Witness Statement Ref. No. 059/2

NAME OF CHILD: Raychel Ferguson

Name: John Gordon Jenkins

Title: Dr.

Present position and institution:

Hon. Senior Lecturer in Child Health, Queen's University, Belfast (since retirement).

Previous position and institution:
[As at the time of the child's death]

Consultant Paediatrician (Antrim Area Hospital)/ Senior Lecturer in Child Health (Queen's University, Belfast)

Membership of Advisory Panels and Committees:
[Identify by date and title all of those since the date of your last statement]

1987 – 2010 Member of DHSSPS Paediatric Specialist Advisory Committee
1993 – 2003 Member of Clinical Resource Efficiency Support Team
1999 – 2003 Chairman of DHSPSS Hospital Services Subcommittee
2000 – 2004 Vice chairman of N. I. Council for Postgraduate Medical and Dental Education, and Member of the Executive, Audit, Education and Remuneration Committees
2000- 2008 Chairman of DHSSPS Implementation Support Group/Advisory Group on Junior Doctor's Hours
2000 – 2008 Member of the DHSSPS information and communications technology strategy implementation board, and Chairman of its advisory and quality assurance group from 2000 to 2003
2001 – 2004 Chairman, All-Ireland Committee of the Royal College of Paediatrics and Child Health (RCPCH)
2003 – 2010 Chairman of DHSSPS Central Medical Advisory Committee
July 2003 – December 2012 Council Member of General Medical Council (Chairman of Postgraduate Board, Chairman of Standards and Ethics Committee. Member of Education and Training Committee, Research and Development Board, Professional and Linguistic Assessments Board and liaison group with the Academy of Medical Royal Colleges. Represented GMC on other UK bodies, including the National Patient Safety Agency (NPSA) Medical Advisory Panel, BMA Ethics Committee)
September 2003 - April 2010 (when merged with GMC) Member of Postgraduate Medical Education and Training Board (PMETB) (Chair of Training Committee, Assessment Committee, Audit Committee)
October 2005 – March 2007 National Patient Safety Agency 'Safer use of hypotonic solutions in paediatrics' External Reference Group

2005 – 2007 President, Irish Perinatal Society

2005 – to date Member of DHSSPS Medical Education Policy Group

2006 – 2010 Member of DHSSPS Privacy Advisory Committee
2008 – to date Member of DHSSPS Clinical Excellence Awards Committee
2011 – to date Member of N.I. Medical and Dental Training Agency Foundation School Board

2012 – to date Member of DHSSPS Group B Streptococcal Steering Group

January – May 2013 Non-executive Board Member of N.I. Medical and Dental Training Agency
May 2013 – to date Non-executive Board member of Regulation and Quality Improvement Authority

Previous Statements, Depositions and Reports:
[Identify by date and title all those made in relation to the child's death]

022-010a-040 12.11.02 Report

OFFICIAL USE:
List of previous statements, depositions and reports attached:

Ref:	Date:	
012-023-132	30.01.03	Statement
012-030-153	05.02.03	Deposition at the Inquest into the death of Raychel Ferguson
059/1	01.07.2005	Inquiry Witness Statement

IMPORTANT INSTRUCTIONS FOR ANSWERING:

Please attach additional sheets if more space is required. Please identify clearly any document to which you refer or rely upon for your answer. If the document has an Inquiry reference number, e.g. Ref: 049-001-001 which is 'Chart No.1 Old Notes', then please provide that number.

If the document does not have an Inquiry reference number, then please provide a copy of the document attached to your statement.

(1) **Please provide the following information:**

(a) **Your qualifications as of 2001 (please also provide a copy of your CV);**

MB BCh BAO (Queen's University Belfast)	*1974*

Postgraduate degrees and diplomas

MRCP(UK)	*1977*
MD(Hons)	*1980*
FRCP(Edin)	*1989*
FRCPCH	*1997*

(b) **Describe your career history.**

Training posts

1 Aug 74 to 31 Jul 75	*House Officer in General Surgery, General Medicine and Paediatrics at Craigavon Area Hospital, Northern Ireland*
1 Aug 75 to 30 Sep 76	*Senior House Officer in Paediatrics at Royal Belfast Hospital for Sick Children*
1 Oct 76 to 31 Jan 77	*Locum Registrar in Paediatrics at Belfast City Hospital*
1 Feb 77 to 31 Jul 77	*Locum Registrar in Paediatrics at Royal Belfast Hospital for Sick Children*
1 Aug 77 to 31 Jul 78	*Registrar in Paediatrics at Ulster Hospital, Belfast*
1 Aug 78 to 30 Jun 80	*Senior Registrar and DHSS Research Fellow in Neonatal Intensive Care Unit, Royal Maternity Hospital, Belfast*
1 Jul 80 to 30 Jun 81	*Clinical Fellow in Intensive Care Unit, Hospital for Sick Children, Toronto*
1 Jul 81 to 30 Jun 82	*Clinical Assistant in Intensive Care Unit and part time Medical Supervisor in Emergency Department, Hospital for Sick Children, Toronto*
1 Jul 82 to 31 Aug 82	*Senior Registrar in Paediatrics, Royal Belfast Hospital for Sick Children*

