From Dr M McCarthy
Senior Medical Officer

Date 1 June 2004

1 CMO

2 Secretary

3. Angela Smith

INTERVIEW WITH THE IMPARTIAL REPORTER ON THE DEATH OF LUCY CRAWFORD

Issue: **Further briefing material for Minister's attention in advance of the interview with the Impartial Reporter on Thursday 3rd June.**

Timing: **Urgent**

Recommendation: **That Minister notes additional briefing and revised Q & A.**

Background

Further to briefing on 28 May, Departmental officials met today with senior management from Sperrin Lakeland Trust to clarify the course of events following Lucy Crawford's death and the rationale underpinning action taken by the Trust. The issues discussed fall within four areas; the reporting and investigation of the death; communication between the Trust and the Crawford family; the scrutiny into Dr O'Donohoe's clinical practice; and status of current proceedings. Q&A and lines to take have been revised to take account of this morning's meeting.

Report and Investigation of Lucy's Death

1. Lucy's death was, as noted in previous briefing, reported to the coroner's office on 14th April 2000. Her death was also reported to Director of Public Health, WHSSB on the same day. Furthermore he was informed in writing in May 2000 that an internal case review was being conducted by the Trust.

2. The investigation of Lucy's death by the Trust comprised an internal case review in which Dr M Quinn, a paediatrician from Altnagelvin Hospital was invited to participate as an independent medical assessor. The terms of reference for the review, Dr Quinn's report, and the Trust's report are attached. (Appendix 3, 4 and 5). The Crawford family was not made aware of the review until October 2000.

3. On completion of the internal review the Trust initiated a number of changes in practice as outlined in Appendix 6.

Communication with Crawford Family

4. The Crawford family did meet with Dr D O'Hara, the pathologist who had conducted the post mortem on Lucy. They also met (on 5th May 2000) with

Dr O'Donohoe, the paediatrician responsible for Lucy's care in the Erne Hospital. It does not appear that Dr O'Donohoe's meeting with the family went well. He was not able to answer the family's questions about the cause of Lucy's death. Subsequent communication between the Trust and the family was made difficult because of this.

5. On 22nd September 2000 the Crawford family made a formal complaint against the Trust. This complaint was made through the Western Health and Social Services Council which was acting at the request of the Crawford family.

6. In October 2000 the Crawford's were advised that an internal review had been conducted. The Trust offered a meeting to discuss the findings of the review. Over the ensuing months the Trust offered on 5 occasions to meet the family but the offers were not accepted.

7. On 27th April 2001 the family's solicitor advised that legal proceedings would be taken against Trust. This was settled out of court in December 2003.

8. The Trust acknowledged that their communication with the family could have been improved and that in retrospect, if they had met with the family as soon as possible after Lucy's death, some of the communication problems could have been avoided. An apology to the family was issued by Mr Mills on 19 March 2004 (Appendix 7).

Dr O'Donohoe's Practice

9. Following the coroner's inquest Dr O'Donohoe's role in the care of Lucy Crawford has been referred to the General Medical Council for consideration. In relation to Dr O'Donohoe's professional and personal conduct between 2000-2002 the Trust has taken a number of actions to assure itself that

Dr O'Donohoe's practice does not put other patients at risk. A summary of actions is attached. (Appendix 8).

Current Proceedings

10. Following the coroner's verdict, the Trust will undertake a root cause analysis exercise into events around Lucy Crawford's death. The terms of reference have been agreed and are attached (appendix 9).

11. Further litigation brought by Mr Crawford against the Trust, following the inquest into Lucy's death, is ongoing and is expected to take some time.

Summary

12. In summary, the actions of the Trust have included the following:

- in the absence of a formal system for reporting untoward deaths, the Trust very appropriately informed the WHSSB, both verbally immediately after Lucy's death, and in writing writing in May 2000.

- There were several offers for the Trust to meet with the Crawford family but the Trust recognise that communication was an area in which they were not successful. The Trust did apologise to the Crawford family.

- In relation to Dr O'Donohoe's practice the Trust have appropriately scrutinised this and have involved professional bodies.

13. I attach the Q&A material (Appendix 1) in which some responses have been revised in light of this mornings discussions with the Trust's senior management.

DR MIRIAM McCARTHY

Senior Medical Officer

APPENDIX 1

QUESTIONS & ANSWERS

Q1 Are you happy with the investigation conducted by the Trust?

A1 The Trust acted correctly in conducting a case review into Lucy's death, which they commenced immediately after her death and completed within 3 months. To ensure an external perspective, the Trust involved a consultant paediatrician from another hospital. The Review made a number of recommendations and the Trust have taken action to implement these, including improved fluid management practice, and improved arrangements for documentation of prescribed fluids.

Q2 Do you think Lucy's death should be properly investigated by holding an independent inquiry?

A2 The cause of Lucy's death was fully and comprehensively investigated during the coroner's inquest. I accept the coroner's verdict and do not think that an independent inquiry will provide additional information.

I believe it is much more important that we invest our efforts in making sure that a similar tragic death does not happen again. In particular the guidance on the prevention of hyponatraemia issued by the Chief Medical Officer, and our current work to develop a system for reporting adverse incidents will help avoid a similar event in the future.

Q3 Was the Sperrin Lakeland Trust negligent in failing to report Lucy's death to your Department?

A3 No. At the time of Lucy's death there was no formal requirement for Trusts to report a death such as Lucy's to the Department. The Trust did however notify the Director of Public Health at the Western Health and Social Services Board immediately after Lucy's death and also formally advised him that a case review was being conducted. My Department has been working to strengthen arrangements for the reporting of adverse incidents and will shortly be issuing guidance.

Q4 Without a formal system how does your Department expect to hear of untoward deaths?

A4 The current system, in which untoward deaths are reported to the coroner's office provides a mechanism by which unexplained deaths are appropriately investigated. Each year, there are about 15,000 deaths in Northern Ireland, the majority of which occur in hospitals. Almost 3,500 deaths are reported to the coroner annually and approximately 1,400 coroners post-mortems are concluded.

The existing system of reporting deaths is to be strengthened. The Home Office proposals *Reforming the Coroner and Death Certification Service*, proposes that all deaths will be reported to the coroner's office through newly established medical examiners. This will be introduced over a number of years.

Q5 Are you satisfied with the quality care provided by the Trust?

A5 In light of the coroner's verdict I know that the quality of care received by Lucy was of the standard we would expect. I am however satisfied that lessons have been learnt and that appropriate steps have been taken to ensure a similar case does not occur again. These steps include action by the Trust to improve

practices and procedures, the CMO guidance issued to Trusts, and the adverse incident reporting system to be introduced shortly.

Q6 How many untoward deaths are reported to your Department each year?

A6 There are only a small number of deaths reported directly to the Department each year. I know that the Northern Ireland Adverse Incident Centre, which receives reports of any untoward incidents associated with medial equipment and devices, received 241 reports of incidents. Since 1998 there have only been 4 reported cases of deaths related to medical devices.

Q7 Why was Lucy's death not reported to the coroner?

A7 Lucy's death was reported to the coroner's office. Following discussion of the case between the state pathologist and a consultant paediatrician a decision was made that a coroner's post-mortem was not required. This decision was based on the available evidence at the time of Lucy's death.

Q8 The CMO is on record as stating that she knew about Lucy's death in 2001 then corrected her statement the next day. Can you comment?

A8 Let me emphasise the sequence of events. Lucy died in April 2000. Raychel Ferguson died in 2001 and her inquest was held in February 2003. It was only after the inquest into Raychel's death, in which a verdict of hyponatraemia was reached, that the coroner was made aware that Lucy and Raychel may have both died from hyponatraemia. Therefore the CMO was informed of Lucy's death in March 2003, and this she has confirmed on record.

Q9 When were you informed of Lucy's death?

A9 I was formally notified of Lucy's death following her inquest in March 2004. However, the Chief Medical Officer was informed in March 2003 and her office very appropriately brought Lucy's death and Rachel's to the attention of the National Patient Safety Agency, which is responsible for the safety of patients in the NHS.

Q10 Why was Dr Sumner not called in earlier by the Department?

A10 When CMO's guidance was being developed the working group included paediatricians, paediatric intensive care specialists, a specialist in laboratory medicine and a nurse. Dr Sumner, a well-recognised expert on fluid management and hyponatraemia, made a valuable contribution in formulating the guidance and he has recently praised it.

Q11 Will you apologise to Lucy's family for her death?

A11 I know that the Trust have apologised to the Crawford family for failings in the service. I too apologise for the tragic death of Lucy although I know that no words will ease the loss for her family.

Q12 Will you meet the Crawford family?

A12 Yes, I have written to Mr and Mrs Crawford and I have offered to meet with them.

Q12a Are you satisfied with the way the Trust communicated with the Crawford family?

A12a The Trust offered on a number of occasions to meet with the Crawford family and also communicated with them in writing. However in hindsight the Trust have acknowledged that communication could have been much better.

Q13 The CMO appears to disagree with the verdict of the coroner, can you comment?

A13 I want to emphasise that I fully and unconditionally accept the verdict of the coroner regarding the cause of Lucy's death. I also want to stress that the CMO has gone on public record to the Irish News and to yourself endorsing the findings of the coroner.

Q14 Do you think that Lucy's death was due to an idiosyncratic reaction to fluid?

A14 I am not of course a clinician. Hyponatraemia and its cause is a complex matter that I don't pretend to fully understand. What I want to put on public record is that I fully and unconditionally accept the coroner's verdict on Lucy's death. I do know that there is still ongoing debate about fluid management in children and specifically about the risk of hyponatraemia. The prevention and treatment of hyponatraemia is a complex area but I am content that the guidance issued by the CMO which is currently in place will ensure that hyponatraemia can and will be prevented in children.

Q15 Surely the doctor treating Lucy should have been aware of the possibility of hyponatraemia?

A15 Hyponatraemia was not as you have said in your articles, a widely known risk of fluid administration. In fact, there is still considerable debate among paediatricians regarding the most appropriate intravenous fluid therapy for children. The area of fluid administration in a sick child remains a complex area and within the past few weeks a series of articles published in the highly respected paediatric journal.

