Statement about Investigation of the Death of Rachel Ferguson on 9th June 2001

I, Dr. Raymond Fulton, MB, FRCP London, was Medical Director of Altnagelvin Hospitals H&SS Trust at the time of Rachel Ferguson's death on 09/06/01. I was responsible for investigating the circumstances of her death within the hospital and to make recommendations for any action to prevent recurrence.

On 12/06/01 I set up a Critical Incident Enquiry involving all relevant clinical staff to establish the clinical facts. As a result of this six Action Points were agreed and circulated to all present on 13/06/01 (*see attachment 1*).

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On 14/06/01 following Action Point 1 Dr Nesbitt, Clinical Director for Anaesthetics, wrote to me saying he had found that Solution 18 was currently used in several hospitals in Northern Ireland. He said he had reviewed the literature, which had convinced him that Solution 18 should not be used in surgical paediatric patients. He stated that henceforth Solution 18 would not be used in these circumstances in Altnagelvin (*see attachment 2*).

On 18/06/01 at a meeting of Medical Directors with Dr. I Carson, Medical Advisor to the CMO, at Castle Buildings I described the circumstances of this death. There were several anaesthetists present, some of whom said that they had heard of similar situations though it was not clear if there had been fatalities. I suggested that there should be regional guidelines.

On 22/07/01 I rang the Chief Medical Officer, Dr Henrietta Campbell, and informed her of the circumstances of the death. I suggested she should publicise the dangers of hyponatraemia when using low saline solutions in surgical children. I said there was a need for regional guidelines. Dr Campbell suggested that CREST (*Regional Guidelines Group*) might do this.

In Mid June 2001 I rang Dr. W McConnell, the Director of Public Health at the Western Health & Social Services Board, and described the circumstances of the death. He said he would discuss the matter at his next meeting with the Chief Medical Officer and the Directors of Public Health of the three other Health Boards. I sent him reprints from the British Medical Journal on Hyponatraemia (*see attachment 3*)

On 05/07/01 Dr McConnell wrote to confirm that he had discussed the case with the CMO and DPHs. Each DPH had agreed to alert the Paediatricians in their respective Board areas to the hazards of Hyponatraemia (*see attachment 4*).

On 6/07/01 Mrs Burnside, Chief Executive of Altnagelvin Hospitals H&SS Trust, contacted the CMO to personally advocate a regional review (*see attachment 5*). I remember seeing a reply from the CMO agreeing to set up a regional Enquiry Group and that Dr Nesbitt would be a member.

On 14/01/02 I arranged for the CMO to view a presentation by Dr Nesbitt on Hyponatraemia while she was visiting Altnagelvin Hospital to present accreditation to the Trust's HSDU.

On 09/04/02 I chaired a meeting of relevant clinical staff to review the Action Plan of 12/06/01 in light of the publication of Departmental Guidelines on Hyponatraemia. (*see attachment 6*)

On 01/05/02 Dr. Nesbitt, who succeeded me as Medical Director, wrote to the CMO enquiring if the death of a child some years previously from hyponatraemia in the RBHSC had been reported to the Department. Dr. Nesbitt had become aware of the RBHSC case whilst investigating the death of Rachel Ferguson *(see attachment 7).*

On 10/05/02 Dr. Campbell replied that the Department had not been made aware of the first case either by the Royal Victoria Hospital or the Coroner. She had only become aware of the original case whilst working on developing guidelines following the death of Rachel Ferguson (*see attachment 8).*

Throughout this process I was struck by the wish of all concerned to learn from this death, which is unique in their experience. I received full co-operation from all clinical staff who are extremely distressed by Rachel's death.

RA Fulton

DR RAYMOND FULTON
12/11/02

attachments 1-8

(1)

AGREED ACTION FOLLOWING CRITICAL INCIDENT MEETING 12/06/01

1 Review evidence for use of routine post-operative low electrolyte IV infusion and suggest changes if evidence indicates. No change in current use of Solution 18 until review.

Action Dr Nesbitt

2 Arrange daily U&E on all post-operative children receiving IV infusion on Ward 6. 485

Action Sister Miller

3 Inform surgical junior staff to assess these results promptly.

Action Mr Gilliland

4 All urinary output should be measured and recorded while IV infusion ~~progress~~ in progress.

Action Sister Miller

5 A chart for IV fluid infusion rates to be displayed on Ward 6 to guide junior medical staff.

Action Dr McCord

6 Review fluid balance documentation used on Ward 6.

Action A Witherow

R A FULTON
Medical Director **13/06/01**

(2)

Dr G A Nesbitt
Clinical Director
Anaesthetic Department
Altnagelvin Hospital

Date: 14th June 2001

14 JUN 2001

Dr Raymond Fulton
Medical Director

Re: Fluid management in Children

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Dear Dr Fulton, Raymond.

