. BMJ 1992, 304: 1218-1222.



Edward Sumner

22 January 1996

CURRICULUM VITAE

OF

EDWARD SUMNER, MA BM BCh FRCA

FEBRUARY 1995

NAME: Edward Sumner

ADDRESS: [redacted]

DATE OF BIRTH: 17.8.1940

MARITAL STATUS: [redacted]

DEGREES HELD:

MA, BM, BCH Oxford 1966
FRCA London 1971

ACADEMIC DISTINCTIONS:

First Class Honours Degree
Animal Physiology
Oxford 1963

Nuffield Prize
Primary Fellowship
London 1969

DATE OF MEDICAL REGISTRATION: March 1968

PRESENT APPOINTMENT:

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

Appointed September 1973

Director of the Department of Anaesthetics : 1987 - 1992

Director: Cardiac Intensive Care Unit : 1988 - 1993

Honorary Senior Lecturer : University of London

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

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

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

PREVIOUS APPOINTMENTS:

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

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

TEACHING EXPERIENCE:

Teaching in the Institute of Child Health, London:

Annual Advanced Course in Paediatrics

Annual Course in Paediatric Intensive Care

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

Annual St Bartholomew's Hospital Final Fellowship Course.

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

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

Presented papers at the Association of Paediatric Anaesthetists:

London 1974 : Mass Spectrometry - Clinical Applications.

Newcastle 1976 : Wilson-Mikity Syndrome

Edinburgh 1982 : Congenital Diaphragmatic Hernia

Dublin 1985 : Management of Phrenic Palsy in the Infant.

London 1988 : Analgesia for Newborns

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

Continuing Medical Education Day - 1991, 1993, 1994

1982 European Congress, London.

Paper - Congenital Diaphragmatic Hernia
Poster - Caudal Analgesia

Royal Society of Medicine

1982 - Congenital Diaphragmatic Hernia
1982 - Paediatric Anaesthesia Symposium

1980 Stockholm, Sweden : Paediatric mechanical ventilation.

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

1982 Bonn, Germany - Cardiac anaesthesia.

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

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

Prague 1983 - Congenital Diaphragmatic Hernia.

Royal College of Surgeons, London: Symposia:

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

Paris 1983 - Congenital Diaphragmatic Hernia.

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

Lectures at Intensive Care Meetings:

Birmingham 1984
Rotterdam 1984
Tubingen 1984

Lectured at Thoracic Anaesthetic Meeting, London 1983, 1984.

Open Heart Surgery Congress - Bombay 1985.

Pulmonary Hypertensive Crisis - Paediatric Intensive Care. Brussels 1985.

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

Association of Anaesthetists, London:

1986 - Neonatal Analgesia
1987 - Cyclopropane

Neonatal Anaesthesia - Gothenburg, Sweden : 1986

Neonatal Anaesthesia - Oslo, Norway : 1986.

Paediatric Anaesthesia, Basel, Switzerland : 1987, 1994.

Lectures:

Barbican - ICU Update
September 1991

: Respiratory Support in Paediatrics

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

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

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

Munich
July 1992
: Neonatal anaesthesia

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

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

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PUBLICATIONS:

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EDITORIAL DUTIES:

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LEARNED SOCIETIES:

Association of Anaesthetists of the United Kingdom.

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European Society of Anaesthesiology.

RESEARCH INTERESTS:

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

Popularised - epidural analgesia in infants and children.

- axillary artery cannulation in infants.

Projects include - the pulmonary circulation

- gastro oesophageal reflux.

MEDICO-LEGAL WORK:

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

toms. Home monitoring of blood glucose concentrations is economically impracticable for most patients, but easier access to urine dipsticks would probably increase patients' interest and motivation in improved control and would not add greatly to total direct costs.

The need for inpatient admission should also be considered carefully, especially for newly presenting patients. Wherever possible admission is best avoided if the patient and family are able to receive initial daily outpatient education and supervision.[15] Patients should be admitted only if they require nursing care or circumstances do not permit easy attendance at outpatient clinics. Admission rates for diabetic patients in Tanzania are six times higher than in the general population.[16] When patients are admitted careful consideration should be given to the need for investigations. Testing urine four times or more daily for example, may be unnecessary if blood glucose concentrations are also being measured. Consideration should also be given to the period of admission since patients are often kept in the wards until most urine results are glucose free.