Q16 Was the doctor involved negligent?

A16 I am not responsible for the individual actions of doctors. The coroner has referred the papers in this case to the GMC and therefore it would be inappropriate to make any further comment.

Q17 There was an article on hyponatraemia in BMJ as far back as 1992, why did it take so long to introduce new guidelines?

A17 Yes, there were some articles on hyponatraemia but it was not something known widely. Following the death of Raychel Ferguson the Chief Medical Officer convened an expert working group as a matter of urgency to develop guidance on the prevent of hyponatraemia. This guidance was published in 2002 providing practical advice for doctors and nurses who manage the care of children in hospital. This guidance is the first of its kind in the UK and has been commended by Dr Sumner, an expert witness called by the coroner to Lucy's inquest.

Q18 There is another inquest into a young boy's death being held this week. Is this yet another death from hyponatraemia?

A18 There is an inquest currently being held and I am content that the coroner will fully investigate the cause of death. Until its completion I cannot comment on this inquest.

Q19 Was the Trust at fault for not alerting you to Lucy's death?

A19 It was not the Trust's fault but it does point out that there was a gap in the arrangements for informing me of such events. This was hampered by the absence of a formal system here or anywhere else in the UK to report untoward deaths within hospitals at the time of Lucy's death. In Northern Ireland there

are about 15,000 deaths each year, the majority of which occur in hospital. Approximately 3,500 of all deaths each year are reported to the coroner. Measures are being taken both by the coroner's office and by the health service to establish a system, which will identify untoward deaths and allow early action to be taken.

Q20 Should the Chief Executive of the Trust resign?

A20 I am satisfied that the Trust investigated the case properly and I see no reason for the Chief Executive to resign. What I would say, as you have quite rightly pointed out in one of your articles, is that the Erne Hospital is a fine one, with dedicated, able and professional staff.

Q21. Would the new arrangements you have outlined prevented events, such as the Lucy Crawford case, from happening?

A21. These arrangements, taken in conjunction with other initiatives will help to promote safety in the HPSS and should help minimise the risk of something going wrong and causing harm to a service user. But no system can offer a total guarantee that nothing untoward will happen. We can however make sure that through training, through good risk management, through governance and by independent inspection that the safety mechanisms designed to prevent such things from happening are as fail-safe as possible.

Q22. Why has it taken so long to make decisions on Best Practice-Best Care?

A22. Since the consultation exercise was completed in 2002, there have been developments taking place elsewhere which could have a direct impact on the proposals set out in "Best Practice - Best Care". Of particular significance were changes to the directions and legislation governing NICE and the Commission for Health Improvement. Consideration of such developments needed to be

taken account of before taking final decisions on the arrangements required for the HPSS.

Q23. What has been done?

A23 Legislation came into force in February 2003 placing a statutory Duty of Quality on all HPSS providers. From April next the new Regulation and Improvement Authority will be working to improve standards of treatment and care. Just last week, I announced that Brian Coulter would be the Chairman of the new body and work is now under way to get the new organisation established.

Q24. What is the duty of quality on the HPSS?

A24. By placing a statutory duty of quality on chief executives of HPSS organisations we will for the first time be able to ensure the quality of services delivered, in the same way that financial probity is adhered to. The introduction of clinical and social care governance will bring together all existing activity relating to the delivery of high quality services such as education, training, audit, risk management and complaints management.

Q25. To which organisations will the statutory duty apply?

A25. This statutory duty will cover both Health and Social Services and will apply to HSS Boards, HSS Trusts and those Special Agencies which provide services directly to users e.g. The Northern Ireland Blood Transfusion Agency.

Q26. What is Clinical and Social Care Governance?

A26. Clinical and Social Care Governance is a framework within which HPSS organisations are accountable for continuously improving the quality of their

services and safeguarding high standards of care and treatment. Clinical and Social Care Governance is about organisations taking corporate responsibility for performance and providing the highest possible standard of clinical and social care.

KEY MESSAGES

- The death of a child is tragic and I want to offer my most sincere sympathy to Lucy Crawford's family.

- I fully accept the coroner's verdict on the cause of Lucy's death.

- I acknowledge that my Department did not know of Lucy's death until 2003. In 2000 there was no formal system for reporting deaths such as Lucys. We have developed a mechanism for the reporting of untoward events in the Health Service and will be issuing interim guidance shortly.

- I am satisfied that the cause of Lucy's death was fully and comprehensively investigated by the coroner and I do not think that any further investigation is required.

- The circumstances surrounding Lucy's death and the subsequent inquest raised a number of important issues, which my Department is addressing, including the reporting of untoward events in hospitals, and good records management.

- It is important that we learn from the lessons of Lucy's death and we have done so. Following the death of Raychel Ferguson from hyponatraemia in 2001, the Chief Medical Officer acted immediately to develop guidance that would prevent a similar incident happening again. This guidance has been incorporated into clinical practice since 2002 and is currently being reviewed in light of the verdict on Lucy's death and any emerging evidence.

- Under clinical governance arrangements introduced last year, my Department is strengthening the systems for quality assurance with Trusts. In particular,

work is underway to improve the mechanism for reporting and investigating untoward incidents in hospitals.

- Accurate record keeping, found seriously lacking in Lucy's case is a very important matter within the health service. My Department is currently working to ensure that measures are in place to maintain good medical record keeping.

Distribution List

Dr Carson
Dr Mock
Mr Hamilton
Mr Hill
Mr Sullivan
Mr McCann
Dr Briscoe
Mr Shannon
Mr Mulhern

Appendix 12

Acute Services Directorate

[redacted]

21 April 2000

Dr Quinn
Consultant Pediatrician
Altnagelvin Hospital
Londonderry

Dr Quinn

Re: Lucy Crawford

Further to my telephone conversation I am enclosing for your information a copy of the notes of the most recent admission of the late Lucy Crawford.

I would be grateful for your opinion on the range of issues discussed which would assist Dr Anderson and my initial review of events relating to Lucy's care.

These were:

1 The significance of the type and volume of fluid administered.

2 The likely cause of the cerebral oedema.

3 The likely cause of the change in the electrolyte balance ie was it likely to be caused by the type of fluids, the volume of fluids used, the diarrhoea or other factors.

I would also welcome any other observation in relation to Lucy's condition and care which you may feel is relevant at this stage.

Can I thank you for agreeing to offer your assistance.

Yours sincerely

E Fee (Mr)
Director of Acute Hospital Services

DHSSPS

Appendix 1

DATE: 22 June 2000

Mr Eugene Fee
Director of Acute Hospital Services
Sperrin Lakeland Trust
Tyrone County Hospital
OMAGH
BT79 0AP

Dear Mr Fee

Medical Report on Lucy Crawford

I have reviewed the notes of this child as requested and will make a short summary and some comments on the possible sequence of events in this case.

Lucy had been admitted on 12.4.00 at around 19.30hours. Her G.P's letter stated that she had been pyrexic, not responding to Calpol, that she was drowsy and lethargic, that she was floppy and not drinking. He noted her temperature to be 38 C and wondered if she could possibly have a urinary tract infection. On admission the history revealed that the fever had been going for 36 hours and indeed that she had been vomiting for a similar period of time. She had been off her feeds to an extent of 5 days and that she was drowsy for about 12 hours. Her stools were reported to be normal. She had a temperature of 38 C on admission and was noted to be 9.14kgs. This would be around the 2nd centile for her age. Her capillary refill time was said to be > 2 seconds. Her abdomen was soft and bowel sounds were present. A diagnosis of viral illness was made.

Her urines were checked. A blood count revealed a somewhat raised WCC at 15 with 13000 of these being neutrophils. Urea & electrolytes were essentially normal apart from a raised urea at 9.9. It is reported that the taking of oral fluids by the child should be encouraged. An intravenous line was inserted at 23.00hours by a Consultant Paediatrician and solution 18 was started. It would appear that this continued at a rate of 100mls/hour over the next 4 hours. The child also drank about 150mls prior to this. At around 02.30hours the child passed a very large runny bowel motion and was transferred into a side room. At around 02.55hours of 13.4.00 the mother buzzed a nurse to say that the child was rigid. When the nurse saw the child she confirmed that it was rigid in the mother's arms and called a second nurse at around 0.300hours. Lucy's colour was recorded as being satisfactory and

her respirations were satisfactory. A junior doctor was bleeped at that stage and the child was turned on her side and given some oxygen. 2.5mgs of Diazepam was administered rectally. However it is recorded that within one minute of this a large bowel motion occurred and I suspect most of the Diazepam was expelled. On reviewing the child's electrolytes in and around that time it was decided that because the sodium was low that normal saline should be given. At 03.20hours it was noted the respiratory effort was decreased. An airway was inserted and the child was bagged with bag and mask. She was ultimately intubated by an Anaesthetist and Flumazenil, 100mcg was given. Her pupils were noted to be fixed and dilated. She was transferred to the intensive care in the Erne Hospital and ventilated in a high percent of oxygen. Mannitol 20% was given and intravenous Claforan.

At 06.30hours she was transferred to the Royal Belfast Hospital for Sick Children's ICU and I understand that she subsequently died.

I have subsequently been made aware that the Pathologist reported that the child had a significant pneumonia and cerebral oedema.

I will attempt to answer a few questions which obviously came up from reviewing the notes.

Why was the child noted to be floppy in the first place?

I suspect she may well have been quite ill on admission. The raised WCC with a predominance of neutrophils may go along with a bacterial infection and could have been due to the pneumonia which was found on P.M. However as stated before this is speculation.

Was the child dehydrated on admission?

I think the urea measurement of 9.9 on admission does indicate a degree of dehydration. This level of urea would certainly not go with renal failure.

Fluids.

She was treated with Solution 18 which would be appropriate. On looking at the volume of fluids over the 7 hour period between admission and 3.00a.m. when she had the possible seizure she got a total of 550mls. This would include 150mls oral and 400mls i.v. as the intravenous drip was running at 100mls/hr over a 4 hour period. Calculating the amounts over that period of time this would be about 80mls/hr. I

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have calculated the rates of fluid requirements. If she was not dehydrated she would have required 45mls/hr. If she was 5% dehydrated it would have worked out at 60mls/hr and 10% dehydration works out at 80mls/hr. I would therefore be surprised if those volumes of fluid could have produced gross cerebral oedema causing coning. I have however noted that there was no prescription written for the fluids indicating the volume per hour that should be given.