I have contacted several hospitals including The Royal Hospital for Sick Children and made enquiries about peri-operative fluid management.
The Children's Hospital Anaesthetists have recently changed their practise and have moved away from No.18 solution (fifth normal NACL in 4% Dextrose) to Hartman's solution. This change occurred 6 months ago and followed several deaths involving No.18 solution.
Craigavon Hospital and the Ulster Hospital both use Hartman's intra-operatively and No.18 post-operatively as is our practise. The Anaesthetists in Craigavon have been trying to change the fluid regime to Hartman's postoperatively but have met resistance in the Paediatric wards where, as in Altnagelvin, they have followed a medical paediatric protocol.

In view of recent events, and papers on the subject, and the fact that the Children's Hospital no longer uses No.18 solution, I have decided to recommend that we do the same. I have spoken to Sister Miller in the Paediatric ward and also to Dr McCord who both are in agreement. Dr McCord has agreed to add this to the protocol he is developing for calculating the amount of fluid to be prescribed. He has further agreed that, pending discussion with his colleagues, fluid management in postoperative children should be under the supervision of paediatricians.

To summarise: Altnagelvin Hospital has followed what is a widespread and accepted policy of using No.18 solution for postoperative fluids. There is evidence to show that this policy is potentially unsafe in certain children who have undergone a surgical procedure. The Children's Hospital has ceased to use it and Craigavon is trying to effect a change in this direction. As from today we will no longer be routinely using this fluid in the management of surgical cases.

Yours sincerely,

G A Nesbitt Clinical Director

cc Theresa Brown Risk Management Coordinator

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Home Help Search/Archive Feedback Search Result

BMJ 2001;322:780-782 (31 March)

Clinical review

Lesson of the week

Acute hyponatraemia in children admitted to hospital: retrospective analysis of factors contributing to its development and resolution

Do not infuse a hypotonic solution if the plasma sodium concentration is less than 138 mmol/l

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Michael Halberthal, *fellow* [a], **Mitchell L Halperin**, *professor* [b], **Desmond Bohn**, *professor* [c].

[a] Department of Critical Care Medicine, Hospital for Sick Children, Toronto, Ontario, Canada M5G 1X8, [b] Division of Nephrology, St Michael's Hospital Toronto, Toronto, Ontario, Canada M5B 1A6, [c] Department of Critical Care Medicine, Hospital for Sick Children, Toronto

Correspondence to: D J Bohn dbohn[redacted]

Hyponatraemia (plasma sodium concentration less than 136 mmol/l) is acute if the decrease in natraemia occurs within 48 hours. The major dangers from this are brain cell swelling and herniation. [1 2] Two factors are required for hyponatraemia to develop: a source of electrolyte free water and vasopressin to prevent the excretion of that water.[3] Electrolyte free water is given routinely as maintenance fluids based on formulas developed in studies in healthy children more than 40 years ago. [4 5] There are many reasons to anticipate that vasopressin will be released in sick patients (box).[6] Patients with an acute illness may arrive in hospital with a low plasma sodium concentration because of previous water intake. Hence, to minimise the potential threat of brainstem herniation it is important to measure the plasma sodium concentration if intravenous solutions are to be given.

Causes of vasopressin release

- Hypernatraemia (most important stimulus, but not in these patients)
- Low "effective" circulating volume (greater than 7% decrease in extracellular fluid volume)
- Nausea, pain, anxiety
- Drugs (some act through inducing nausea)
- Afferent stimuli by way of the vagus nerve, for example, lung lesions

- Disturbances of the central nervous system (meningitis, encephalitis)
- Metabolic and endocrine disorders for example, hypothyroidism, hypoadrenalism, porphyria

We describe symptomatic hyponatraemia developing over 48 hours in children. In each patient, hypotonic solutions were infused using current guidelines.[7] We related the volume of electrolyte free water given to the decrease in natraemia and assessed whether actions of vasopressin persisted to guide emergency corrective therapy.[8]