The small proportion of direct costs due to nurses' and doctors' services reflects the low rates of pay of medical staff in most sub-Saharan countries. A lecturer in medicine, for example, is paid $60 monthly. The reasons for such low rate of remuneration are understood, but attention must also be paid to this problem since the motivation and interest of those caring for patients can have a significant impact on the quality of care.

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Hyponatraemia and death or permanent brain damage in healthy children

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Abstract

Objective—To determine if hyponatraemia causes permanent brain damage in healthy children and, if so, if the disorder is primarily limited to females, as occurs in adults.

Design—Prospective clinical case study of 16 affected children and a review of 24 412 consecutive surgical admissions at one medical centre.

Patients—16 children (nine male, seven female; age 7 (SD 5) years) with generally minor illness were electively hospitalised for primary care. Consultation was obtained for the combination of respiratory arrest with symptomatic hyponatraemia (serum sodium concentration ≤128 mmol/l).

Main outcome measures—Presence, gender distribution, and classification of permanent brain damage in children with symptomatic hyponatraemia in both prospective and retrospective studies.

Results—By retrospective evaluation the incidence of postoperative hyponatraemia among 24 412 patients was 0·34% (83 cases) and mortality of those afflicted was 8·4% (seven deaths). In the prospective population the serum sodium concentration on admission was 138 (SD 2) mmol/l. From three to 120 inpatient hours after hypotonic fluid administration patients developed progressive lethargy, headache, nausea, and emesis with an explosive onset of respiratory arrest. At the time serum sodium concentration was 115 (7) mmol/l and arterial oxygen tension 6 (1·5) kPa. The hyponatraemia was primarily caused by extrarenal loss of electrolytes with replacement by hypotonic fluids. All 16 patients had cerebral oedema detected at either radiological or postmortem examination. All 15 patients not treated for their hyponatraemia in a timely manner either died or were permanently incapacitated by brain damage. The only patient treated in a timely manner was alive but mentally retarded.

Conclusions—Symptomatic hyponatraemia can result in a high morbidity in children of both genders, which is due in large part to inadequate brain adaptation and lack of timely treatment.

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Introduction

In previous studies from our laboratories we have described the symptomatology, clinical course, effects of treatment, and pathological findings in more than 225 adults (aged over 16) with symptomatic hyponatraemia.[1-8] Although the actual incidence of hyponatraemia seems to be similar among men and women,[8,9] almost all adult patients suffering hyponatraemic brain damage are women. Although there are a number of reported paediatric cases of hyponatraemia,[10-12] there are few reported cases of death or permanent brain damage among children with this disorder,[13,14] and most such children had pre-existing neurological disorders.[15-17] Neither the gender distribution nor the incidence of brain damage among children with hyponatraemia is known.[10,12-17] Among children suffering brain damage from hyponatraemia neither the type nor the gender distribution is known. We describe both a prospective and a retrospective analysis of generally healthy children who were elect

ively hospitalised. Sixteen children who developed severe symptomatic hyponatraemia either died or suffered permanent brain damage. Unlike the situation in adults, both males and females were adversely affected among these children.

Patients and methods

Prospective studies—Over a period of six years (1984-90) we were consulted about 16 previously healthy children (aged under 16) who had developed symptomatic hyponatraemia and either died or suffered permanent brain damage. These 16 patients were seen in consultation from five tertiary and nine community hospitals. The age of the children was 7 (SD 5) years (range 1·5 to 15 years), and the gender distribution was nine males and seven females. The mean weight was 23·8 (12·9) kg (range 10 to 52 kg). Symptomatic hyponatraemia developed within five days of admission to the hospital.