Career posts

1 Sep 82 to 31 Oct 99	*Consultant Paediatrician, Northern Health and Social Services Board/United Hospitals HSS Trust, Northern Ireland - Waveney Hospital, Ballymena until March 1994, then Antrim Hospital*
1 Nov 99 to 31 Dec 2010	*Senior Lecturer in Child Health, Queen's University of Belfast (QUB) and Consultant Paediatrician, Antrim Hospital*

Management Appointments

1 Jan 94 to 31 Mar 96	*Clinical Director, Woman and Child Health Directorate, United Hospitals Group*
1 Mar 96 to 31 Dec 98	*Medical Director, United Hospitals HSS Trust*

(2) **In respect of the evidence provided by you to the Inquiry in Witness Statement 059/1:**

(a) **As to your reply to Question 1, why did you not make reference to the fact that you were briefed with the report of Dr. Warde prior to the preparation of your final statement?**

In Question 1 of Witness Statement 059/1, I was asked what information I had been provided with to prepare my statement (report) in relation to the death of Raychel Ferguson. Whilst I acknowledge that I should have made clear for the Inquiry that I did come into possession of Dr Warde's report and had commented on it in a letter to DLS (022-004-013 and 014), I was not, however, provided with Dr Warde's report in advance of the preparation of my report for DLS (022-010a-040). I believe, although I cannot be certain given the passage of time, that in answering Question 1 of Witness Statement 059/1, I referenced (and then summarised in my answer) the Schedule of Documents that had been sent to me by DLS on 1st November 2002 (160-115-001 and 002).

For the avoidance of doubt, Dr Warde's report was sent to me under cover of a letter from DLS dated 23rd January 2003 (160-045-001). I believe that I received this letter and report on Monday, 27th January 2003. I was asked to provide any comments that might assist the Trust urgently in view of the imminent Inquest (due to commence on 5th February 2003), which I did by letter on the same date (022-004-013 and 014).

(b) **Whether you agreed with the content of the draft Press Statement and did you think it was sufficiently accurate for release and, if not why not?**

I had no opinion on this issue as I was not asked nor did I become involved in any such decision. I note that the draft Press Statement was faxed to or from 'Trust [Headquarters]' on 26th March 2002.

(c) **Whether you gave any advices about the content of the draft Press Statement?**

No.

(d) *"Colleagues in the Children's Hospital in Belfast had begun to consider the need for a change in the routine use of Solution 18..."* (Ref: WS-059/1 p.3) please:

(i) Identify these colleagues;

The earliest documentary information currently available to me was from Dr Jarlath McAloon, Consultant Paediatrician (also at Antrim Hospital) in a memo dated 27th June 2001 (titled "Use of hypotonic hydration fluids") (copy attached) which he circulated to all consultants in our Team. In this memo, Dr McAloon made reference to ongoing discussions at RBHSC regarding revision of fluid protocols. In his memo, Dr McAloon did not, however, make reference to specific individuals.

(ii) Indicate the change they were considering;

The use of Solution 18 (a fifth normal saline) being reserved for children under 10kg, with half normal saline combined with 2.5% Dextrose being used in place of a fifth normal in the older age group. In his memo received in Antrim on 1st October 2001, Dr Taylor reflected ongoing discussions regarding the use of Solution 18 and alternatives. This was shortly after the first meeting (on 26 September 2001) of the Working Group chaired by Dr Paul Darragh (DHSSPS), which I had been unable to attend.

(iii) State when they were considering the change;

During that period. I cannot be more specific as I was not personally involved in any such discussions prior to September 2001.

(iv) State when you first learnt this information?

See answer to (i) above.

(e) *"At that time there were no established national or local systems to ensure that such issues were brought to the attention of clinicians more widely"* (Ref: WS-059/1 p.3) please state:

- Whether at that time you served on the Chief Medical Officer's Special Advisory Committee;

  Yes. My understanding is that the Committee was known as the Paediatric Specialist Advisory Committee. This Committee normally met annually.

- Whether this afforded you an opportunity to raise issues of significance which might then be widely disseminated;

  Yes, within the paediatric community.

- Whether hyponatraemia and/or the death of Raychel Ferguson was brought to the attention of this committee?