Was there evidence of renal compromise?

I have noted that there was a urinary output and that there was no oedema of the face or peripheries noted. Ward testing of the urine showed some protein and ketones. However lab testing did not confirm proteinuria. The ketones would certainly be present in any child who is not eating well or indeed is vomiting.

Did the child have a seizure or did she "cone" at 3.00a.m?

I feel it is very difficult to say what happened in and around this time. It is certainly possible that she had a seizure and may even have had a period of time when she was hypoxic before medical attention was drawn to the fact she was unwell. However I cannot say that this is the case. It may be that mother informed the ward staff immediately she noted the problem but again this is not clear to me from the notes provided.

Apnoea.

This could have occurred as the result of a seizure. It could have occurred as a result of coning. I have looked at the possibility that it could have been due to medication with rectal Diazepam. I note the child was given 2.5mgs but it was stated that within one minute of administration of this she had a large bowel motion and I presume most of the Diazepam actually came out. Certainly the recommended dose of Diazepam that can be given to a child who is seizing is 500mcg/kg. Therefore she could have been given up to 4.5mgs and certainly 2.5mgs given rectally to this age of child for a seizure would be appropriate. I am aware that some child have idiosyncratic reactions to Diazepam but normally this would be if they are given by the intravenous route and these events are very rare.

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Was the resuscitation adequate?

The notes state that the child had a good heart rate and colour throughout this event and that initially the child's respirations were adequate. Obviously when she became apnoeic in and around 03.20hours she required an airway insertion and bagging and she was ultimately then intubated by an Anaesthetist. During resuscitation it obviously became apparent that the child's sodium had dropped to 127 and potassium down to 2.5 and a decision to use normal saline was made. I am not certain how much normal saline was run in at that time but if it was suspected that she was shocked then perhaps up to 20mls/kg could have been given.

I hope these comments are helpful. I find it difficult to be totally certain as to what occurred to Lucy in and around 3.00a.m. or indeed what the ultimate cause of her cerebral oedema was. It is always difficult when simply working from medical and nursing records and also from not seeing the child to get an absolutely clear picture of what was happening. However I hope I have attempted to be as objective as possible with the information available to me.

Yours sincerely

R J M QUINN, MB, FRCP, DCH, MFPaedRCPI
Consultant Paediatrician

DHSSPS

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REPORT RE: THE REVIEW OF LUCY CRAWFORD'S CASE

BACKGROUND

On Friday 14 April 2000 Dr O'Donohoe, Consultant Paediatrician advised Dr Kelly, Medical Director, that 17 month old Lucy Crawford had been admitted to the Children's Ward, Erne Hospital on Wednesday 12 April 2000. She was admitted at around 7.30pm and had deteriorated rapidly early on 13 April 2000 morning. This deterioration in Lucy's condition led to emergency resuscitation within the Paediatric Department, a transfer to the High Dependency Unit, Erne Hospital, and a subsequent transfer to the Royal Belfast Hospital for Sick Children's Intensive Care Unit, where she died.

In light of the unexpected development and outcome of Lucy's condition it was agreed that a review would be established in keeping with the developing arrangements for Review of Clinical Instances/Untoward Events. This review has been conducted by Dr Anderson, Clinical Director, Women & Chilren's Directorate and Mr Fee, Director of Acute Hospital Services with an input from Dr Kelly, Medical Director. External assistance and advice was made available by Dr Quinn, Consultant Paediatrician, Altnagelvin Hospital.

PURPOSE OF REVIEW

The main purpose of the review was to trace the progression of Lucy's illness from her admission to the Erne Hospital and her treatments/interventions in order to try and establish whether:

a) There is any connection between our activities and actions, and the progression and outcome of Lucy's condition
b) Whether or not there was any omission in our actions and treatments which may have influenced the progession and outcome of Lucy's condition
c) Whether or not there are any features of our contribution to care in this case which may suggest the need for change in our approach to the care of patients within the Paediatric Department or wider hospital generally

PROCESS OF REVIEW

1. The case notes were reviewed
2. All staff within Sperrin Lakeland Trust who had an involvement in Lucy's care were asked to provide a written comment/response of their contribution to Lucy's care
3. Some separate discussions were held with Sister Traynor (appendix 11) and Mrs Martin, Infection Control Nurse
4. Dr Quinn, Consultant Paediatrician, Altnagelvin Hospital, was asked to give his opinion on 3 specific issues. A copy of the patient's notes were made available to Dr Quinn
5. The outcome of the postmortem was considered
6. A meeting was held between Dr Kelly, Dr Quinn and Mr Fee on Wednesday 21 June 2000 to share with him the result of the autopsy and seek his comment and a formal response on the issues raised. Dr Quinn's report dated 22 June 2000 is included as appendix 1.

FINDINGS

Lucy Crawford was admitted to the Children's Ward, Erne Hospital on 12 April 2000 at approximately 7.30pm having been referred by her General Practitioner. The history given was one of 2 days fever, vomiting and passing smelly urine. The General Practitioner's impression was that Lucy was possibly suffering from a urinary tract infection. The patient was examined by Dr Malik, Senior House House Officer, Paediatrics, who made a provisional diagnosis of viral illness. She was admitted for investigation and administration of IV fluids. Lucy was considered to be no more or less ill than many children admitted to this department. Neither the postmortem result or the independent medical report on Lucy Crawford, provided by Dr Quinn, can give an absolute explanation as to why Lucy's condition deteriorated rapidly, why she had an event described as a seizure at around 2.55am on 13 April 2000, or why cerebral oedema was present on examination at postmortem.

ISSUES ARISING

1 Level of Fluid Intake

Lucy was given a mixture of oral fluids and intravenous infusion of solution 18 between her admission, at around 7.30pm on 12 April 2000, and the event that happened around 2.55am on 13 April 2000. Dr Quinn is of the view that the intravenous solution used and the total volume of fluid intake, when spread over the 7 ½ hour period, would be within the accepted range and has expressed his surprise if those volumes of fluid could have produced gross cerebral oedema causing coning.

There was no written prescription to define the intended volume. There was some confusion between the Consultant, Senior House Officer and Nurses concerned in relation to the intended volume of fluid to be given intravenously. There is a discrepancy in the running total of the intravenous infusion of solution 18 for the last 2 hours. There is no record of the actual volume of normal saline given when commenced on a free flowing basis.

2 Level of Description of Event

Retrospective notes have been made by nursing and medical staff in respect of the event which happened at around 2.55am on 13 April 2000. In all of these descriptions and the subsequent postmortem report the event is described as a seizure. With the exception of Nurse McCaffrey's report, little detailed descriptions of the event are recorded and no account appears to be in existence of the mother's description who was present and discovered Lucy in this state.

3 Reporting Incident

While a procedure for reporting and the initiation of an investigation into Clinical Instances/Untoward Events was not in existence universally, at the time of Lucy's admission to the Erne Hospital, Dr O'Donohoe proactively reported the unexpected outcome of Lucy's condition to Dr Kelly, Medical Director.

4 Communications

The main communication issue identified within this review was the confusion between all those concerned in relation to the intended prescribed dosage of intravenous fluids. The record shows that Dr O'Donohoe's intention or recollection was that Lucy should have 100mls bolus of fluids in the first hour and 30mls hourly thereafter. While the Nursing staff held a clear view that the expressed intention was to give 100mls hourly until Lucy passed urine. Furthermore this was considered by the Nursing staff interviewed to be a standard approach in such circumstances. This clearly demonstrates the need for standard protocols for treating such patients and the need, in keeping with required practice, to have a clearly written prescription.

5 Documentation

The main issues identified here are the need for clearly documented prescriptions for intravenous fluids, the accurate documentation of the fluid administration, and the need to document patients or parents descriptions of unusual clinical events, such as the seizure, describing the detail which may be required at a later date.

6 Care of Family

Mrs Doherty, Health Visitor, and Dr O'Donohoe were proactive in offering support to the family and given the opportunity to explain where possible the reasons for the change in Lucy's condition and support them in their bereavement.

7 Team Support

All team members involved in Lucy's care were shocked and traumatised by the unexpected deterioration in her condition. A team briefing consisting of all disciplines did not take place. Such a process may help support those concerned and reduce the fear of attempts to apportion blame between team members.

8 Linkage with the Regional Centre

A number of issues arose in respect of our link with Regional Services in this case. These included the arrangements to support the transfer of such patients, the need for greater communication between the local hospital and the regional hospital in respect of feedback which is to be given to parents in such instances and the significant time delay in getting access to the final postmortem report.

9 Recommendations

a) the need for prescribed orders to be clearly documented and signed by the prescriber

b) the importance for standard protocols to be readily available in the ward against which treatment can be compared

c) that all team members involved in the care of the child, on the night in question, would probably benefit from a joint meeting and discussion of this report/findings; and

d) that it would be appropriate for another meeting with the family to appraise them of all of the knowledge and opinions that we have at this point. Whilst we are not in a position to give them definite answers we may at least be able to demonstrate our openness and show to them the measures that have been taken to analyse the care of Lucy's admission.

31 July 2000

FEB 2004.

CHANGES TO CARE

- SINCE APRIL 2000

⊙ Documentation

• There has been update training for ALL Nursing staff on Records and record keeping to NHC standards

• There is also ~~[illegible]~~ audit system now in place in relation to missing records.

⊙ observations

• In June 2000 a formal an Emergency Admissions Policy was in place
This specifies minimal observation standards within the first 24 hours of admission

⊙ Weighing

• Children's weights are now double checked by a second person

• If children (toddlers) are very restless and are weighed with a parent this is recorded and as soon as possible later is reweighed individually.

• Weighing Scales are serviced/recalibrated if necessary every 6 months as per manufacturers instructions. If there is considered to be problems in between then a 24 hours response time to request. There was no such ~~response~~ request during April, May, & June 2000.

⊙ Fluids

• Soln 18 is not used as any first line I.V. fluids on the ward.