Epidemiological studies—We retrospectively studied all surgical admissions to a 456 bed tertiary paediatric university teaching hospital over three years (1989-91). The records of all paediatric (age under 16) surgical patients were evaluated for those who had postoperative hyponatraemia (serum sodium concentration 128 mmol/l or less) and the number who either died or suffered permanent brain damage as a result of the hyponatraemia. The epidemiological data were generated by computer search of the hospital records using the SAS database[18] to obtain information on all paediatric surgical patients who had a postoperative serum sodium concentration of 128 mmol/l or less. There were 24 412 consecutive inpatient operations over the three years ended 31 December 1991. In addition, we calculated an approximation of the incidence of hyponatraemic brain damage in children in the United States from our epidemiological data plus a statistical database from the medical literature.[19 20]

Results

STUDY PATIENTS

The table shows the clinical circumstances which resulted in hospitalisation of the 16 patients. All data are presented as means (SD). Symptoms were not known in three patients, who were either too young (less than 18 months) or intubated and thus unable to vocalise any complaints. Of the remaining 13 patients, 11 had progressive lethargy, weakness, nausea, and emesis and 12 had headache. All patients suffered respiratory arrest after a mean of 37 hours (range three to 120 hours) from the start of intravenous fluid administration.

CLINICAL COURSE

At admission the serum sodium concentration was 138 (2) mmol/l. As early as two hours after starting hypotonic fluid administration those patients able to communicate became progressively more lethargic and complained of headache and nausea, with subsequent emesis. All such symptoms were generally unresponsive to conventional agents (phenothiazines and narcotics). After a mean of 37 hours all 16 patients suffered respiratory arrest, at which time the serum sodium concentration was 115 (7) mmol/l and urine osmolality 676 (66) mmol/kg. This level of urine hypertonicity in the presence of hyponatraemia suggests that the plasma antidiuretic hormone concentration was raised.[21] The onset of respiratory arrest was often explosive in nature, and hyponatraemia was generally not considered as a possible cause.

Immediately after respiratory arrest but before oxygen administration or intubation the arterial oxygen tension was evaluated in 11 patients and was 6·0 (1·5) kPa. During the 37 hours between the time of admission and onset of respiratory arrest the patients had received a mean of 125 (83) ml hypotonic intravenous fluids per kg daily. Urine output was 34 (34) ml/kg per day and other fluid losses averaged 28 (25) ml/kg per day (nasogastric suction, n=2; emesis, n=10; cerebrospinal fluid drainage, n=1; not charted, n=3) with mean net output of 74 (82) ml/kg daily and net positive fluid balance of only 27 (14) ml/kg per day. Hyponatraemia in these children was thus largely due to extensive extrarenal loss of electrolyte containing fluids with replacement by hypotonic fluids. Most of the intravenous fluids were administered as 280 mmol glucose per litre either in water or in sodium chloride 38 mmol/l, but the plasma glucose concentration was

racteristics of 16 children with symptomatic hyponatraemia

asc o	Gender and age (years)	Weight (kg)	Serum sodium (mmol/l)		Duration of intravenous fluid treatment (hours)	Net fluid intake (ml/kg)	Net fluid output (ml/kg/*	Clinical history	Hospital procedures	Respiratory arrest	Treatment after respiratory arrest	Clinical outcome
			Initial	Lowest								
	M 3·5	2·27	139	114	46	246	222	Fever, dysphagia, pharyngitis, tonsilitis	Antibiotics+fluids	Yes	154 mM sodium chloride	Vegetative, quadriplegia
	F 5	18·0	141	123	14	96	33	Tonsillitis	Tonsillectomy	Yes	None	Died
	F 4	18·2	139	115	21	114	NA	Tonsillitis	Tonsillectomy	Yes	None	Died
	M 15	44·6	134	101	74	164	73	Fever, dysphagia, pharyngitis, tonsilitis	Antibiotics+fluids	Yes	154 and 514 mM sodium chloride	Aspiration pneumonia, sepsis, died
	M 3·5	15·0	138	121	9	61	5	Tonsillitis	Tonsillectomy	Yes	None	Died
	F 12	31·8	137	120	33	57	11	Elbow fracture from car accident	Setting of fracture	Yes	514 mM sodium chloride; intubation	Ambulatory, mental retardation
	M 4	16·4	139	118	27	109	88	Elbow fracture from fall	Setting of fracture	Yes	None	Died
	M 3	10·0	137	113	8	300	NA	Stricture of urethra; tonsillits	Urethral dilatation; tonsillectomy	Yes	None	Died
	F 1·5	10·6	137	114	120	283	253	Hydrocephalus	Ventriculoperitoneal shunting	Yes	None	Vegetative
	M 9	27·0	137	120	32	79	NA	Fractures from car accident	Operative setting of fractures	Yes	None	Vegetative
	F 15	52·0	138	102	94	87	57	Fractures from car accident	Operative setting of fractures	Yes	154 mM sodium chloride; intubation	Vegetative and blind
	F 4	16·8	138	107	16	88	56	Tonsillitis	Tonsillectomy	Yes	None	Died
	2	11·4	138	116	3	123	NA	Undescended testicle	Orchiopexy	Yes	None	Died
		15·0	138	119	12	40	11	Severe epistaxis	Posterior packing	Yes	None	Died
	.2	42·0	137	123	19	34	9	Fever, appendicitis, ruptured appendix	Appendicectomy plus drainage	Yes	None	Died
	F 12	28·5	134	116	66	113	72	Pneumonia	Antibiotics+fluids	Yes	None	Vegetative
an	7	23·8	138	115	37	125	74					
	5	12·9	2	7	34	83	82					
	1	3·2	1	2	9	21	24					