  Hyponatraemia was brought to the attention of this Committee (after September 2001). I cannot recall whether, or not, Raychel's death was specifically mentioned.

(f) *"Most Paediatric Units were still using their traditional regimes based on Solution 18 until further concerns were raised within Northern Ireland in September 2001"* (Ref: WS-059/1 p.3), what were those further concerns?

Information detailed above relating to risks of hyponatraemia.

(3) In respect of your Report on Raychel Ferguson dated 12th November 2002 (Ref: 022-010a-040) please:

(a) Identify those *"statements provided by the doctors and nurses involved in Raychel's care"* as studied by you;

Drs Johnston, Trainor, Morrison, Makar, McCord, Nesbitt, Date, Jamison; Mr Gilliland; Sr Millar; Staff Nurse Noble.

(b) State whether you were permitted an opportunity to interview the nurses and doctors involved in the care of Raychel;

I did not interview the nurses and doctors involved in the care of Raychel.

(c) Identify those *"two deaths"* cited by you as raising concerns in Northern Ireland in September 2001 as to the use of Solution 18;

Trevor Birney of UTV contacted me on 22nd April 2004 explaining that he was researching a programme "on the deaths of Raychel Ferguson and Lucy Crawford".

When subsequently interviewed by Trevor Birney, I had mentioned that the Working Group had been set up by Dr Campbell after the DHSSPS became aware of the deaths of two children from hyponatraemia (as stated in Dr Campbell's letter dated 25 March 2002 which introduced the new Guidance to the service). He then asked me for the names of the two children and I replied that they were Lucy Crawford and Raychel Ferguson. This reflected my understanding that part of the impetus for establishing the Working Group was that the DHSSPS had become aware of the death of Raychel Ferguson at the RBHSC in 2001, and that the other child had been Lucy Crawford (who had died in April 2000). This understanding was based on a statement made by Dr Campbell (during an interview in March 2004) that her department had become aware of Lucy Crawford's death around the date of Raychel Ferguson's in June 2001. In fact, I subsequently became aware that the DHSSPS was not informed of the death of Lucy Crawford at that time, but that the second child referred to in that letter was Adam Strain (who had died some years previously in Belfast). I had had no involvement in, or knowledge of, the care of Adam or any subsequent proceedings, and so assumed (wrongly) that the two children were the two who had subsequently become known to me. I was first made aware of Lucy's death in February 2002.

I believe that the two children were, in fact, Adam Strain and Raychel Ferguson.

(d) In relation to your observation *"while it is possible in retrospect to form the opinion reached by Dr. Sumner that Raychel must have suffered severe and prolonged vomiting..."* state whether you formed this opinion, and if so when;

From reading the statements provided to me and Dr Sumner's report in preparing my initial report (022-010a-040), I was aware that this was a matter where opinions differed between those involved. I was not provided with further information relating to this, and the content of my subsequent report to the Inquest (012-023-132 and 133) did not necessitate me forming a view on it. However, I pointed out in my initial report (and, again, in my letter dated 27th January 2003 (022-004-013)) the importance of this issue receiving further consideration.

(e) In respect of your impression that *"they acted in accordance with established custom and practice in the Unit at that time"* particularise the custom and practice referred to;

The use (in the Unit) of Solution 18 for provision of intravenous fluids in childhood (as described by Dr Makar in his statement for the Coroner).

(f) *"It is however important that further details are obtained of relevant nursing and medical procedures and management in relation to fluid administration and post-operative monitoring of fluid intake, urine output and other losses such as vomiting. In particular information needs to be obtained regarding the local policy for post operative fluid administration in children. Was the prescribed regime in this case in keeping with this guidance"* advise as to whether you were supplied with these details, information, policies or guidance;

I was not.

(g) *"If... In my opinion, their actions do not amount to negligence"* indicate whether you were asked to give an opinion in respect of negligence or an opinion for H.M. Coroner's Inquest;

I was not asked by DLS to give an opinion in respect of negligence. I was asked by DLS to consider the papers and thereafter prepare a report for DLS/the Trust. I had not previously prepared a report in respect of a Coroner's Inquest, my only previous experience of preparing medico-legal reports being in clinical negligence claims.

(h) *"...One sector of the medical profession can become aware of risks..."* identify the sector you refer to?

This was intended as a general statement as it could apply to any sector, but specifically in this case (as stated in my previous statement) to information identified in specialist paediatric anaesthesia or intensive care literature, which was not widely disseminated to a wider (for example, paediatric) sector.