We reviewed all patient charts (306 charts) with a recorded diagnosis of hyponatraemia for the past 10 years. Patients were included if their decrease in natraemia was to less than 130 mmol/l and this occurred within 48 hours, if intravenous fluids were given, and if an underlying disease did not compromise renal handling of sodium or water. Thirty patients had acute hyponatraemia. Crucial information was missing for seven, leaving 23 patients in the study group. The median age was five years (range one month to 21 years), with males predominating (18 of 23); 13 developed hyponatraemia in the postoperative period. Fifteen patients were referred to the critical care unit after the development of symptomatic hyponatraemia while receiving intravenous fluids 11 were from the hospital wards and four were transferred from other institutions. Symptoms included seizures (18 patients) and vomiting,17 a warning sign of an increased intracranial pressure. Treatment was withdrawn from five patients after brainstem coning. One patient sustained permanent, severe neurological damage.

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All the children received hypotonic fluids while their plasma sodium concentration was less than 140 mmol/l, because of the wide belief in paediatric practice that "maintenance fluids" should be hypotonic.[9] In fact the volume of maintenance fluid given was 50% greater than recommended values in 16 of the 23 patients. This infusion of hypotonic fluids increased the risk of acute hyponatraemia and brain swelling because vasopressin is typically present in this setting.[1 2 10 11] In quantitative terms, some of the electrolyte free water infused was retained in six of the patients because their urine sodium plus potassium concentration was less than 25 mmol/l (fig 1). In six patients more electrolyte free water was infused than needed to cause the observed decline in natraemia (points above line of identity in fig 2). The remainder of the patients had a decrease in natraemia that exceeded the decline if the entire volume of electrolyte free water infused was retained (points below broken line in fig 2). Therefore there was either another non-recorded input of water or the excretion of a large volume of hypertonic urine (a desalination of infused isotonic saline[12]).

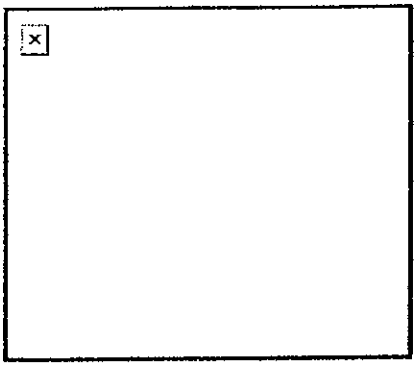

Fig 1. Concentration of sodium and potassium in urine at nadir observed for plasma sodium concentration in 17 patients (numbers represent rate of urine flow)

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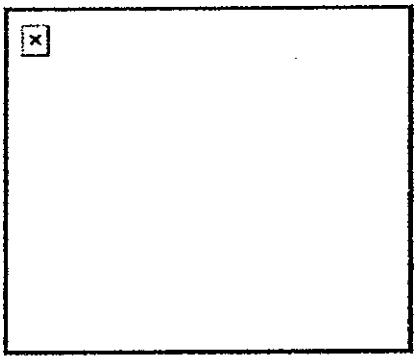

Fig 2. Comparison of decline in plasma sodium concentration with amount of electrolyte free water given in 17 patients. Difference in sodium concentration was between initial value and that at its nadir

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One objective of our study was to assess the renal actions of vasopressin. Because six patients had very hypotonic urine at their recorded nadirs of natraemia, their plasma sodium concentration might have been much lower before water diuresis began (fig 1). Had their plasma sodium concentration been measured after this large water diuresis, the erroneous conclusion might have been drawn that acute hyponatraemia had never been present. Hence its incidence may be much higher than shown by an analysis of hospital records. Therefore acute hyponatraemia could have been an occult cause of morbidity and mortality.

Another implication of cessation of the release of vasopressin concerns treatment. Treatment for acute, symptomatic hyponatraemia causes a prompt decline in the size of brain cells.[10] Hypertonic saline (3%) is the commonest treatment for shrinking brain cell volume, thereby lowering intracranial pressure. Treatment must be prompt because deterioration may be rapid and irreversible, even when symptoms are mild. Enough hypertonic saline (a total of 5 mmol of