ine + emesis + gastric drainage + cerebrospinal fluid. NA = Not available.

diagnosed. Four patients (two male, two female) subsequently developed the syndrome of central diabetes mellitus and central diabetes insipidus[5] with hypotonic polyuria. In these four patients the mean serum sodium concentration rose (without treatment) from 114 (6) mmol/l to 164 mmol/l and the glucose concentration to 31·1 mmol/l. None of these patients had been treated for their hyponatraemia.

OUTCOME

All 16 patients either died or suffered permanent brain damage (table): one was mentally retarded, 10 died, and five were in a persistent vegetative state which persisted for follow up intervals of at least two years. Twelve patients received no specific treatment for their hyponatraemia. Of these, nine died and three remained in a persistent vegetative state.[22] Four patients were eventually treated with intravenous sodium chloride 154 and 514 mmol/l (table) such that the serum sodium concentration was increased from 108 (9) to 138 (4) mmol/l in 44 hours. The average delay from respiratory arrest to start of treatment was eight hours, all four patients were comatose, apnoeic, and intubated at the time treatment was begun, and none awoke either during treatment or for three days thereafter. Only one patient (case 6), who survived mentally retarded, was treated within 10 minutes of respiratory arrest.

NECROPSY FINDINGS

Postmorten examination of the brain was performed in 10 patients (three girls, seven boys). In nine patients who had received no treatment and died in less than 48 hours there was cerebral oedema and herniation on gross examination of the brain. The brain weight (unfixed) in six patients (three male, three female) whose mean age was 3·8 years was 1354 (95) g. For comparison, the normal brain weight in men is 1450 g, in women 1250 g, in 4-5 year old boys 1300 g, and in 4-5 year old girls 1150 g.[23] Thus brain weight was increased by more than 10% above control values for children of the age range studied.[23] That transtentorial herniation was present in all nine patients subjected to postmortem evaluation correlates well with the observation that the human brain can expand by only about 5-7% of its normal volume[24] before herniation occurs. We have shown that men's brains can usually adapt to hyponatraemia within a few hours whereas women's brains may not adapt within several days.[8] In all 16 children presented here the brains were unable adequately to adapt to hyponatraemia.

EPIDEMIOLOGICAL FINDINGS

Among 24 412 paediatric surgical admissions to a 456 bed university paediatric hospital there were 83 (0·34%) patients who developed hyponatraemia. Among these, seven (8·4%) died of complications of the hyponatraemia. Among the seven deaths, four were in boys and three in girls. Hence the incidence was 340 cases of paediatric postoperative hyponatraemia and 29 hyponatraemic deaths per 100 000 inpatient operations on children. There are 2·02 million paediatric inpatient operations a year in the United States.[19 20 25] The estimated yearly incidence in the United States is 7448 cases of paediatric postoperative hyponatraemia, with 626 such hyponatraemic deaths in children. The most common inpatient operations on children in the United States[20] are to the nose, mouth, and pharynx (17%); digestive system (17%); musculoskeletal system (15%); and nervous system (13%), of which 43% are performed in girls. This was essentially the distribution in our series, in which 92% of operations were in these four groups and 44% of the patients were female (table).

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. The permanent brain damage can include pituitary infarction with resultant central diabetes insipidus and mellitus, a syndrome not previously described in children.[5] The incidence of postoperative hyponatraemia in children (0·34%) was less than in adults (1-4%).[8 21] However, among paediatric patients who developed symptomatic hyponatraemia the incidence of permanent brain damage was substantially higher than in adults.[8 21] Both the types of surgery and gender distribution among our 16 patients (table) were the same as the most common operations and gender distribution in the United States as a whole,[20] and thus our 16 patients were representative of the spectrum of elective paediatric surgical patients.