• The DHSSPS Wall chart "Risk of Hyponatraemia" is prominently displayed on the wall in the Treatment Room where I.V. fluids are erected.

• All children on I.V. fluids have Electrolyte estimation done 4-6 hourly

• Medical notes (Core) now have a section set out for fluid replacement 'Calculations' before any child is commenced on I.V. fluids

[illegible]

[illegible]

[illegible]

DHSSPS

075-013-060

SPERRIN LAKELAND
HEALTH AND SOCIAL CARE TRUST

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Minicom Service is available within the Trust on request

Date: 19th March 2004

Mr & Mrs Neville Crawford
[redacted]

Dear Mr and Mrs Crawford

I am writing on behalf of the Trust to indicate our regret and apologies for the failings in our service at the time of Lucy's death in April 2000. These failings, not fully identified in our original review became evident later in the process following another reported death in Northern Ireland. At that time we sought, through your legal representatives, to reach settlement on the legal proceedings.

We cannot presume to lessen the grief of your loss. However please be assured that the procedures linked to Lucy's death were reviewed and changed. Furthermore we fully intend to formally reflect on the findings of the Coroner to ensure that we, and others, learn the lessons of Lucy's tragic death.

It remains a matter of regret to the Trust that the opportunity to discuss these matters more openly with you was complicated by legal processes.

I trust that even at this time that the sentiments expressed above may offer some reassurance.

Yours sincerely

Hugh Mills
Chief Executive
bor/mmcg/0652

Trust Headquarters, Strathdene House, T&F Hospital, Omagh, Co Tyrone, BT79 0NS
Telephone Number: [redacted]
Fax Number: (02[redacted]
E-mail: [redacted]
Minicom Service is available within the Trust on request

Our ref: ***LS 3***
Date: ***30 April 2004***

Dr H Campbell
Chief Medical Officer
DHSSPS
Castlebuildings
Stormont Estate
BELFAST
BT4 3SQ

Dear Dr Campbell

Thank you for your letter of 01 April 2004. I apologise for the delay in responding.

Attached you will find the information, compiled by Jim Kelly on the response of the Trust to concerns relating to Dr Jarlath O'Donohoe's professional and personal conduct 2000-2002. Jim has also provided information on the contribution Jarlath has made to our Paediatric service since this time.

Currently Dr O'Donohoe is on sick leave, however I have received a report from our Occupational Health Consultant recommending his return to work for teaching and non-clinical duties. I am discussing this with Dr Diana Cody, current Medical Director and Eugene Fee.

One of the specific issues for the Trust which we mentioned at our meeting was the Trust's process for selection of other Consultants to provide external opinions for the type of internal review we conducted following Lucy Crawford's death. We feel it would be essential that arrangements are established for the identification of suitable clinicians, some of whom may be from outside N. Ireland. I would welcome your views on whether such panels would be co-ordinated by your office and if this will be part of the Department's guidance on "Adverse Incidents: Notification and Follow up Action".

RESPONSE TO CMO ~ KEY POINTS

In response to concerns raised relating to Dr. O'Donohoe's professional and personal conduct during the period 2000 – 2002 the following actions were taken by the Trust.

- Other key senior professionals (medical and nursing) within the paediatric service were interviewed to ensure children were not being put at risk. In particular the lead paediatrician during this period (Dr. Halahakoon) and the senior sister confirmed that they did not feel Dr. O'Donohoe's practice was unsafe.
- A number of consultant locums supported the two permanent paediatricians during this period. None of these locums expressed concerns relating to the professional competency of Dr. O' Donohoe
- The LC case was reviewed by Dr. Murray Quinn who alongside his report provided the opinion that there was no requirement for precautionary suspension
- Cases giving rise to initial concern, including the LC case, were formally reviewed through the Royal College of Paediatricians at the request of the Trust (Dec 2000) and a report provided to the Trust by the RCP nominee Dr. Moira Stewart RVH in May 2001. The Medical Director met with Dr. Stewart (Jun 2001) to check aspects of the report and to clarify if any deficiencies warranted precautionary suspension or referral to the GMC. The advice was that this action was not required.
- A formal health check through Occupational Health was undertaken to ensure there were no medical issues of concern.
- The Trust sought advice from CSA legal advisors (Nov 2001) on correct steps relating to ongoing risk management of the concerns.
- The WHSSB was advised of the LC case in May 2000 and the report from Dr. Quinn. They were additionally advised in 2001 of the RCP involvement and the subsequent report provided. Detailed discussions took place between the Medical Director and the Dir. Of Public Health Dr. Mc Connell on next steps in managing ongoing concerns.
- The Royal College of Paediatricians was asked in Feb 2002 to provide a more in depth assessment of Dr O'Donohoe's competence and practice because of further concerns raised by a Staff Grade in paediatrics. The college detailed Dr. Stewart (RVH) and Dr. Boon (Royal Berkshire Hospitals) to investigate and provide advice to the Trust. The two paediatricians visited the Trust on the 24th & 25th of June 2002 interviewing a wide variety of staff including a local General Practitioner, reviewing clinical notes and previous incident reports and viewing the clinical environment. After their visit they met with the Chief Executive and the Medical Director to advise a number of proposed actions but advised that GMC referral or suspension was not required.
- The Trust worked with the WHSSB 2002/2003 to increase investment in the paediatric team and successfully recruited three permanent paediatricians during 2003, redefined roles and responsibilities of the paediatricians and increased secretarial support.
- Dr.. O'Donohoe during the period 2003 – 2004 was acting as lead paediatrician for the Trust and demonstrated significant commitment to the change agenda including introduction of new policies and procedures. He was very active in enhancing junior doctor training and led the Trust through a successful RCP training inspection Dec 2003. He has actively participated in appraisal.
- There were no issues of concern relating to professional competence or personal conduct raised since 2002 and all his colleagues are keen to see him return to work as soon as possible. (Dr. O' Donohoe is currently on sick leave)

Dr J Kelly, Medical Director 2000-2003

21 April 2004

DRAFT 5

SPERRIN LAKELAND

HEALTH AND SOCIAL CARE TRUST

ROOT CAUSE ANALYSIS EXERCISE : LC Case

TERMS OF REFERENCE

Background:

On 20/02/04 the Coroners Inquest concluded its findings on the circumstances nature and cause of the tragic death of Lucy Crawford. Aspects of the clinical care are currently subject to consideration by the GMC, after referral by the Coroner. The Trust is co-operating fully with the GMC in this regard.

It has been acknowledged, in the course of the management of this case, that a number of process and systems issues warrant examination and reflection.

This proposed Root Cause Analysis (RCA) exercise is being commissioned for this purpose.

Principles:

This exercise will be:

- overseen by a Steering group established by the Trust Chairman (membership set out below)
- undertaken in a manner to provide independent analysis
- focused on the Trust's process and systems, as per the agreed scope set out below
- used to inform regional authorities, as appropriate, of any relevant/pertinent lessons for wider dissemination
- undertaken in a way to ensure early transference of lessons emerging from the analysis rather than await final report production.

Scope:

The root cause analysis will examine:

- adverse incident investigation process
- complaints handling process
- litigation process (including preparation for Inquest)
- media/public relations processes and
- related cpd/cme processes regarding updating of professional standards
- Key staff involved in the processes set out above will be invited to participate and contribute to the RCA exercise
- Currently the Trust is approaching the family to assess their preparedness to engage with this process
- Findings for the RCA will be presented to the Steering group along with any recommended remedial actions.
- A final report will be provided to the Trust Chair and Chief Executive and the CSCG committee for adoption.

Membership of Steering Group:

The group will be chaired by a Non Executive Director of the Trust. The following additional members have been identified to secure independent views, a consumer perspective and professional overview:

- Trust Medical Director
- Chief Nurse, WHSSB
- Chief Officer, WHSSC
- Representative of the CSCG Support team

Process & Resources:

- External expertise on RCA methodology will be sourced via the NI CSCG support team. The Trust will meet costs in this respect.
- Guidance and support will be provided by the CSCG support team representative – costs for this will be met by the Director of the NI CSCG support team.
- Limited administrative support will be provided by the Corporate Affairs directorate through the CSCG Project Officer.
- A workplan will be agreed with the RCA Consultant(s) at an early stage. This will include:
    - Core groups for engagement/participation
    - Timescales/key timelines
    - Reporting arrangements

Timescales:

- The exercise should be completed within 4-6 months of initiation.

bor/mmcg/0702

DEBATE

Maintenance fluid therapy

What routine intravenous maintenance fluids should be used?

N P Mann

An introduction to the debate

Intravenous maintenance fluid is widely used in general paediatric practice and more children who come into hospital receive intravenous fluid than in the past. The intravenous route is frequently used because enteral maintenance or rehydration treatment is more labour intensive and uses valuable staff time; furthermore modern pumps for delivery of fluids are safe. Nevertheless in developing countries the enteral route is still more widely used even for sick dehydrated children.

Are there are any dangers of intravenous fluids? Clearly there is a possibility of miscalculation of infusion rates and also the potential for mistakes in terms of dosing errors with additives. It has been widely recognised in recent years that there is a high incidence of hyponatraemia in children treated with intravenous maintenance fluids. Is this because of excessive water or too little salt?

Moritz and Ayus discussed the high frequency of hyponatraemia in these children in their paper in *Pediatrics* in February 2003.[1] They suggested the use of isotonic saline rather than use of hypotonic fluids for maintenance therapy. More than 20 years ago there were concerns about profound neonatal hyponatraemia causing neurological problems in infants as the result of either excessive or the wrong kind of fluid given to mothers during labour.[2]

It is therefore timely to revisit this problem. Two experts have been asked to give their views to encourage further debate (see accompanying articles[3 4]). Do write to *ADC* with your comments about how paediatric practice in this area can be improved.