sodium chloride per litre of body water[13]) is needed acutely to lower intracranial pressure sufficiently to minimise this risk (the plasma sodium concentration should be increased by 5 mmol/l over several hours). Because an excessively rapid rate of correction of hyponatraemia might have deleterious effects,[6] hypertonic saline should not be given if there is a brisk water diuresis. For example, the plasma sodium concentration will also increase by 1.2 mmol/l/h if 6 ml of electrolyte free water are excreted per kilogram per hour (total body water is close to 600 ml/kg; 6 ml is a 1% change of 120 mmol/l). Whereas excretion of hypotonic urine indicates that electrolyte free water is being excreted (6 of 17 patients, fig 1), it is also important to consider the rate of urine flow. Little electrolyte free water was excreted in the index oliguric patient (flow 0.16 ml/kg/h). By contrast, the excretion of electrolyte free water was high enough to increase the plasma sodium concentration by close to 3 mmol/l/h in the polyuric index patient who recovered (15 ml/kg/h). Vasopressin continued to act in patients excreting isotonic or hypertonic urine, so hypotonic intake must be avoided in them. With these high urine tonicities a further decrease in natraemia would be anticipated if the urine output was high (index case designated with a urine output of 5.3 ml/kg body weight, fig 1).[12] Finally, vasopressin concentrations may decline abruptly, increasing the excretion of electrolyte free water.

Serious symptoms may become evident when hyponatraemia approaches 120 mmol/l, but there are cases where symptoms become evident with a higher plasma sodium concentration, whereas others tolerate this electrolyte disorder without developing seizures.[14] Apart from underlying conditions that might make a patient more susceptible to seizures, a possible important factor could be the extracellular fluid volume of the brain. If this volume was expanded by a large infusion of isotonic saline, a higher intracranial pressure might be present at a given degree of hyponatraemia. Moreover, because there is a relatively larger proportion of brain cell volume to extracellular fluid volume in young patients, they are more vulnerable to an increase in brain cell volume.

Study limitations

Because of a reporting and referral bias, the incidence of adverse outcomes from hyponaetremia cannot be deduced from these data. Our results highlight the dangers of the routine use of hypotonic solutions when vasopressin acts. The currently used guidelines for maintenance fluids in children admitted to hospital must be changed because they do not take into account the unpredictability of vasopressin secretion. We recommend that the concentration of plasma sodium should be measured when starting an intravenous infusion. If it is less than 140 mmol/l then isotonic and not hypotonic fluids should be given. The use of hypotonic solutions should be reserved for patients who have a plasma sodium concentration greater than 140 mmol/l. If a patient receives intravenous fluid that exceeds 5% of total body water (30 ml/kg) then their plasma sodium concentration should be measured. If an intravenous infusion is started to give drugs, a small volume should be used, and the solution should be isotonic if possible.

Acknowledgments

Contributors: MH collected the data and drafted the original manuscript. MLH analysed the data and coauthored the manuscript. DB had the original idea and coauthored the manuscript; he will act as guarantor for the paper.

Footnotes

Competing interests: None declared.

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(*Accepted 26 July 2000*)

(43)

WESTERN
HEALTH AND SOCIAL SERVICES BOARD

AREA BOARD HEADQUARTERS

WMcC/HP

5 July 2001

Dr R Fulton
Medical Director
Altnagelvin Health & Social Services Trust
Glenshane Road
Altnagelvin
Londonderry

492

Dear Raymond

As we agreed, I raised this issue for discussion at the recent CMO/DPH meeting. Dr Campbell indicated that her CMO update was already at the printers and therefore it would be difficult to include anything within it. Knowing this, it was suggested that I would write to each of my Director of Public Health colleagues who would then take responsibility for drawing the issue to the attention of any relevant paediatric settings within their respective Boards.

I enclose a copy of the letter which I have sent to them and I have also taken the liberty of copying to them the articles which you helpfully sent to me. I hope you do not mind my suggestion within the letter that anyone who wanted more detailed discussion might contact you for further information.

Best wishes.

Yours sincerely

Dr W W M McConnell
DIRECTOR OF PUBLIC HEALTH

Enc

15 Gransha Park, Clooney Road, Londonderry, N. Ireland BT47 6TG.
Telephone: L'Derry [redacted]
Fax. No. [redacted]

RF DLS

160-244-010

(48)

WMcC/HP

5 July 2001

To:

Directors of Public Health

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Dear

I am writing to you, as agreed at our recent meeting, with further details regarding the unfortunate death of a young girl at Altnagelvin Hospital. It appears that the use of hypotonic saline is still common practice in a number of paediatric units although there has been information around for a few years suggesting this does present risks to a very small number of children in the acute perioperative period. I have enclosed some articles which you might find of benefit in relation to this.