The hyponatraemia in these children seems to have been caused by extensive extrarenal loss of electrolyte containing fluids and intravenous replacement with hypotonic fluids (table) in the presence of antidiuretic hormone activity. Increased plasma concentrations of antidiuretic hormone are usually found in both children and adults with hyponatraemia,[9 12-14 16 26] and the hormone has multiple cerebral and vascular effects which can impair the ability of the brain to adapt to hyponatraemia.[27 28] However, the genesis of hyponatraemia in children is usually different from that in adults. In adults there has often been administration of very large quantities of intravenous fluid (net retention 63 ml/kg per day in adults *v* 28 ml/kg per day in children; $p<0·01$)[3 5] or diuretic induced loss of cations.[2 6 29] It is important to recognise that in children, when there is substantial extrarenal loss of electrolytes, a minimal positive balance of hypotonic fluid can lead to fatal hyponatraemia. Another major factor which may have contributed to the high morbidity among these children was the virtual absence of timely treatment in the presence of obvious symptoms.[10 11 16 17] Furthermore, the types of operations and the clinical conditions in this patient population were similar to those most common in the United States.[20] Thus the index of suspicion for electrolyte disorders in generally healthy children undergoing elective surgery may be quite low.

BRAIN ADAPTATION TO HYPONATRAEMIA IN CHILDREN

In adults oestrogens seem to impair the ability of the brain to adapt to hyponatraemia and androgens may augment such adaptation.[30-33] However, prepubescent children have only minimal to absent concentrations of either hormone, thus negating such effects. Most adults suffering permanent brain damage from hyponatraemia are female,[3 5 7 8] but in the current series a minority of affected patients (43%) in both the prospective and retrospective studies were female. Thus unlike the marked gender differential in adults, male and female children seem to be at similar risk of developing hyponatraemia encephalopathy (NS (χ^2 test)). Furthermore, neither the actual concentration of serum sodium nor the rapidity of development of hyponatraemia seemed to predict the ultimate outcome in these 16 children (table). Hyponatraemia developed over a mean of 37 hours and the range of serum sodium values was 101-123 mmol/l, values quite similar to those previously reported in children with symptomatic hyponatraemia who did not develop brain damage.[10 12 13 16]

EFFECTS OF PHYSICAL FACTORS

When hyponatraemia was present all 16 children had radiological evidence (computed tomography, magnetic resonance imaging) of cerebral oedema

whereas at necropsy nine of 10 evaluated had cerebral oedema with herniation. These findings show that adequate adaptation of the brain to hyponatraemia had not occurred. There are several unique characteristics of the paediatric central nervous system which may impair the ability to adapt to hyponatraemia. Such characteristics may include physical factors resulting from differences in the ratio of intracranial capacity to brain size, cerebrospinal fluid volume, and brain water and electrolyte content.

The early adaptation of brain to hyponatraemia involves a loss of blood and cerebrospinal fluid followed by extrusion of sodium from brain cells.[34 35] Later adaptation includes loss of potassium and possibly amino acids, which act further to decrease brain cell osmolality and limit the gain of water.[1 34] In humans and laboratory animals brain water content is more than 2·5 times higher in the young, decreasing progressively with age.[36-38] In children the ratio of brain to skull size is such that there is less room for expansion of the paediatric brain in the skull than there is in adults.[39] As adults age there is a progressive decline in the brain volume whereas skull size remains constant.[39] Hence anatomically there is decreased room for expansion of the brain within the skull in children as compared with adults.[23]

Adult brain size is reached at about age 6 whereas full skull size is not reached until age 16. Additionally, the intracerebral volume of cerebrospinal fluid is more than 10% greater in adults than in the young.[39] When brain swelling occurs the intracerebral loss of cerebrospinal fluid increases the available volume in which the brain can expand.[35 40] As the percentage of cerebrospinal fluid in the brain increases with age[38 39] adults of both genders have more room in the rigid skull for the brain to expand than do children.[39] Furthermore, the brain intracellular concentration of sodium is about 27% higher in children than in adults[37] and may reflect a relative decreased ability to pump sodium out of the brain in children. In the presence of hyponatraemia this will result in a greater osmolar gap between brain and plasma in the young. It has been shown that in newborn puppies with hyponatraemia the brain is unable to extrude cations[38] whereas adult animals with hyponatraemia can readily transport sodium out of the brain.[1 31 34]