(4) The Assistant Director of Legal Services wrote to H.M. Coroner on 29th March 2002 and stated (Ref: 160-163-003) *"another issue which is of concern to the Trust is Dr. Sumner's conclusions in page 4 of his Report in the comments numbered 2 and 5 that the deceased suffered very severe and prolonged vomiting. This conclusion is strongly disputed by the Trust. The nurses who were caring from the deceased during the relevant period have been interviewed in detail about this matter and they are all of the opinion that the vomiting suffered by the deceased was neither severe nor prolonged."* In respect of this please state:

(a) **Whether you were provided with the interviews of the nursing staff involved in Raychel's care;**

I was not provided with any interviews of nursing staff involved in Raychel's care. I was provided with statements from Sr Millar and Staff Nurse Noble under cover of a letter from DLS dated 1st November 2002. I was not made aware of the existence of any interviews.

(b) **Whether you analysed the notes, statements and records provided you to determine the instances of recorded vomiting;**

I considered Raychel's notes and statements provided under cover of a letter from DLS dated 1st November 2002 and believe that I noted the instances of vomiting referred to.

(c) **Whether you were able to dissent from Dr. Sumner's view based on the information you had?**

I was not in a position to dissent from Dr Sumner's view based on the information that I had and highlighted in my report a number of areas that required elucidation.

(5) **In relation to the Chief Medical Officer's Working Group on the Prevention of Hyponatraemia please confirm:**

(a) **Your role in this work;**

Member of the Group (but unable to attend its first meeting on 26th September 2001).

(b) **Whether you considered the case of Raychel Ferguson in respect of this work;**

It was clear from the outset that the Working Group had been set up as a result of a recent death. I do not recall Raychel's case being considered as part of the Working Group. I note from the minutes of the meeting on 26th September 2001 that Dr Taylor refers to "cases seen in RBHSC", which may, or may not, have included Raychel. Specific details of an individual child or children may have been mentioned at the first meeting of the group on 26th September 2001, but I was unable to attend on that occasion. As I understood it, the Group's remit was not to review the management of any individual case(s), but rather to develop Guidance as soon as possible.

(c) **Whether you considered the case of Lucy Crawford in respect of this work;**

I do not recall the case of Lucy Crawford being considered as part of the Working Group. As I understood it, the Group's remit was not to review any individual case(s), but rather to develop Guidance as soon as possible. Specific details of an individual child or children may have been mentioned at the first meeting of the group on 26th September 2001, but I was unable to attend on that occasion. I first became aware of Lucy's death in February 2002.

(d) **Whether you kept any notes/records or memoranda in relation to this work;**

Notes of meeting 26 September 2001 (007-048-094 to 096)).

(e) **When you first became aware of the article by Arieff *et al* (BMJ 1992)-"*Hyponatraemia and death or permanent brain damage in healthy children*" (Ref: Ref: 011-011-074);**

To the best of my knowledge, on 5th February 2003 (I had noted this date on my copy of the paper).

(f) **Whether the Working Group was aware of this article;**

I do not know if the individual members of the Working Group were aware of this article, however, it was not drawn to my/the Group's attention at any meeting that I attended.

(g) **Whether Drs. Peter Crean and Robert Taylor were members of the Working Group;**

Both are listed in the notes of the first meeting as having attended.

(h) **Whether Drs. Crean and Taylor drew to the attention of the Working Group the fact that they were aware of this Arieff *et al* paper in June 1996 and that they had formally drawn it to the attention of H.M. Coroner by statement (Ref: 011-014-107a);**

I have no recollection of Drs Crean and Taylor drawing the Arieff et al paper to the attention of the Working Group or that they had drawn it to the attention of HM Coroner by statement.

(i) **Whether you incorporated your learning on hyponatraemia within your teaching at Queen's University Belfast?**

I did not formally teach about fluid management at Queen's University, Belfast. My clinical teaching at Queen's University was delivered during the attachments of 4th year students to the Paediatric Unit at Antrim Hospital, where issues were addressed as they came up in a set sequence of seminars and in relation to the care of individual children (teaching shared with trainee doctors). While the principles of fluid management have always been important in Paediatrics, specific emphasis on this issue developed only after the events of 2001. I have referred to the importance of this issue and its appropriate management as this has been relevant to my subsequent teaching contributions.

(6) **In respect of the letter from DLS dated 23rd January 2003 (Ref: 160-045-001) and the request made of you that you *"...consider Dr. Warde's report and provide me with any further comments which you have which might assist the Trust"* please state whether you were able to reconcile this request with your status as an independent expert?**

My interpretation of this request was that I was to assist the Trust by commenting on the issues raised in Dr Warde's report, without prejudice to how my comments might influence the Trust in relation to the inquest. This I did in my response (022-004-013), which was necessarily provided with minimal time for detailed consideration (in fact, on the same day), and therefore concentrated on issues relevant to my own previous report.