Arch Dis Child 2004;89:411

Correspondence to: Dr N P Mann; Npmann2@aol.com

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Maintenance fluid therapy

Pouring salt on troubled waters

D Taylor, A Durward

The case for isotonic parenteral maintenance solution

Intravenous fluid and electrolyte therapy for acutely ill children has been a cornerstone of medical practice for well over 50 years. The scientific methodology behind fluid regimens generated much debate in the early 1950s following the pioneering work of Darrow, Talbot, Gamble and others who recognised the important relation between caloric expenditure and requirements for water.[1]

Caloric expenditure was originally calculated according to body surface area, which at the bedside required either tables or nomograms.[2] In 1957 Holliday and Segar simplified this approach, relating energy expenditure to one of three weight based categories (<10 kg, 10–20 kg, >20 kg).[3] Electrolyte requirements were also calculated on a weight basis, producing an "ideal" hypotonic solution comprising 0.2% saline in 5% dextrose water (0.18% saline in 4% dextrose in the United [illegible] subsequently adopted on a global scale and is recommended in current paediatric and medical textbooks.

Advances in our understanding of water and electrolyte handling in health and disease have called into question the validity of the Holliday and Segar approach. Specifically, many authors have reported how hypotonic maintenance fluid may result in iatrogenic hyponatraemia in hospitalised patients, often with devastating consequences.[4–10] In this article we re-evaluate each of the concepts on which this traditional regime is based (energy expenditure, and water and electrolytes requirements) and use this to make the case for an alternative, namely isotonic fluid.

PITFALLS OF THE WEIGHT BASED HOLLIDAY AND SEGAR APPROACH

Energy expenditure

Talbot originally estimated basal metabolic rate in children based on water [illegible] by presenting *total* energy requirements (basal metabolic rate plus growth and activity) using this data to relation to body surface area (fig 1). Holliday and Segar further advanced this by indexing energy expenditure to body weight rather than surface area, assuming 1 ml of water loss was associated with the fixed consumption of 1 kilocalorie.[3] The typical fluid losses for children (table 1) thus equate with an energy requirement of 120 kcal/kg/day for a 10 kg child.[10]

There are two main flaws with this approach. First, it is now known that resting energy expenditure is closely related to *fat free mass* which includes muscle and the four major metabolic organs (heart, liver, kidneys, and brain).[10] Eighty per cent of the resting energy expenditure is accounted for by these four organs which comprise only 7% of total body mass. As a result, the use of weight alone to calculate energy expenditure may significantly overestimate caloric requirements. On average, the weight based method overestimates energy requirements in infants by 14% compared to the surface area method (fig 1).[4] Second, energy expenditure in healthy children, on whom historic models are based, is vastly different in acute disease or following surgery. Using calorimetric methods, energy expenditure in these patients is closer to the basal metabolic rate proposed by Talbot, averaging 30–60 kcal/kg/day.[11] This overestimate is multifactorial, ill

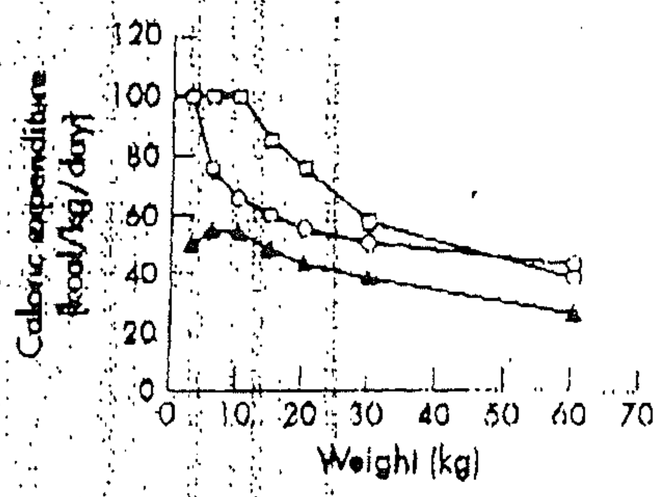

Figure 1 Daily caloric expenditure according to the weight based method of Holliday and Segar and by surface area method of Crawford, and basal metabolic rate. Comparison of two different methods for calculating caloric expenditure across weight ranges (open squares = Holliday and Segar's weight based method; open circles = Crawford's surface area method*; referenced against basal metabolic rate**).

patients are catabolic, often relatively inactive, and, in the intensive care environment may be pharmacologically sedated or muscle relaxed.[14–17] Almost half of the caloric intake suggested by Holliday and Segar is designated for growth, an unrealistic goal in acute disease.[18] Although fever and sepsis per se may increase metabolic rate this is usually limited to less than 1.5 times the basal metabolic rate, burns being an exception.

Water requirements

Historically water requirements have been based on crude estimates of both insensible (skin, respiratory tract) and sensible (urine and stool) water losses.

Insensible water loss

This was generously estimated at 930 ml/m^2/day (27 ml/kg/day).[19] Recent data suggest the true figure may be only half of this, with basal insensible losses from the skin being 250 ml/m^2/day (7 ml/kg/day) and via the respiratory tract 170 ml/m^2/day (5 ml/kg/day).[19] Additionally many other risk factors may reduce insensible water loss such as use of humidifiers in ventilated patients (80% reduction in respiratory water loss) or a thermo neutral environment.[17] Bluemle *et al* have shown insensible water losses of as little as 330 ml/m^2/day (10 ml/kg/day) in catabolic acute renal failure patients.[20]

*Crawford calculated caloric expenditure based on the calories utilised per surface area of the body. The calculated caloric expenditure at each body surface area increment can be converted to weight by cross-regression sur-

Table 1 Typical water losses per 100 kilocalories (kcal) of energy expended for a healthy 10 kg child

Source of water loss	Estimated water loss (ml per 100 kcal/day)
Insensible	
Skin	30
Respiratory	15
Sensible	
Stool	10
Minimal sweating	10
Urine	50
Total	115

Data calculated according to Pickering and Winters.[19]

Urinary loss of water

According to Holliday and Segar, urinary water losses for healthy children amount to 50–60 ml/kg/day[4] based on the work of Pickering and Winters (table 1).[19] The basis of this fluid regime was the observation that 15/28 infants and 20/25 children (unspecified diagnoses) who were given intravenous dextrose produced urine with an "acceptable" urine osmolarity between 150 and 600 mosm/l H_2O.[4] They presumed patients with dilute urine received too much water and conversely those with concentrated urine too little water.

Today we recognise this does not take into account the overriding influence of antidiuretic hormone (ADH) on urine flow rate.[21] When ADH is present, the renal solute load is effectively excreted in a smaller urine volume producing concentrated urine. Under these conditions urine output is often less than half the values observed in healthy children (approximately 25 ml/kg/day).[22] An increase in ADH is common during many childhood diseases, in response to stress (pain, fever, surgery) or secondary to use of opiates and non-steroidal anti-inflammatory drugs.[23–25] Under these conditions the administration of free water frequently leads to hyponatraemia because the kidneys are unable to excrete the water load.[3 6 26] Interestingly, the type of fluid administered may influence ADH levels. Judd *et al* showed that 0.9% saline but not 5% dextrose reduced ADH concentrations postoperatively.[27]

Thus the total fluid loss (sensible plus insensible) during acutely illness or following surgery may amount to approximately half that suggested by 15 ml/100 kcal burnt.[4] Thus, all these factors need consideration when assessing overall water balance.

Electrolyte requirements

To healthy breast fed infants Holliday and Segar computed a dietary sodium intake of 1 mEq/100 calories per day.[4] Darrow recommended 3 mEq of sodium per 100 calories of energy expended per day.[4] This is based on urinary excretion rates of sodium in healthy, milk fed infants. However, daily electrolyte requirements in disease may differ considerably from this. For example, large urinary losses of sodium and potassium may occur through the phenomenon of desalination.[27 28] Furthermore, Al-Dahhan *et al* showed a beneficial effect on neurodevelopmental outcome from doubling the daily sodium intake (4 to 5 mmol/kg) in neonates.[29] This refutes the assumption that the neonatal kidney is incapable of "handling" a high sodium load. The recent discovery of the most potent natriuretic hormones, urodilatin and gut-related natriuretic peptide has also shed new light on sodium regulation.

The rationale behind the traditional approach is to balance sodium intake to match sodium loss. However, this fails to appreciate the *single* most important role of sodium in acute illness, namely maintenance of plasma tonicity.[25 26] There is a strong inverse relation between plasma sodium concentration and intracellular volume.[30] Cell membranes are permeable to water but not electrolytes. As sodium is the major extracellular cation (and hence osmole), it regulates the movement of water across cells along an osmotic concentration gradient, thus explaining cellular swelling in the presence of hyponatraemia.

It is also important to recognise the role of potassium in the regulation of tonicity balance. Potassium is a major intracellular osmole, and may directly influence extracellular sodium concentration by altering the distribution of water between fluid compartments.[25] Potassium loss, both urinary and stool, may be significant in disease; yet its direct influence on serum sodium concentration is often not considered.[31 32]

Tonicity of intravenous fluids

It is crucial that clinicians appreciate the difference between osmolarity and tonicity. The osmolarity of a solution is the number of osmoles of solute per litre of solution. The tonicity of a solution refers

NB

Table 2 Approximate sodium concentration, in vitro osmolarity, in vivo tonicity, and theoretical volume of electrolyte free water (EFW) provided by commonly used intravenous solutions

Intravenous solution	Sodium* (mmol/l)	In vitro osmolarity† (mOsm/l H_2O)	In vivo tonicity‡ (mOsm/l H_2O)	Volume of EFW§ per litre infused
5% dextrose	0	286	0	1000
0.18% saline in 4% dextrose	30	300	60	824
0.45% saline	75	150	154	500
0.45% saline in 5% dextrose	75	432	150	500
0.9% saline	154	308	308	0
0.9% saline in 5% dextrose	154	586	308	0

*The apparent discrepancy between the in vitro sodium concentration (0.9% saline) of 154 mmol/l and the in vivo plasma sodium of 144 mmol/l is due to the phenomenon of pseudohyponatraemia. In human plasma, approximately 7% of the plasma volume is occupied by albumin and lipid, falsely lowering the true sodium concentration plasma by 10 mmol/l (7% of 155).
†In vitro osmolarity refers to the number of osmoles of solute per litre of solution.
‡In vivo tonicity refers to the total concentration of solutes which exert an osmotic force across a membrane in vivo (excludes the osmotic effect of dextrose because it is rapidly metabolised in blood).
§Calculated on the basis that electrolyte free water distributes to the intracellular and extracellular space in a ratio of 2:1.

is equal to that of electrolyte free water, as it contains no salt or other active osmole (zero tonicity). Every litre of 5% dextrose infused results in the expansion of the intracellular and extracellular fluid space by one litre (two thirds of this distributes to the intracellular space and one third to the extracellular space). Similarly, for every litre of 0.18% saline in 4% dextrose water infused, only 1/5th (200 ml) is isotonic to plasma (table 2). The remaining 800 ml is electrolyte free water, which will expand the intracellular fluid compartment. This is particularly relevant if excretion of water is limited by ADH.[3 7 21 * 31] This fluid shift may even occur in the absence of hyponatraemia.[32] Small increases in tissue water through the use of hypotonic fluids may be harmful in conditions such as cerebral oedema where minor increases in cerebral water may lead to disproportionately large increases in intracranial pressure.