I know from discussions about this issue with Dr Raymond Fulton, Medical Director at Altnagelvin Hospital, that some paediatric settings within Northern Ireland have made appropriate changes but that in others, while the information may be known by anaesthetic staff, there has not necessarily been discussion regarding change between anaesthetists, surgeons and paediatricians.

I felt it might be helpful to draw this issue to your attention in order that each of us can check with those paediatric settings in our respective Boards to see whether modification in practice may be needed. I hope that the articles are helpful to you. If further more specific information is required I am sure that Dr Fulton would be happy to discuss this with anyone who contacted him.

Best wishes.

Yours sincerely

Dr W W M McConnell
DIRECTOR OF PUBLIC HEALTH

Enc

Sally Doherty

From: Sally Doherty
Sent: 26 July 2001 11:08
To: 'Henrietta.campbell@[redacted]
Subject: electolyte balance in post operative children

I am writing further to Dr Fulton's conversation with you rgarding the above, and am concerned to ensure that an overview of the research evidence is being undertaken. I beleive that this is a regional, as opposed to local hospital issue, and would emphasise the need for a critical review of evidence.

I would be extremely grateful if you would ensure that the whole of the medical fraternity learned of the shared lesson.

I await to hear further from you.

Stella.

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(6)

CRITICAL INCIDENT REVIEW MEETING 09/4/02

To review the Action Plan of the critical incident meeting of 12-6-2001 following the death of Rachel Ferguson.

1 Review evidence for use of routine post-operative low electrolyte IV infusion and suggest changes if evidence indicates. No change in current use of Solution 18 until review.

- **An immediate review was undertaken and a decision was taken that from all Surgical patients (including orthopaedic) to receive I V Hartmans Solution and 6 hourly BM's.**

2 Arrange daily U&E on all post-operative children receiving IV infusion on Ward 6.

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- **This was immediately actioned by Sister Miller. The phlebotomists take the blood. It is not clear who is responsible for ordering the blood. Mrs Witherow and Mrs Brown will prepare ward guidelines**

Acion T Brown A Witherow

3 Inform surgical junior staff to assess these results promptly.

- **This was immediately actioned by Mr Gilliland. All staff are informed at induction. This information should be included in the Junior Doctors handbook. At the moment blood results come up on the computer. This does not show the normal range. Agreed that all bloods are to be reported to the Surgeons routinely. Anne Witherow to speak to Dr. M O'Kane to ascertain if the normal ranges can be put on the computer.**

Action A Witherow

4. All urinary output should be measured and recorded while IV infusion is in progress.

- **The fluid balance sheet has been revised to allow recording of urinary output and vomit.**

5. A chart for IV fluid infusion rates to be displayed on Ward 6 to guide junior medical staff.

- **The chart was prepared and displayed by Dr Mc Cord by July 2001.**

6. Review fluid balance documentation used on Ward 6.

- **The fluid balance sheet has been revised to show exact timing of IV Fluids, and when they have been discontinued. It was noted that there is a Regional Group currently reviewing this form. We will await receipt of the revised form.**

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7. Need to agree responsibility for the prescribing and management of fluids post operatively. Agreed that Dr. Nesbitt will discuss with Anaesthetists and agree a maximum time that postoperative fluids will be prescribed by anaesthetists.

Action Dr. Nesbitt

8. Departmental guidelines received April 2002 regarding fluid management in all children have been displayed on ward 6, theatres and A&E.

R A Fulton

R A FULTON
11-4-2002

(7)

Dr G A Nesbitt
Medical director
Altnagelvin Hospital

1st May 2002

Dr Henrietta Campbell
Chief Medical Officer
Castle Buildings
Upper Newtownards Road
Belfast BT4 3SJ

Re: Hyponatraemia in Children receiving intravenous fluids

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Dear Dr Campbell,

Following the death of a child in Altnagelvin Hospital, which is thought to have followed severe hyponatraemia, many steps have been taken to ensure that such an event does not occur again. We are all anxious to learn from what was a dreadful experience and to share vital information with others. Guidance issued from your Department will help in this regard and we are grateful for the recent posters on the subject.

I am interested to know if any such guidance was issued by the Department of Health following the death of a child in the Belfast Hospital for Sick Children which occurred some 5 years ago and whose death the Belfast Coroner investigated. I was unaware of this case and am somewhat at a loss to explain why.

I would be grateful if you could furnish me with any details of that particular case for I believe that questions will be asked as to why we did not learn from what appears to have been a similar event.