(7) **In respect of your Report of 30th January 2003 (Ref: 160-242-001) (as reproduced as your Witness Deposition for Inquest) (Ref: 012-030-153) please state:**

(a) **When you amended and edited your 12th November 2002 opinion to produce your January 2003 opinion;**

At some point between 12th November 2002 and 30th January 2003 – I have no recollection or documentation which enable me to determine the exact date, but this is likely to have been in late January 2003.

(b) Why you made these changes;

As I have no documentation which enables me to directly answer this question, I can only describe the context in which it took place. My initial report was prepared in response to a request from the DLS dated 1st November 2002, which referred to the Coroner's inquest but did not specify the issues on which my opinion was being sought. My previous experience in providing such reports had been in relation to clinical negligence claims, so I prepared my initial report (dated 12th November 2002 (022-010a-040)) in the format I had previously used for that purpose. I have no clear recollection, but infer that I was later informed that the format and content needed to be revised for the different circumstances of an inquest and that the issue of negligence was not one on which my opinion was being sought. I, therefore, revised my report to provide my opinion for the Coroner on the issues relating to the condition of hyponatraemia itself. I was content to do so as it did not require me to comment on areas on which Dr Sumner had greater experience and expertise (namely, postoperative care of children). In addition, I had not been provided with the further information that I had requested (and would have needed) in order to do so, and also because, in my opinion, those specific aspects had been appropriately dealt with by Dr Sumner.

As I had never encountered this complication in all my years of ***medical*** *paediatric practice since 1975, my aim in the report was, from a paediatric perspective, to set out the approach to use of Solution 18 in medical (as opposed to surgical) paediatric practice, and the changes which had already taken place in that practice since 2001. It was important that the wider aspects of the issues relating to hyponatraemia should be provided to the inquest, and I regarded these (and in particular the issues relating to awareness and dissemination of these issues across the relevant clinical specialties and throughout the UK) as being more appropriate to my expertise than those relating to the operative and postoperative management, which had already been dealt with in detail by Dr Sumner, Consultant Paediatric Anaesthetist, in his report.*

(c) Whether these changes were discussed by you with any representatives of the DLS or AHHSST;

I have no recollection or documentation which enables me to answer this question, other than as explained at 7(b) above.

(d) Whether you liaised with anyone in respect of the changes to be made to your final report, and if so who;

I have no recollection or documentation which enables me to answer this question, other than as explained at 7(b) above.

(e) Were you asked, advised or directed to make these changes and if so by whom;

I have no recollection or documentation which enables me to answer this question, other than as explained at 7(b) above.

(f) Why you did not think it relevant to include in your evidence to H.M. Coroner:

(i) Your fluid calculations;

They were not, in my view, relevant to the issues on which my report to HM Coroner was concentrating.

(ii) Your view of Dr. Sumner's opinion in respect of Raychel's vomiting;

My contribution to the inquest did not, in my view, necessitate me forming a view on this issue.

(iii) Your reading of the vomiting accounts given in the witness statements;

My contribution to the inquest did not, in my view, necessitate me forming a view on this issue.

(iv) The failure to provide the additional details and information as sought by you?

My contribution to the inquest did not, in my view, require this information and, in any event, Dr Sumner had dealt with these issues in his evidence.

(8) In relation to the H.M. Coroner's Inquest into the death of Raychel Ferguson please state:

(a) Whether you attended pre-Inquest consultations with the Directorate of Legal Services and/or representatives of the AHHSST and if so when and with whom;

I have no recollection or documentation which enables me to answer this question.

(b) H.M. Coroner informed the Chief Medical Officer by letter of 11th February 2003 (Ref: 012-064-323) that *"Dr. John Jenkins, Senior Lecturer in Child Health at Queen's University, who also gave evidence... concurred with all the views expressed by Dr. Sumner"* if this is so, as to why you did not express this concurrence in your Reports;

As far as I can recall, my concurrence with Dr Sumner's views developed as I heard him explain those views during the inquest itself, including his comments where "he expressed praise for the new protocol that has been drawn up and commented that Northern Ireland was ahead of the rest of the United Kingdom" (012-064-323).

(c) Why you did not inform H.M. Coroner of your two separate Reports of 12th November 2002 (Ref: 022-010a-040) and 27th January 2003 (Ref: 160-215-002);

I have no recollection or documentation which enables me to answer this question. My initial report had been prepared for DLS, and (as explained above and far as I can infer from the changes I made), I believe that I was informed that my contribution to the inquest should more appropriately deal with the wider aspects. If I had been made aware that reference should have been made to my first report I would have done so.