The incidence and neurological complications of acute hyponatraemia

Hyponatraemia is a common biochemical finding in hospitalised children and is most commonly due to excess water intake rather than salt loss.[3 7 22 15] Shann and Germer showed an incidence of hyponatraemia (Na ≤134 mmol/l) as high as 45% in hospitalised children with pneumonia and 30% in bacterial meningitis.[1] Hanna *et al* recently reported a 30% incidence of admission hyponatraemia in infants with bronchiolitis requiring intensive care admission in the United Kingdom, 13% of which had seizures.[7] Halberthal *et al* was able to show a direct link between hyponatraemia and the use of hypotonic maintenance fluid.[7] The neurological complications of acute hyponatraemia include encephalopathy with seizures, irreversible brain damage, or brain death from cerebral herniation.[3 10] Children are also among the most susceptible to hyponatraemic brain injury.[3 6] Fatal hyponatraemia can occur within hours of hypotonic fluid administration, particularly if standard fluid maintenance rates are used (100–120 ml/kg/day).[10]

THE RATIONALE FOR ISOTONIC MAINTENANCE FLUID

The paramount consideration in the choice of intravenous fluid is the requirement to maintain serum sodium at a normal level. The use of isotonic solutions such as 0.9% saline is more appropriate in acutely sick children as they do not theoretically expand the intracellular fluid space. Isotonic solutions preserve intracellular function and integrity, by minimising changes in plasma sodium concentration and tonicity.

Use of 0.9% saline as maintenance fluid, if combined with appropriate fluid restriction, will result in a two to threefold increase in daily sodium intake compared to the traditional regime. However, the concern that this may cause severe hypernatraemia is without foundation because the sodium concentration and tonicity of 0.9% saline is similar to plasma. Andersen *et al* showed a rise in plasma sodium only after intravenous administration of hypertonic 3% saline but not 0.9% saline, despite a temporary positive sodium balance.[33] Heer *et al* showed chronic sodium loading in volunteers does not produce an increase in plasma sodium, body water, or weight as previously suggested.[34] Many of the historical assumptions concerning sodium handling are based on salt depleted subjects. Indeed massive sodium loads from large volume resuscitation of infants and children with sepsis (80–180 ml/kg/day) using 0.9% saline did not produce hypernatraemia.[35] Additionally an epidemic of hypernatraemia has not been documented in hospitalised adults where isotonic maintenance fluids are routine. When present, the aetiology of hypernatraemia in this scenario is frequently due to well recognised factors such diabetis insipidus or over-use of loop diuretics.[14]

The debate as to the optimal isotonic fluid is ongoing. For example, Hartman's solution has a more physiological concentration of chloride than 0.9% saline and hence does not cause hyperchloraemia. The benefit of Hartman's solution versus 0.9% saline is not currently known. It is important to stress that dextrose may be added to these isotonic solutions (commonly in concentration of 5–10%), when clinically indicated to avoid hypoglycaemia without changing the solution's in vivo *tonicity* (table 2). Recent evidence suggests that a 1% dextrose solution following uncomplicated paediatric surgery may be adequate.[37] A suitable solution for neonates and infants is 0.9% saline in 5% dextrose water, which is commercially available. We advocate 0.9% saline (with or without added dextrose) as a safe maintenance solution, both perioperatively and in the acute phase of most childhood illnesses requiring hospitalisation (for example, pneumonia, bronchiolitis, and meningitis). Here, the water retaining effect of antidiuretic hormone may necessitate a moderate degree of fluid restriction (50–60%) to prevent fluid overload. The concept of fluid maintenance should not be confused with replacement therapy where abnormal or excessive quantities of water and electrolytes may be lost. In this instance the biochemical composition and tonicity of the replacement solution should approximate that which is lost.

CONCLUSION

We have shown a number of pitfalls in the Holliday and Segar approach to parenteral therapy, namely that it focuses on fluid and electrolyte requirements for healthy children. In acute disease or following surgery, caloric expenditure, insensible water losses, and urine output are frequently much less than in health (often 50–60% of the reference values). Furthermore, this approach fails to recognise the importance of tonicity with its central role in the distribution of water between fluid

compartments (intracellular and extracellular space).

We therefore agree with Moritz and Ayus who advocate isotonic solutions such as 0.9% saline for routine fluid maintenance in children.[38] Hypotonic solutions, such as 0.18% or even 0.45% saline, are potentially dangerous when renal water excretion is limited by ADH. This raises a significant ethical barrier to conducting a randomised control study as most acutely ill or postoperative patients have increased ADH levels. There are few occasions in medicine where mortality could be reduced by a task as simple as changing from a hypotonic maintenance solution to an isotonic one.

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Maintenance fluid therapy

Rubbing salt in the wound

M Hatherill

The case against isotonic parenteral maintenance solution

In a recent review, Moritz and Ayus have suggested that isotonic parenteral maintenance solution (PMS) should be used to prevent hospital acquired hyponatraemia in children.[1] Hospital acquired hyponatraemia may be exacerbated by non-osmotic production of antidiuretic hormone (ADH) associated with conditions such as bronchiolitis (33%), pneumonia (31% and 45%), bacterial meningitis (50%), and postoperative pain or nausea.[2–7] Although it has been termed a syndrome of inappropriate antidiuretic hormone secretion (SIADH), it may be more accurate to refer to non-osmotic ADH production, since haemodynamic baroreceptor stimuli, such as hypovolaemia, may be physiologically appropriate despite the adverse effect on sodium.[8–11]

The reported morbidity and mortality associated with hospital acquired hyponatraemia have given momentum to calls for increasing the tonicity of PMS.[1 12–14] Implicit in such proposals are the assumptions that hyponatraemia results from a net sodium deficit, exacerbated by hypotonic PMS, and that this sodium deficit may be avoided by using an isotonic solution.[1 12 13] Therefore, if we contemplate a change in practice, we must consider whether

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Theoretical effect of variation in the volume of PMS

Both examples apply to a 10 kg child with non-osmotic ADH production, TBW = 6 l, estimated isotonic (Na + K = 154 mmol/l) urine output of 1 ml/kg/h, estimated IWL of 35 ml/kg/day, and initial sodium = 140 mmol/l.

Example 1
100 ml/kg/day hypotonic solution (0.2% sodium chloride equivalent, Na + K = 34 mmol/l).[1]

Water		**Sodium/potassium**	
Input 100 ml/kg	= 1 l	Input (Na+K)	= 34 mmol
Urine output 25 ml/kg/day	= 0.25 l	Urine output (Na+K)	= 39 mmol
IWL output 35 ml/kg/day	= 0.35 l		
Water balance	= +0.4 l	**(Na+K) balance**	= −5 mmol

New Na = [(Na × TBW) + balance (Na+K)]/[TBW + water balance][9]
New Na = [(140 × 6) −5]/[6 + 0.4]
= 130 mmol/l

Example 2
60 ml/kg/day hypotonic solution (0.2% sodium chloride equivalent, Na + K = 34 mmol/l)

Water		**Sodium/potassium**	
Input 60 ml/kg	= 0.6 l	Input (Na+K)	= 20 mmol
Urine output 25 ml/kg/day	= 0.25 l	Urine output (Na+K)	= 39 mmol
IWL output 35 ml/kg/day	= 0.35 l		
Water balance	= 0 l	**(Na+K) balance**	= −19 mmol
New Na	= [(140 × 6) −19]/[6 + 0]		
	= 137 mmol/l		

hyponatraemia is indeed caused by a deficit of sodium, or by an excess of water, and whether the logical response should be a change to the electrolyte content, or the prescribed volume of PMS.

This article will examine the flaws in the argument for increasing the tonicity of PMS, and explore an alternative hypothesis: that reducing maintenance fluid volume would be equally or more effective as a prophylactic measure against hyponatraemia. Although previous authors have suggested a reduction in the amount of prescribed maintenance fluid, it should be emphasised that the merits of either proposal have yet to be tested in large prospective clinical trials.[4 11 13]

The case against a change to isotonic PMS as a prophylactic measure against hospital acquired hyponatraemia in children hinges on four key issues:

- Traditional volume recommendations for PMS are greater than actual requirements in children at risk of non-osmotic ADH production.[15 16]
- Electrolyte-free insensible water loss (IWL) should be included in the calculation of a tonicity balance in children.[17]
- The principal mechanism leading to hyponatraemia is the primary antidiuresis (dilution), not the secondary natriuresis (desalination).[16 17]
- In the absence of randomised controlled trials, there is insufficient evidence to support the safety, effectiveness, or relative merit of isotonic PMS in children.