PREVENTION AND TREATMENT OF HYPONATRAEMIC ENCEPHALOPATHY

Symptomatic hyponatraemia can best be prevented by not infusing hypotonic fluids to hospitalised children unless there is a clear cut indication for their use. Headache, nausea, emesis, weakness, and lethargy are consistent symptoms of hyponatraemia in children. If the condition is allowed to go untreated there can follow an explosive onset of respiratory arrest, coma, and transtentorial cerebral herniation. At present there is no way to predict which children may suffer respiratory arrest. As found recently in adults neither the magnitude of hyponatraemia nor its duration is the major determinant of brain damage.[8] Recent studies show that recovery from symptomatic hyponatraemia in children, even after the onset of seizures and apnoea, may be possible if appropriate treatment is instituted in a timely manner.[11]

When a paediatric patient receiving hypotonic fluids begins to have headache, emesis, nausea, or lethargy the serum sodium concentration must be measured. Although these symptoms are somewhat non-specific, the diagnosis is easily established at minimal cost and with virtually no risk to the patient by evaluating plasma electrolyte values. When symptomatic hyponatraemia is diagnosed the patient should be moved to a location where constant monitoring can be provided, such as the intensive therapy unit. Hypertonic sodium chloride (514 mmol/l) should be infused as described,[4 11 41] such that the serum sodium concentration is increased to 125-130 mmol/l but by no more than 25 mmol in the initial 48 hours. In addition to hypertonic sodium chloride, treatment may include intubation and assisted mechanical ventilation when required.

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Addendum

After submission of this paper a report appeared describing 34 paediatric patients with water intoxication.[42] Two of the patients became hyponatraemic secondary to intravenous hypotonic fluid administration (serum sodium concentrations 112 and 114 mmol/l). Both suffered respiratory arrest and died, and at necropsy both had cerebral oedema. These two patients had a clinical course similar to the 16 in our series. The other 32 patients had oral water intoxication, and all survived because of timely and appropriate treatment.

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First use of heroin: changes in route of administration over time

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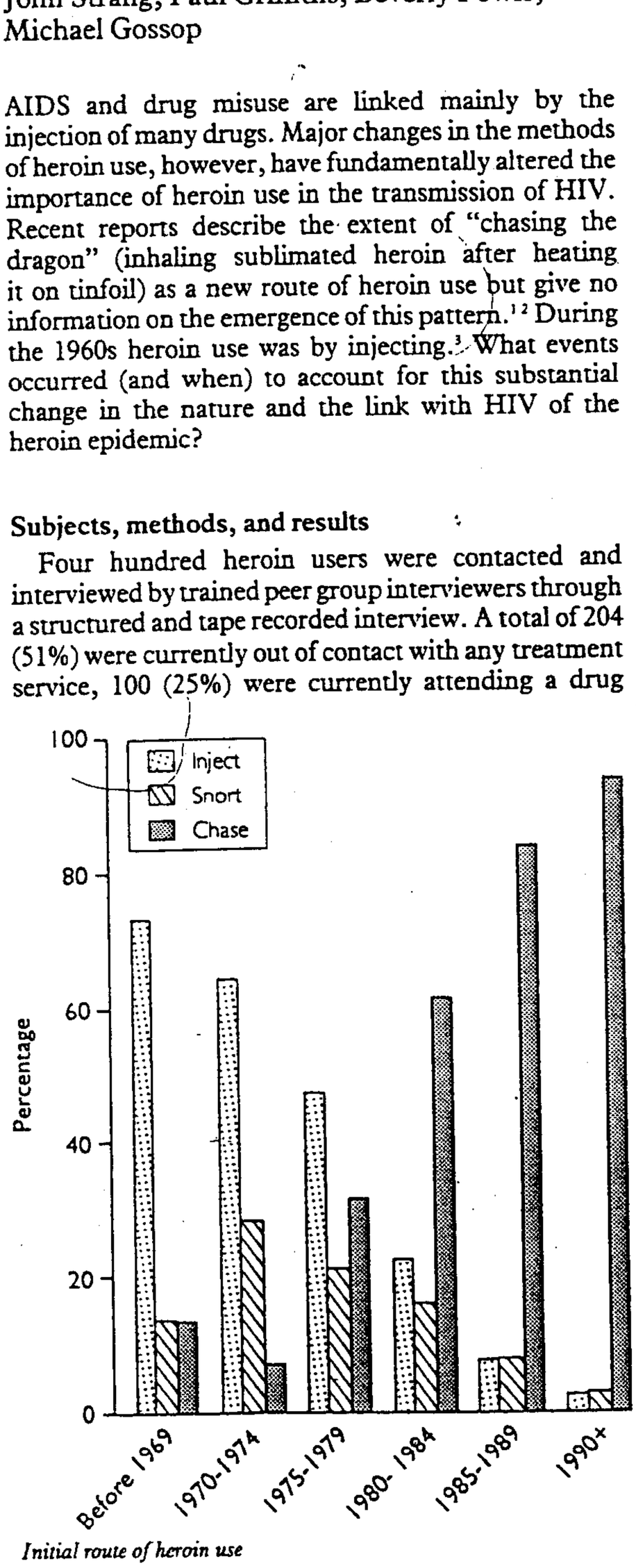
AIDS and drug misuse are linked mainly by the injection of many drugs. Major changes in the methods of heroin use, however, have fundamentally altered the importance of heroin use in the transmission of HIV. Recent reports describe the extent of "chasing the dragon" (inhaling sublimated heroin after heating it on tinfoil) as a new route of heroin use but give no information on the emergence of this pattern.[1,2] During the 1960s heroin use was by injecting.[3] What events occurred (and when) to account for this substantial change in the nature and the link with HIV of the heroin epidemic?