(d) Why you did not inform H.M. Coroner that Dr. Warde (Consultant Paediatric Anaesthetist) had also given his opinion by way of formal report on the causes and circumstances of Raychel's death and that you had been briefed with a copy of his Report;

I was given no reason to do so. Dr Warde's report had not resulted in any change in my opinion, and was not relevant to the issues on which my contribution to the inquest was concentrating. For completeness, whilst I had been provided with a copy of Dr Warde's report in late January 2003, it had not been sent to me when I was initially briefed in November 2002.

(e) Whether you were informed as to the reasons for Dr. Warde's non-attendance at the Inquest;

No.

(f) Why you did not inform H.M. Coroner as to the death of Lucy Crawford, the relevance of the facts and circumstances of her death?

I was not aware of any reason to do so. The Guidance on the Prevention of Hyponatraemia in Children had been published and disseminated in March 2002 and, in my view, awareness was already high.

(9) Please provide such additional comment as you think relevant. It would be of very considerable assistance if you could attach such documentation as you may hold which relates to procedures, strategies, policies or other issues of relevance.

THIS STATEMENT IS TRUE TO THE BEST OF MY KNOWLEDGE AND BELIEF

Signed: John Jenkins Dated: 24 June 2013

united hospitals

Antrim Hospital . 45 Bush Road . Antrim . BT41 2RL . Telephone: 01849 424000

M E M O

FROM: **Dr. J. McAloon, Consultant Paediatrician**

TO: **The undernoted**

Cc Dr. Granger, sent 29/6/01

JJ 28/6/01.

DATE: 27th June 2001

RE: Use of hypotonic hydration fluids

Dear John Colleague,

You may already be aware of recent concerns about the appropriateness of the use of hypotonic solutions for hydration in patients with a low Sodium. The issue has recently been highlighted by the death of a child in the Province. Please find enclosed two recent papers highlighting the problem. I understand that the Protocols in RBHSC may shortly be revised with the use of a fifth normal saline being reserved for children under 10Kg, with half normal saline combined with 2.5% Dextrose being used in place of a fifth normal in the older age group.

This is an issue that this Department will also need to review and I understand that the Medical Director of the Trust has already made some enquiries through one of the anaesthetists.

My suggestion is that we adopt the same Protocols as RBHSC whenever these are produced and in the meantime highlight with our Juniors the potential complications of hyponatraemia in association with the use of hypotonic solution. I think there may also be an issue here for our nursing staff in terms of oral re-hydration of patients admitted with gastro-enteritis where very often I notice that the patients are either drinking water or diluting orange as opposed to an appropriate rehydration solution.

I suggest that any thought you have be co-ordinated through Calum and the matter discussed at the next Consultant and Staff Grade meeting. As you know in the Induction Programme there is a session on Dehydration and Intravenous Fluids in which I would like to highlight this new awareness and the consensus (if possible)

on management.

Comments please for the next consultant meeting.

Yours sincerely,

Dr. J. McAloon

To: Dr. [redacted]
Dr.
Dr.
Dr. J. Jenkins
Dr. [redacted]
Dr.
Dr.
Dr.

The ROYAL HOSPITALS

1/10 Copy to each. Consultant.

Dear Jordan,

I had spoken to you previously about a meeting on Dilutional Hypovolaemia at Castle Buildings with Dr Paul Darragh, Chair. I enclose a draft recommendations for your interest. I have consulted widely within and without this hospital and have made quite reasonable suggestions. However it has become clear that 0.18%NaCl/4%Glucose will no longer be a recommended intravenous fluid for children over 1 year of age.

The clinical problems are actually not directly related to that fluid but rather to miscalculations in body weight, fluid maintenance and poor monitoring of fluid balance and blood chemistry. It is therefore most important that these areas are addressed as well as any change to the fluid type.

Following the death of a child from hyponatraemia (from another hospital in our PICU) I have spoken to our paediatric pharmacist, Anne Burns and completed a "yellow card" hazard to CSM. I have also audited our incidence of admissions to PICU with hyponatraemia.

At the meeting one of my colleagues was quite insistent on the move to Normal saline (no glucose component) which seemed to accepted by the group. However after speaking to several paediatricians I suggested to this group that the fluids should contain "at least 0.45% NaCl/2.5% Glucose." I am very concerned about the move to Normal saline as a recommended fluid, even in the postop child.

1. Any consequent cases of hypoGLYCAEMIA will cause the guideline to ridiculed.
2. It will be practically difficult and potentially dangerous to change fluids at 12 hours eg at 03.00am
3. It will be much more difficult to get the stakeholders to accept such an extreme change.

For these reasons I believe that the recommendation should remain "Infuse at least 0.45% NaCl in 2.5% Glucose solution."