Yours sincerely,

G A Nesbitt Medical Director

(8)

Department of Health, Social Services & Public Safety
An Roinn Sláinte, Seirbhísí Sóisialta agus Sábháilteachta Poiblí

From The Chief Medical Officer:
Dr Henrietta Campbell CB

Castle Buildings
Upper Newtownards Road
Belfast BT4 3SJ

Telephone: [redacted]
Fax:

E-Mail: [redacted]

Dr G A Nesbitt
Medical Director
Altnagelvin Hospitals HSS Trust
Altnagelvin Area Hospital
Glenshane Road
LONDONDERRY
BT47 6SB

10 May 2002

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Dear Dr Nesbitt

HYPONATRAEMIA IN CHILDREN RECEIVING INTRAVENOUS FLUIDS

Thank you for your letter of 1 May regarding guidelines for the prevention of hyponatraemia in children receiving intravenous fluids.

Your letter referred to a Coroner's case five years ago in which the cause of death of a child was reported to be due to hyponatraemia. This Department was not made aware of the case at the time either by the Royal Victoria Hospital or the Coroner. We only became aware of that particular case when we began the work of developing guidelines following the death at Altnagelvin.

I would like to thank the staff at Altnagelvin for alerting me to the need for guidelines and for all the assistance which was given to their development.

With kind regards.

Yours sincerely

HENRIETTA CAMPBELL (Dr)

Statement about investigation of the death of Rachel Ferguson on 9 June 2001

I was Medical Director of Altnagelvin Trust at the time of Rachel Ferguson's death on 09 06 01. I was responsible for investigating the circumstances of her death within the hospital and to make suggestions for any action to prevent recurrence. The following is the sequence of action I undertook.

12 06 01 — I set up a Critical Incident Enquiry involving all relevant clinical staff to establish the clinical facts. As a result of this 6 Action Points were agreed and circulated to all present on 13 06 01 (Enclosure 1).

14 06 01 — Following Action Point 1 Dr Nesbitt, Clinical Director, Anaesthetics, wrote to me saying he had found that solution 18 was currently used in several hospitals in Northern Ireland. He said he had reviewed the literature which had convinced him that Solution 18 should not be used in surgical paediatric patients. He stated that henceforth Solution 18 would not be used in these circumstances in Altnagelvin (Enclosure 2). ٤٩٩

18 06 01 — At a regular meeting of Medical Directors at Castle Buildings I described the circumstances of this death. There were several anaesthetists present some of whom said they had heard of similar situations though it was not clear if there had been fatalities. I suggested that these should be regional guidelines.

22 07 01 — I rang the Chief Medical Officer, Dr Campbell, and informed her of the death. I suggested she should publicize the dangers of hyponatraemia when using low saline solutions in surgical children. I said there was a need for regional guidelines. Dr Campbell suggested that CREST (the regional Guideline group) might do this.

Mid June 2001 — I rang the Director of Public Health at Western Health Board (Dr McConnell) and described the death. He said he would discuss the circumstances at his next meeting with the Chief Medical Officer and the Directors of Public Health of the three other Health Boards. I sent him reprints from British Medical Journal on hyponatraemia.

05 07 01 — Dr McConnell wrote to confirm that he had discussed the case with the CMO and DPHs. Each DPH had agreed to alert the paediatricians in their respective Board areas to the hazards of hyponatraemia (Enclosure 3).

26 07 01 — Mrs Burnside, Chief Executive, Altnagelvin, contacted the CMO to advocate a regional review (Enclosure 4). I remember seeing a reply from CMO agreeing to set up a regional Enquiry Group and that Dr Nesbitt would be a member.

14 01 02 I arranged for the CMO to view a presentation by Dr Nesbitt on hyponatraemia while she was visiting Altnagelvin to present accreditation to the Hospital HDSU.

09 04 02 I chaired a meeting of relevant clinical staff to revise the Action Plan of 12 06 01 in view of the publication of guidelines on hyponatraemia. A new Action Plan is being agreed between surgeons, anaesthetists, paediatricians (to follow).

Throughout this process I was struck by the wish of all concerned to learn from this death which is unique in their experience. I received full co-operation from all clinical staff who are extremely distressed by Rachel's death.

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I feel our response was rapid and directed towards specific action to prevent recurrence. The documentation attached details the action.

Dr R Fulton

11/04/02

Encs