RAYCHEL FERGUSON (Aftermath of Lucy Crawford's Death)

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Introduction

I have been asked by the Inquiry into Hyponatraemia Related Deaths to provide a consultant paediatrician's viewpoint on certain aspects of the events which were associated with Lucy's death in 2000. In doing so I have been specifically asked to assist the Inquiry in

..examining the steps that were taken by both the Erne Hospital as well as by the RBHSC in their attempts to establish the cause of Lucy's deterioration and death, and to determine whether, given what was known to each of those organisations at that time, those steps could be considered adequate.

BRIEFING

Having regard to the terms of reference of the Inquiry and a list of issues which have been set out I was asked to address a number of points:.

- The role of Dr Quinn, Consultant paediatrician at Altnagelvin hospital who provided an external paediatric opinion to the Erne Trust in 2000
- The part played by Dr Dara O'Donoghue Intensive care Fellow in the intensive care unit at Royal Belfast Hospital for Sick Children (RBHSC) and to comment on his role in the death certification and whether the approach taken was appropriate by reference to any guidance on certification at the time.
- What steps should Dr Hanrahan RBHSC have taken to reach a definite diagnosis of the cause of Lucy's death
- What information should have been given about the purpose of the post-mortem and, in respect of consent, whether this should have been obtained in writing and to comment on the adequacy of the consent process.
- Whether the working pathogenesis as set out in the autopsy request form by Dr Caroline Stewart, formulated by the clinicians at the time (dehydration and hyponatraemia, cerebral oedema, acute coning and brain death) should have been reported to the Coroner's Office.
- Whether it was reasonable for Dr Hanrahan to take the view that a drop in sodium level from 137 to 127 within the time Lucy was in Erne Hospital could not have led to cerebral oedema and to comment on his view that he was unaware of the extent of Lucy's hyponatraemia because he did not know that repeat electrolytes were taken after quantity of normal saline had been run in.
- Whether given what was known at the time it was reasonable to certify the death as 1(a) cerebral oedema (b) dehydration (c) gastroenteritis.

- To comment on the adequacy of the audit procedures which RBHSC had in place measured against expected standards of the time taking account of Dr Taylor's answers.

- In the light of lack of referral of Lucy's death to the RBHSC Critical Incident review group of which Dr Chisakuta was a member to comment on whether Lucy's death was adequately considered by RBHSC for the purposes of clinical governance given the standards of the time.

I have also been asked to consider and report on a number of related governance issues in a set of questions posed to me including consideration of matters at the Erne hospital, Royal Belfast Hospital for Sick Children (RBHSC) and Western Health and Social Services Board (WHSSB)

In order to produce my report I have been provided with a range of background documents to which I will make reference during the course of this report. I have also been provided with a list of Issues which the Inquiry will address in relation to Lucy's case. I am informed that the Inquiry considers that *Arising out of those issues it can be seen that the Inquiry is concerned to investigate the various opportunities that were available at the time of or shortly after Lucy's death which might have facilitated a proper understanding of how her death had been caused. The Inquiry is interested to assess whether those opportunities were properly used, and whether any shortcomings on the part of responsible persons or bodies led to a failure to accurately identify the cause of death.*

I have been informed that the Inquiry will examine certain of the clinical, hospital management and Trust governance issues arising from Lucy's death. The Inquiry is particularly concerned to examine why the contribution played by hyponatraemia in causing her death was not recognised at the time and acted upon. The Inquiry team have informed me that what appears to be clear is that until the Coroner's Verdict was announced in 2004 it remained the publicly stated position that the cause of Lucy's death was as had been described in her death certificate, namely, a cerebral oedema due to or as a consequence of dehydration and gastroenteritis.

Therefore, by June 2001, some 14 months after Lucy's death, when Raychel Ferguson was admitted for treatment in the Altnagelvin Hospital, there had been a failure to identify and disseminate the true cause of Lucy's death. As a consequence of this it might be contended that the medical profession and health care providers in Northern Ireland were deprived of an opportunity to extract and learn appropriate lessons from Lucy's case before Raychel died.

In due course, the Inquiry may wish to reach conclusions on what impact these failures may have had for the diagnosis and management of Raychel's condition in the Altnagelvin Hospital.

My background

I am a retired consultant paediatrician from Pinderfields Hospital Wakefield and was director of children's services there. In addition I was an honorary senior lecturer in paediatrics at the University of Leeds. I retired in 2006 but until 2008 acted as the national clinical lead for child health in the NHS IT programme.

I was in clinical paediatric practice in general paediatrics for 36 years, 28 years of which as a consultant. At one time I was Clinical Director for all non-surgical clinical specialties and held other posts in hospital management listed in my cv. During my time as a consultant paediatrician I continued and developed my interest in emergency paediatrics and led projects researching into the common paediatric presentations in children (including gastroenteritis) . I was Honorary Secretary of the British Paediatric Association for 5 years from 1989 and the Vice President of its successor body Royal College of Paediatrics and Child Health for 4 years. In this role I was involved in development of standards of paediatric practice both in general and specialist paediatric practice and also provided reviews of paediatric and child health services in a number of parts of the United Kingdom at the request of health authorities. Over seven years from 1996 I was seconded part time to the Department of Health England as the DH Paediatric Adviser. In this last position I selected and commissioned the first NICE guidelines on children and through the Department of Health established national audits in paediatric intensive care, neonatal blood spot screening and newborn intensive care. Additionally I negotiated the development of investigation of childhood deaths at all ages (rather than confined to infancy as was already in place). Since retirement I have continued my practice as an expert legal adviser in paediatrics.