I have tried to get the convenors of the group to consult more widely prior to any drafting of guidance. I think you may be getting a few letters on this topic as I have discussed the subject with as many colleagues as possible.

Yours truly,

Bob Taylor, PICU

JBJ 3/10/07. Paper prepared by Bob Taylor.
Comments to JM&A by 8/10/07 please.
J.

Hyponatraemia in children

Dilutional Hyponatraemia has been documented in otherwise healthy children following routine elective surgery. It occurs in often female children 3-10 years of age and is associated with "stress". Risk factors include; Hypernatraemia, dehydration (>7%), Stress; Nausea, pain, anxiety, certain drugs, disturbances of the Central Nervous System and Metabolic and Endocrine disorders.

A fluid for children recommended for many years as a standard is 0.18 NaCl in 4% Glucose. It contains 30 mmol/l of sodium which when administered at the calculated rate (4 mls/kg/hour for the first 10 kgs body weight) provides the daily requirement of sodium and glucose.

0.18 NaCl in 4% Glucose is **isotonic***in vitro* ie has the same osmotic potiental so will not cause fluid shifts within the body. However in the catabolic (sick) child the glucose is metabolised rapidly causing the fluid to become ***hypotonic*** thereby leading to massive fluid shifts. At the same time because of the loss of fluid from the circulation often combined with a degree of dehydration a potent anti-diuretic hormone (ADH) response causes the kidneys to retain water resulting in a low volume concentrated urine, high in sodium. This may be compounded by the administration of a "fluid challenge" to elicit an improved urinary output.

This is a "double whammy" excess free water is administered and excess free water is retained. Water is drawn across blood capillaries into the interstitial and intracellular spaces. The child will become "puffy" looking and of greater consequence the brain will swell with the shift of water, leading to seizures and herniation of the tentorium and death. Therefore to prevent hyponatraemia we must limit the free water component of intravenous fluids AND monitor urine output and serum chemistry.

Halberthal M et al studied 23 patients studied with acute hyponatraemia. All received hypotonic fluids (plasma Na+ < 140 mmol/l). 16 (70%) received excessive maintenance fluids (>50%). 13 (57%) were postoperative patients and 18 (78%) developed seizures. 5 (22%) Died (Brainstem death), 1 severe neurological deficit.

diabetes. Most patients have no means of assessing control apart from the presence or absence of symptoms. 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 [illegible] postmortem examination. All 15 patients not tr[illegible] for their hyponatraemia in a timely manner [illegible] died or were permanently incapacitated by [illegible] damage. The only patient treated in a t[illegible] manner was alive but mentally retarded.**

***Conclusions*—Symptomatic hyponatraemia [illegible] result in a high morbidity in children of both ge[illegible] which is due in large part to inadequate [illegible] adaptation and lack of timely treatment.**

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Introduction

In previous studies from our laboratories [illegible] described the symptomatology, clinical course, [illegible] of treatment, and pathological findings in more [illegible] 225 adults (aged over 16) with symptomatic [illegible] natraemia.[1-8] Although the actual incidence of [illegible] natraemia seems to be similar among men and [illegible] women,[8,9] almost all adult patients suffering [illegible] natraemic brain damage are women. Although [illegible] are a number of reported paediatric cases of [illegible] natraemia,[10-12] there are few reported cases of [illegible] permanent brain damage among children with [illegible] disorder,[13,14] and most such children had pre-existing [illegible] neurological disorders.[15-17] Neither the gender [illegible] bution nor the incidence of brain damage [illegible] children with hyponatraemia is known.[10,11,13] [illegible] children suffering brain damage from hyponatr[illegible] neither the type nor the gender distribution is [illegible] We describe both a prospective and a retrospective analysis of generally healthy children who were [illegible]

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

Clinical characteristics of 16 children with symptomatic hyponatraemia

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, tonsilltis	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
M 4	16·8	138	107	16	88	56	Tonsillitis	Tonsillectomy	Yes	None	Died
M 2	11·4	138	116	3	123	NA	Undescended testicle	Orchiopexy	Yes	None	Died
M 6	15·0	138	119	12	40	11	Severe epistaxis	Posterior packing	Yes	None	Died
M 12	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
	23·8	138	115	37	125	74					
	12·9	2	7	34	83	82					
	3·2	1	2	9	21	24					

*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 24412 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 c
with symptomatic hyponatraemia (101-123 m
can abruptly develop respiratory arrest and eith
or develop permanent brain damage. The perm
brain damage can include pituitary infarction
resultant central diabetes insipidus and melli
syndrome not previously described in children
incidence of postoperative hyponatraemia in c
(0·34%) was less than in adults (1-4%).[8 21] Ho
among paediatric patients who developed sympt
hyponatraemia the incidence of permanent
damage was substantially higher than in ad
Both the types of surgery and gender distri
among our 16 patients (table) were the same as t
common operations and gender distribution
United States as a whole,[20] and thus our 16 p
were representative of the spectrum of elective
tric surgical patients.