PRINCIPLES OF MAINTENANCE FLUID THERAPY

In order to avoid hyponatraemia (or hypernatraemia) a tonicity balance must be preserved, by matching input and output of both water and electrolytes to maintain an isotonic final product.[18] Each input and output may be divided into two components, the volume of water, and the content of effective osmols (sodium and potassium), so that the net effect on tonicity may be calculated from the sum of these separate components.[19]

A nephro-centric approach to maintenance fluid therapy that ignores IWL will contain an inherent error, since *all inputs* and *all outputs* need to be considered.[18 19] Although such an approach might be acceptable in adults, children have greater proportional surface area, and the magnitude of error would increase with the proportion of IWL.[20]

It is important to note that IWL, the "perspiratio insensibilis" of Santorio, represents loss of *electrolyte-free* water.[21] Estimated IWL may be derived from data reported in hospitalised infants and smaller children, ranging from 29 to 54 ml/kg/day for a 10 kg infant.[22 23] After endogenous water of oxidation (270 ml/m²/day) is subtracted, net IWL would amount to 30–35 ml/kg/day.[24] Approximately one third of IWL occurs via the respiratory tract, and two thirds via insensible evaporation from the skin.[24] Since cutaneous IWL is determined by body surface area, net IWL varies with age, and may be as little as 520 ml/day in adults under basal conditions.[10]

In 1956 Holliday and Segar devised a method for calculating maintenance fluid requirements, in which both insensible and urinary water losses were based on energy expenditure.[4] Maintenance electrolyte needs of 3 mmol/kg/day sodium and 2 mmol/kg/day potassium were somewhat arbitrarily based on the amount delivered by human breast milk feeds (1 mmol/kg/day sodium and 2 mmol/kg/day potassium).[13 14]

Caloric expenditure was estimated as 100 kcal/kg/day for an infant weighing up to 10 kg, so that water loss could be calculated per kg body weight.[14] Using this approach, IWL for a 10 kg infant would be calculated as 50 ml/kg/day, with 16 ml/kg/day subtracted for endogenous water of oxidation, equating to net insensible loss of 34 ml/kg/day.[14] Urinary losses, based on the water required to excrete the solute load of cows' milk, would be calculated as 66 ml/kg/day, or 2.75 ml/kg/h.[14]

The sum of the net IWL (34 ml/kg) and renal water loss (66 ml/kg) produced

Hypovolaemia

the arithmetically pleasing calculation of 100 ml/kg/day.[11] Moritz and Ayus assert that this formula for calculating water needs "clearly has passed the test of time". However, even though almost half a century has passed, the formula has not been put to the test.

PROBLEMS WITH TRADITIONAL MAINTENANCE RECOMMENDATIONS

Urine output may be 1 ml/kg/h, or less, if determined by non-osmotic ADH production rather than solute load, and therefore children at risk of hyponatraemia may receive 40–50 ml/kg/day over and above their actual maintenance water needs.[3] It is also notable that hospital acquired hyponatraemia may be associated not only with hypotonic PMS, but with amounts of fluid that exceed, by up to 50%, even currently recommended maintenance volumes.[2 3 10-12 19]

Individual maintenance water needs also depend on motor activity, temperature, and biological work.[13 14] Since the energy expenditure of physically immobile, critically ill children may be less than 40 kcal/kg/day, their maintenance water requirement would be reduced.[14 15] We might expect a further 30% reduction of IWL in patients breathing warmed humidified air through a ventilator circuit, which illustrates an important aspect of fluid balance in critically ill ventilated children.[9] If their fluid requirement is dramatically reduced, by virtue of lower respiratory and cutaneous IWL, and the sodium requirement is unchanged, the concentration of PMS required to deliver that sodium increases.[9 14 15] However, this consideration does not apply to the vast majority of hospitalised children with non-osmotic ADH production, whose reduction in fluid loss is predominantly urinary (high electrolyte content), rather than insensible (zero electrolyte content).[1 2 9 14 15]

REDUCTION IN MAINTENANCE FLUID VOLUME

Previous authors have suggested a reduction of maintenance fluid volume in high risk patients, and fluid allowance of 50 ml/kg/day is standard practice for infants with bronchiolitis in some centres.[2 3 12 13] The rationale for avoiding such "fluid restriction" is that it may be disadvantageous to children with hypovolaemia.[5 6] Three prospective studies address this issue in meningitis.[5 6 16] Powell *et al* showed that plasma vasopressin fell with the administration of additional fluid, suggesting an appropriate ADH response to hypovolaemia.[5] Singhi *et al* showed that although (hypotonic) fluid restriction normalised serum sodium in hyponatraemic patients, it did not lead to a significant outcome advantage or disadvantage, except in post hoc subanalyses.[16] Duke *et al* compared oral fluid restriction and full intravenous maintenance, with no statistically significant difference in serum sodium or adverse outcome.[6]

Clearly, hypovolaemia and inadequate organ perfusion may be disadvantageous to patients with meningitis.[16] However, neither the volume nor composition of maintenance fluid should be a consideration in the treatment of hypovolaemia, which should be corrected immediately with rapid infusion of resuscitation fluid.[40] It has even been suggested that synthetic colloid, rather than saline, should be used to avoid sodium loading during resuscitation.[41]

MECHANISMS OF HYPONATRAEMIA

ADH increases the permeability of the distal renal tubule and collecting duct, resulting in renal conservation of water and inappropriately high urinary sodium concentration, so that children who develop hyponatraemia may excrete urine isotonic to plasma.[6 42 43] Excessive ADH production has also been termed a phenomenon of salt loss or "desalination", based on the secondary increase in net urinary sodium loss, possibly due to suppression of aldosterone, increased natriuretic peptide, or increased glomerular filtration, which occurs after over-expansion of the intravascular space.[6 16 17 42–45]

Experimental models show that the acute hyponatraemia is primarily dilutional, while the secondary natriuresis contributes to the maintenance of ongoing hyponatraemia.[16 17] In a model of 1-desamino-D-arginine vasopressin (DDAVP) infusion, two thirds of the acute hyponatraemia was ascribed to water retention, and one third to sodium depletion.[16] In a similar experiment, rats infused with DDAVP (but not arginine vasopressin) maintained constant sodium balance, and hyponatraemia resulted from water retention alone.[43]

It is important to note that the secondary "desalination" may be prevented by fluid restriction.[17] In normal adults given pitressin, there was no increase in natriuresis if fluid intake were restricted to prevent over-expansion of the intravascular space.[17] It follows that the administration of isotonic saline may be futile unless fluid volume is also reduced, since ongoing natriuresis may negate the effect of this intervention.[17 46 47]

Studies in surgical patients show that while the fall in sodium is related to the volume of electrolyte-free water administered, the sodium falls even if isotonic fluid is administered to produce a net positive sodium balance, evidence of the primary dilutional nature of the hyponatraemia.[46 48 49] Therefore, if the fundamental problem is antidiuresis, rather than natriuresis, surely the principle of treatment should be less fluid, not more salt?

THEORETICAL EFFECTS OF VARIATION IN MAINTENANCE FLUID REGIMEN

It has been suggested that a tonicity balance should be used to predict changes in natraemia, rather than an electrolyte-free water approach.[18] Changes in sodium are related to the ratio between effective osmols (sodium and potassium) and total body water, and the term "isotonic" refers to a solution in which the sum of both sodium and potassium amounts to 154 mmol/l.[1 47 50] Changes in sodium may then be predicted by calculating a tonicity balance from the net gain or loss of effective osmols and water.[18 19] Five per cent dextrose is considered necessary for maintenance of normoglycaemia and cerebral metabolism.[34] However, although the additional dextrose increases the osmolality of PMS, we would not expect it to affect serum sodium, since glucose is not an effective osmol.[1 18]

From the examples in the box, it is apparent that giving 100 ml/kg/day of hypotonic (0.2% saline equivalent) PMS to a child with non-osmotic ADH production might result in a clinically significant fall in sodium from 140 mmol/l to 130 mmol/l. It can be seen from the large positive fluid balance, and small negative sodium balance, that this fall would be primarily dilutional. If tonicity were increased from 0.2% to 0.9% saline equivalent, with volume unchanged at 100 ml/kg/day, hyponatraemia might be prevented at the expense of a large positive fluid balance.

If instead of increasing sodium content, the amount of hypotonic PMS (0.2% saline equivalent) were decreased to 60 ml/kg/day, we might expect a clinically insignificant fall in sodium to 137 mmol/l over a period of 24 hours, but with no increase in total body water. This minor fall in sodium may not even occur if fluid restriction effectively reduces the natriuresis.[17]

LACK OF EVIDENCE FOR ISOTONIC PARENTERAL MAINTENANCE SOLUTIONS

Changes in sodium may be predicted by theoretical manipulation of tonicity balance, but it should be emphasised that current recommendations for water and

electrolyte needs have not been rigorously tested, and are based on estimated values for energy expenditure and IWL derived from small historical studies.[1 2] Although individual needs vary, recommendations for administration of PMS should be appropriate for the majority of all hospitalised children, while simultaneously safeguarding against hypo- or hypernatraemia in high risk conditions.[3]

If we consider increasing the tonicity of PMS to prevent hyponatraemia, several fundamental questions are yet to be answered. Would isotonic PMS be safe?[4 5] Crucially, does it work? Is isotonic PMS actually effective in reducing the incidence of hyponatraemia? Would reducing the maintenance volume of hypotonic PMS be equally effective?[6 7] Given that hyponatraemia may occur despite isotonic fluid administration, and despite a positive sodium balance, isotonic PMS may not be effective in preventing hyponatraemia, unless fluid volume is also reduced.[8 9]

NB!!

A large multicentre randomised trial is needed to compare the current standard of care (hypotonic PMS) with (a) isotonic PMS, (b) isotonic PMS at reduced volume, and (c) hypotonic PMS at reduced volume, in children at risk of hyponatraemia. A recent review considers the ethical aspects of such a trial, in which equipoise must be maintained.[10] Clearly, it would be unethical to perform a study in which the balance of evidence suggests that one treatment arm is inferior to the other. It would be equally unethical to perform a study in which lack of scientific rigour jeopardises the validity of the findings.[12]

The morbidity and mortality associated with hypotonic PMS is not disputed, but also underlines the pitfalls of adopting a standard of care without robust evaluation.[5 11–13] For the reasons outlined above, we may not assume that isotonic PMS would be superior to the current regimen, nor that isotonic PMS is without potential disadvantages.[11 14] A prospective trial to compare the effect of different maintenance fluid regimens on sodium and fluid balance would be both feasible, and ethically acceptable. If serial measurement of sodium and effective data safety monitoring could be ensured. Therefore, until it can be shown that isotonic maintenance fluid is both safe, and effective, in preventing hospital acquired hyponatraemia, calls for widespread change in practice are premature.[15 16]

SUMMARY

The morbidity and mortality associated with hospital acquired hyponatraemia should prompt re-evaluation of measured energy expenditure, water loss, and electrolyte needs in hospitalised children. Traditional recommendations for maintenance fluid volume exceed actual requirements and contribute to the development of hyponatraemia in children at risk of non-osmotic ADH production. Reducing the volume of maintenance fluid may be a more effective prophylactic measure than an increase in sodium content, and a prospective clinical trial should be performed to resolve this issue. Unless the evidence of such a trial were to support the use of isotonic maintenance fluid in children, an injudicious change in clinical practice may not correct the errors of the past 50 years, but compound them.