SUMMARY CONCLUSIONS

ERNE HOSPITAL

i. Lucy's death in April 2000 was recognised as a serious adverse event by clinical staff and management in Erne hospital. An internal review was set up assisted by a case note review by an external consultant paediatrician, Dr Quinn, with a focus on fluid management and consideration of how the cerebral oedema which led to her death had occurred.

ii. Her death was reported to WHSSB as part of clinical governance procedures. WHSSB staff, to some extent, were consulted for advice and a "steer" on the process of investigation. Lucy's death was not reported to DHSSPS. WHSSB expected that the Trust would do so. The Trust general manager expected WHSSB to do so or to advise the Trust to do so.

iii. The Erne review process was flawed and incomplete.

- Little attempt was made to reconcile reports received and to clarify inconsistency and lack of information. No opinions were sought from Trust consultants who had treated her on how the cerebral oedema might have occurred. One consultant treating Lucy had formed serious concerns about the fluid regime used at the time and considered Lucy's death to be avoidable. His views were not sought (or offered).The report was not shared with contributors denying them an opportunity for addition or correction.

- Dr Quinn reported he had no explanation for the cerebral oedema but his assessment of the fluid regime was incorrect and the report unduly reassuring.

- One of the stated aims of the review was to inform parents yet they were not involved and not informed of the review findings until 2001 after they had initiated a legal claim in late 2000.

- No attempt was made to communicate with the treating clinicians at RBHSC to obtain their views during the review nor to inform them of its findings. Lucy's death was regarded as unexplained after the Erne Trust internal review report in July 2000 and remained so.

- The review identified deficiencies in documentation but did not recognise that Lucy received a fluid regime which contributed to or caused her death.

iv. The Trust medical director did not communicate the review findings to the Coroner although he expected that an inquest was in hand. The review report was received by Erne Trust medical director and chief executive but they did not recognise its shortcomings. No wider external review was set up into Lucy's cause of death.

v. WHSSB also received a the review report but did not appreciate its shortcomings although they had a role in quality management within Erne Trust in relation to adverse

event investigation to ensure that processes were up to standard under the arrangements of clinical governance as commissioners (Set out for this Inquiry by Mr Frawley GM WHSSB).

vi. WHSSB staff now state that following receipt of the review in July 2000 they had advised the Trust to obtain a wider clinical review. This was not done and there is no documentation this advice was received by the Erne Trust nor did WHSSB check that this advice had been followed.

vii. In my opinion if the review had been conducted properly and further extended by requesting further expert review it should have become evident at latest by December 2000 that the fluid regime had contributed to Lucy's death. It is probable that a conclusion could have been drawn similar to that of Dr Boon and Stewart in 2002 that "*More careful attention to detail of the fluid therapy might possibly have avoided this girl' s cerebral oedema and fatal outcome".* It is arguable that a review could have drawn greater attention to the risk of use of No 18 solution as a replacement fluid and provided a caution for dissemination to colleagues but the specific risk for hyponatraemia and cerebral oedema was better known in 2002 compared with 2000. In contrast both N18 solution and normal saline given to Lucy were wrongly used in Lucy. Any medication used incorrectly is associated with risks of adverse outcome and her death may have been regarded to be a result of that misuse with implications to report as a drug error and to make either a referral or report to the Coroner for the Inquest and without considering a need for wider dissemination.

viii. Reviews were conducted by RCPCH on the professional competence of Dr O'Donohoe reported in June 2001 and spring 2002. In each of these, comment was provided to the Trust that the fluid regime may have contributed to Lucy's death. In 2001 the Trust expected an inquest to be planned and they should have informed the Coroner's office of this concern. In 2002 the Trust was aware that no inquest was planned and they should have made a formal referral to the Coroner at latest and, arguably they should have done so when they learnt in October 2001 that no inquest was planned.

ix. WHSSB did not assure themselves that Coroner referrals had taken place as part of the process of investigation of an adverse incident.