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

BRAIN ADAPTATION TO HYPONATRAEMIA IN CH

In adults oestrogens seem to impair the abil
brain to adapt to hyponatraemia and andro
augment such adaptation.[30-33] However, pre
children have only minimal to absent concent
either hormone, thus negating such effe
adults suffering permanent brain damage fr
natraemia are female,[3 5 7 8] but in the curren
minority of affected patients (43%) in both
pective and retrospective studies were fema
unlike the marked gender differential in ad
and female children seem to be at simila
developing hyponatraemia encephalopathy
test)). Furthermore, neither the actual co
of serum sodium nor the rapidity of deve
hyponatraemia seemed to predict the ultima
in these 16 children (table). Hyponatraemia
over a mean of 37 hours and the range of ser
values was 101-123 mmol/l, values quite
those previously reported in children wi
matic hyponatraemia who did not dev
damage.[10 12 13 16]

EFFECTS OF PHYSICAL FACTORS

When hyponatraemia was present all 16
had radiological evidence (computed to
magnetic resonance imaging) of cereb

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, 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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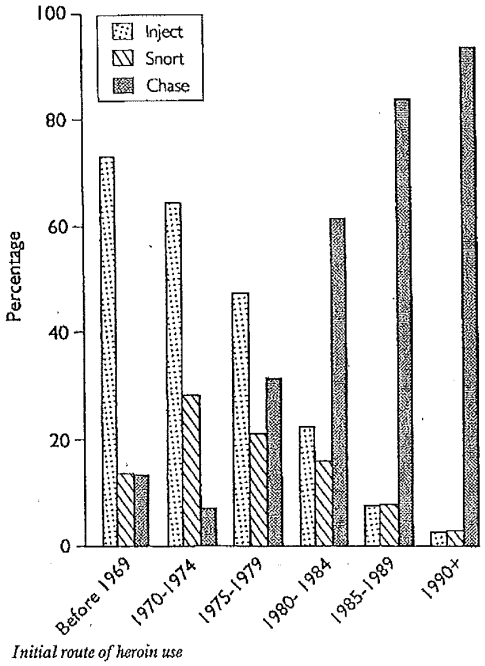
AIDS and drug misuse are linked mainly by the injection of many drugs. Major changes in the methods of heroin use, however, have fundamentally altered the importance of heroin use in the transmission of HIV. Recent reports describe the extent of "chasing the dragon" (inhaling sublimated heroin after 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 exchange scheme. A total of 136 (34%) had n contact with either treatment services or an e scheme. Their ages ranged from 17 to 53 (me 27·6 (6·3) years); 248 (62%) were male; 96 (24 in current employment. There was wide vari first year of use of heroin use (1954 to 1991); started during the '60s, 28 (7%) during the ea 76 (19%) during the late '70s, 124 (31%) du early '80s, 120 (30%) during the late '80s, and during the '90s.

Three different routes of initial drug u identified: injecting, snorting, and "chas dragon." Analysis of these data by year re major change in the annual proportion wh initiated by either injecting or chasing (figure).

"Chasing" was a route of initiation for a mi users up to the late 1970s but has become an inc common route of initiation since 1975. By 19 were as many initiations by chasing as by injecti by 1981 more than half of the initiations int use were by chasing (with the annual pr remaining above half since 1981). By 1985 m three quarters of initiations were by chasing, a 1988, 87 out of 93 initiations (94%) were by During most years, a tenth to a quarter of u initiated by snorting.



Initial route of heroin use

Comment

Heroin use today is not what it was ye Initiation no longer occurs by injecting but by route of "chasing the dragon." The emergen non-injecting routes of heroin use may partly not only the major heroin epidemic in the Kingdom during the 1980s but also its continuation[4] despite the addition of AID potential consequence. Perhaps the protecti taboo against injecting was circumvented a fettered epidemic has developed. In the 1990 all initiations into heroin use in our Londo were by "chasing the dragon," even though in other countries (for example, the United S even in other British cities (for example, Edi continues to be by injection. Should the L London be regarded as an isolated develop few "chasing" cities, or is it an indicator future changes on a wider scale? And wh significance for tomorrow's prevention and programmes?

Our level of ignorance about changing rou administration is not only scientifically distu also interferes with the development of preve treatment programmes. Effective primary pr strategies depend greatly on the adequacy of k about the gateways into drug use, and yet o standing of the phenomenon is informed

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