Subjects, methods, and results

Four hundred heroin users were contacted and interviewed by trained peer group interviewers through a structured and tape recorded interview. A total of 204 (51%) were currently out of contact with any treatment service, 100 (25%) were currently attending a drug clinic, and 124 (31%) were currently attending a needle exchange scheme. A total of 136 (34%) had never had contact with either treatment services or an exchange scheme. Their ages ranged from 17 to 53 (mean (SD) 27·6 (6·3) years); 248 (62%) were male; 96 (24%) were in current employment. There was wide variation in first year of use of heroin use (1954 to 1991): 16 (4%) started during the '60s, 28 (7%) during the early '70s, 76 (19%) during the late '70s, 124 (31%) during the early '80s, 120 (30%) during the late '80s, and 36 (9%) during the '90s.



Initial route of heroin use

Three different routes of initial drug use were identified: injecting, snorting, and "chasing the dragon." Analysis of these data by year revealed a major change in the annual proportion who were initiated by either injecting or chasing (figure).

"Chasing" was a route of initiation for a minority of users up to the late 1970s but has become an increasingly common route of initiation since 1975. By 1979 there were as many initiations by chasing as by injecting, and by 1981 more than half of the initiations into heroin use were by chasing (with the annual proportion remaining above half since 1981). By 1985 more than three quarters of initiations were by chasing, and since 1988, 87 out of 93 initiations (94%) were by chasing. During most years, a tenth to a quarter of users were initiated by snorting.

Comment

Heroin use today is not what it was yesterday. Initiation no longer occurs by injecting but by the new route of "chasing the dragon." The emergence of new non-injecting routes of heroin use may partly explain not only the major heroin epidemic in the United Kingdom during the 1980s but also its apparent continuation[4] despite the addition of AIDS as a potential consequence. Perhaps the protective societal taboo against injecting was circumvented and a less fettered epidemic has developed. In the 1990s virtually all initiations into heroin use in our London sample were by "chasing the dragon," even though heroin use in other countries (for example, the United States) and even in other British cities (for example, Edinburgh)[5] continues to be by injection. Should the change in London be regarded as an isolated development in a few "chasing" cities, or is it an indication of likely future changes on a wider scale? And what is the significance for tomorrow's prevention and treatment programmes?

Our level of ignorance about changing routes of drug administration is not only scientifically disturbing but also interferes with the development of prevention and treatment programmes. Effective primary prevention strategies depend greatly on the adequacy of knowledge about the gateways into drug use, and yet our understanding of the phenomenon is informed largely by