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NEWS AND NOTES FROM THE UK

When I did resident on-call, every now and then a colleague would discover me in front of children's TV on a Saturday or Sunday morning, usually consuming a hurried on-call breakfast. The excuse "It was on when I came in" wore a bit thin, but fortunately now I have another, much better one should I need it. I'm doing market research. It is the same thing that a previous boss would claim he was doing when he read the local newspaper—a rag of doubtful value and variable accuracy. "This is part of my job" he'd say, and believe. "This is our constituency—a fact we forget at our peril. This is what the people who pay our wages think. Or what they are being told to think."

Back to the weekend morning, and if you haven't tried this recently, you should. Watch television as an anthropologist. Count the adverts and examine their strange internal logic. Look at the link between the adverts and the programme content. Find out what our children are being told to think. I'm told that the best way to advertise to children is to pitch the message just beyond their level of understanding. My guess is that this somehow appeals to both their and their parents' sense of premature ability, or it confuses the child while appealing to the parent, or maybe it just confuses both. I do know that after about 20 minutes of watching I'm at least 10 IQ points the poorer.

The next thing to do is to pick up a teen magazine. As I've said, it is market research, so you have a perfect excuse. Look at the seamless segue between content and advertisement. Look at the lifestyle articles telling our teens what they should be buying and where, how they should look and feel, what they should do and when. Actually, it is on this last issue that I find the single redeeming feature of some of these magazines. The problem pages often offer such sensible, down to earth, useful advice that I'm left wondering whether the agony aunts and uncles inhabit a different planet to the rest of the content providers.

How is this excuse for how I spend the occasional 20 minutes on a Saturday morning at all relevant to being a paediatrician? Well, it must be part of our role as child advocates to see that young people at least have a fighting chance of interpreting this deluge of information in a sensible manner. Our response could be to bring up our children in isolation—in a hut in the Scottish Highlands or Australian Outback. We could deny them access to television, magazines, and no unvetted book written since, say, 1950. Then we could release them into the world at 18 and see how they got on, secure in the knowledge that at the very least they'd had a wholesome childhood.

The other alternative, if we accept that the world that we live in is riddled with the media and, by association, advertising, then we could try to teach them a little bit about what we're beginning to understand about how advertising works. Media literacy sounds like a wishy-washy concept, but it is a powerful idea. Discussing with a 10 year old, for example, "Why are the people in this photograph smiling?" Yes, it might be because they're happy, but it might also be because they're being paid to smile, and that this helps you interpret the essential falseness of the photograph. Extend this to why the people in the photograph are thin, or holding cigarettes, and you can see the power.

It is easy to get carried away with this, but it is also very easy to fall into an advertising trap ourselves. If it weren't, if we were completely media savvy, then why would the otherwise extremely sensible and money conscious pharmaceutical companies take us out to dinner? I don't think I was a particularly stupid child, but when I was 10 and saw an aunt smoking John Player Special cigarettes, I did think that they must have been a great brand if they were named after a formula one racing car. It took me a few years to figure out the many falsehoods in that assumption.

You wouldn't take a child outside on a rainy day without making sure they were wearing a coat, would you? Why, then, would we allow a child out into a world populated with anorexic models, cigarettes, guns, fallible rock stars, soft drinks, and fast food, without comparable defences? The mental environment has become very complex, and our children need some sort of protection in order to be able to survive. Now, you'll excuse me please, as my favourite cartoon is about to start ...

I D Wacogne
Ian Wacogne is a consultant in general paediatrics at Birmingham Children's Hospital

From the Permanent Secretary
Clive Gowdy CB

Department of
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and Public Safety

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Dear Alan,

I have taken some time to consider your reply of 8 April to my letter of 29 March. While I was disappointed with that reply, I do not see any merit in opening up a prolonged exchange of correspondence about the rights and wrongs of the events of 25 March – we will both clearly hold to our respective views on the matter. However, I have come to the conclusion that some of the points made in your letter cannot be left unchallenged and so I am sending you this further letter to put our response to these points on the record.

I am particularly concerned at the suggestions in your letter that Dr Campbell was "evasive" in dealing with the questions put to her, that the "veracity" of what she was saying was subject to dispute and that she was contradicting the Coroner's findings. I am sure that you will appreciate that these are very serious comments to make about a person whose honesty, integrity and professional reputation are paramount in fulfilling the difficult and demanding role she performs.

Needless to say, we do not accept that the CMO was in any way evasive or lacking in veracity in the responses she made to the interviewer's questions. You will recall that part of my concern was that the way in which the interview was conducted meant that Dr Campbell was not afforded the opportunity to give the full and frank answers that she wished to make. I should reiterate for the record that Dr Campbell voluntarily agreed to participate in the programme to fully explain the lessons learned and the steps being taken to prevent such a tragedy happening again. There is no question of her doing anything other than being prepared to deal with the facts and that is what she tried to do but was prevented from doing so by the approach adopted by the interviewer.

Your letter makes much of the suggestion that there was a contradiction between the comments made by Dr Campbell and the findings of the Coroner. There is in fact no such contradiction. Let me put it in plain terms. The coroner was correctly identifying that, in terms of the cause of death, it was the administration of the fluid therapy that led directly to the death of Lucy Crawford. What the CMO was saying was that such a fatal consequence was a rare event, that this cause and effect was not commonly known at the time and that administration of this fluid regime was then not an abnormal event in paediatric departments throughout the UK.

I note that Dr Sumner is recorded as disputing the veracity of the CMO's point that the adverse response was not widely known at that time. We would strongly reiterate the point that there was <u>not</u> a widespread awareness of this reaction and, indeed, we understand that this particular fluid therapy was then in common use in the paediatric department of the hospital in which Dr Sumner himself worked.

In fact, there is still considerable debate among paediatricians regarding the most appropriate intravenous fluid therapy for children. The area of fluid administration in a sick child remains a complex area and within the past few weeks a series of articles published in the highly respected paediatric journal, Archives of Disease in Childhood, highlights the debate on this matter among experts and the many complexities surrounding fluid management in general and hyponatraemia in particular. Regrettably, within such a complex area, problems do on occasion arise as emphasised by the death of a child from hyponatraemia in a major UK hospital as recently as 2003, presenting with a similar clinical condition to that of Lucy Crawford.

Your letter also suggests that the CMO gave an unsatisfactory answer on the reporting of the case. I need to correct you on this point. The Chief Medical Officer became aware of the Lucy Crawford case after being written to by the Coroner. We fully accept that Mr Stanley Miller had alerted the Coroner to the case to draw attention to the similarities with the earlier inquest on Raychel Ferguson, but this does not alter the fact that the Chief Medical Officer was made aware of Lucy's death when the coroner brought it to her attention after considering Stanley Miller's comments and re-examining appropriate documents.

What this pointed up in terms of the reporting of untoward incidents was that there was a lacuna in the arrangements for informing the CMO of such events and that we were hampered by the absence of a formal system to report untoward deaths within hospitals at the time of Lucy Crawford's death. In Northern Ireland there are about 15,000 deaths each year, the majority of which occur in hospital. Approximately 3,500 of all deaths each year are reported to the coroner. Within this context and noting the events involving the deaths of these two young girls, it is clear that it is not any absence of reporting that is at issue, but rather that any new system needs to be capable of identifying those incidents that require further scrutiny and the possible alerting of clinicians of any issues of risk.

I want to take the opportunity to say to you that the conclusions which those making the programme formed on this matter and which you set out in your letter are simply not correct. As I have tried to demonstrate, this is a serious and complex issue and it deserves more than the simplistic treatment which it received in your programme and which identified Dr Campbell and her office as at fault. Unfortunately, the important messages which Dr Campbell tried to convey to the public to explain and reassure them were not allowed to be made by the way in which the interview was conducted.

These messages included the important point that, as part of her responsibility to protect the health of population, following the death of Raychel Ferguson, Dr Campbell convened a working group to

develop guidance on the prevention of hyponatraemia. This guidance was published in 2002 and Northern Ireland was the first part of the UK to issue such guidance. It provides very practical advice for doctors and nurses who manage the care of children in hospital. I should add that it has been commended by local clinicians, by the Belfast coroner, and by Dr Sumner who praised the guidelines when giving evidence at the inquest into Raychel Ferguson's death.

Furthermore, Dr Campbell has recently initiated two further steps to ensure that the guidance remains up to date and is fully and properly applied. Firstly, she has sought assurances from Trust Chief Executives that the guidance has been implemented. Secondly, she has asked an international medical expert in the speciality of paediatrics to quality assure the guidance in light of the findings of the inquest into Lucy's death and any of the more recent emerging evidence on hyponatraemia since the publication of the guidance in 2002. It was unfortunate that the interviewer did not give Dr Campbell the opportunity to put these points across since this would have provided the necessary balance to reassure the public of the important steps that have been taken since the deaths of these young girls.

Finally, I believe that it is important to make the point that the relationship between the Department and the media is one in which clarity, trust and confidence are critical. When assisting with a current affairs programme, we take the view that it is part of our role to provide information and comments that will be helpful in improving viewers' knowledge and understanding of health issues. This is why it is so important that there is mutual understanding of how the programme will be conducted. This lies at the heart of our concern about the pre-programme discussion between Kevin Mulhern and Trevor Birney. It is our view that the contemporaneous notes made of the conversation between Trevor Birney and Kevin Mulhern and from which you have quoted in your letter, are selective to say the least. They do not refer to Trevor's comments that the programme would not be seeking to hold the Chief Medical Officer accountable or laying blame at her door. Hence our concern about the nature of the information we were receiving and our need for assurances about future contacts.

Yours sincerely,

Clive.

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