RBHSC :CLINICAL

x. Lucy was admitted to PICU with signs of brain death which were confirmed, investigations were carried out for causation of cerebral oedema and supportive therapy was withdrawn. Her death was reported to the Coroner's office by Dr Hanrahan who was, in my view, responsible for her care although shared with intensivist consultants during her life. Dr Hanrahan was directed to discuss the case with the state pathologist with whom it was agreed a formal Coroner's notification was not needed but that a hospital post-mortem be conducted to clarify the cause of death.

xi. The hospital autopsy preliminary report was received by Dr Hanrahan. The report did not identify the cause of cerebral oedema satisfactorily and did not mention hyponatraemia although features consistent with fluid overload were reported. He should have referred again to the Coroner and not issued a Death Certificate at this point.

xii. Dr Hanrahan was responsible for the content of the issued certificate which was completed illogically in linking dehydration with cerebral oedema. At the time he was unaware of the fluid regime used in Erne and did not review the fluid documentation in the notes. Consequently he overlooked that Lucy had the blood sample showing hyponatraemia shortly after a large volume of normal saline had been infused which may have concealed the scale of the hyponatraemia of which he was aware.

xiii. Thus after Lucy's death Dr Hanrahan's role shows a number of shortcomings:

- He did not review the case records and fluid regime and did not appreciate the volume overload with hypotonic fluid.
- He was aware of the low blood sodium but concluded this as insufficient in severity to cause the cerebral oedema. This point is reasonable given the knowledge of the time as a ***cause*** of cerebral oedema although it should be well known to a paediatric neurologist that small derangements in blood sodium occurring in the context of an existing encephalopathy can be of great clinical significance. However he did not consider that this level of hyponatraemia could also be a sign of the fluid overload nor take account of the weight gain: another sign. Nor, that severity of hyponatraemia at the time of Lucy's collapse could have been greater than was measured after the high volume of normal saline had been given.
- Dr Hanrahan saw the parents before the final autopsy report was produced in June 2000. If he had any concerns about the fluid regime used at that time, he made no note of this but advised parents to see Dr O'Donohoe in Erne. Documentation of this discussion is insufficient.
- Dr Hanrahan did not send a discharge letter to the GP nor to the Erne hospital and he should have done so summarising his views and actions.
- He did not review the final autopsy report and, he did not prepare for or attend the audit mortality meeting in August 2000 when Lucy was discussed.

RBHSC : GOVERNANCE

xiv. Lucy's death was not reported as a critical incident within the RBHSC. In my opinion this was not unreasonable by the standards of the day. Clinical care in RBHSC had not caused any concern and no clinical adverse event took place there.

xv. The audit mortality meeting in August 2000 offered an opportunity for a review of the records, including those from Erne, and to recognise during the preparation of the case presentation the excessive volume of fluids used before admission to PICU, to identify that the level of hyponatraemia recorded at Erne could have been more severe because the blood sample was taken after a large volume of normal saline had been given at the

time of the collapse, and identified that the autopsy report did not properly identify the cause of the cerebral oedema nor of the death. The death certificate issue and content could have been reviewed and challenged because the logical sequence of pathogenesis was flawed.

xvi. The result of the audit could have been either to initiate a wider and more detailed assessment of the fluid balance, or to report concerns to Erne and to refer again to the Coroner. Had any of these steps been taken the contributing factors to Lucy's death might well have been identified before the end of the year 2000.

xvii. The audit processes in place in Belfast appear to have a number of shortcomings and were not in keeping with guidance or even RBHSC practice at the time. The meetings were not adequately minuted, identifying significant outcomes of discussion, there does not appear to be a process of aggregating and analysing trends and the IT support for the process was limited. It was not attended by the intensivists or consultant paediatric neurologist involved in her care nor, the pathologist. There is no evidence of actions planned or taken after any audits were conducted nor, of annual or other reporting to Trust management to the clinical director on audit findings and planned actions within the "audit cycle".

xviii. Audit annex F provides a review of standards and practice in clinical audit in the NHS at the time. And I provide detail commentary on RBHSC audit in Para 680-735.

AUTOPSY

xix. Although hyponatraemia was recognised by the clinical team as a significant clinical feature when requesting the autopsy, this was not mentioned by the pathologist Dr O'Hara in his report. He offered no satisfactory explanation for the cerebral oedema but did raise questions with parents about clinical care at Erne hospital. Arguably he should have referred to the Coroner at this point.

REPORT STRUCTURE

SYNOPSIS Para 1	1-40

- Erne clinical illness and review
- RBHSC Coroner referral; hospital autopsy; Death certificate; Child mortality/audit meeting held RBHSC August 2000.

ERNE HOSPITAL	**Para No**
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ANNEX DOCUMENT –separate document

- **Annex A : DETAILED COMMENTARY ON CLINICAL REPORTS**
    - **Dr Quinn's report June 2000**
    - **Dr Stewart's 2001 report and report from Dr John Jenkins in 2002**
- **Annex B Gastroenteritis : Epidemiology and mortality**
- **Annex C Guidance On IV Fluid Treatment In Gastroenteritis /Dehydration and analysis of change following withdrawl of 0.18% from children's wards in a DGH in 2002.**
- **Annex D: Relevant guidance on completion of death certificates and referral to Coroner and consent for hospital autopsy.**
- **Annex E: Resources for enquiry into childhood deaths in N.I. in 2000 (including information from N.I. CESDI)**
- **Annex F AUDIT ANNEX**

ATTACHED FOR SOURCE OF REFERENCE : ANNEX D FROM REPORT ON CLAIRE ROBERTS FOR THE INQUIRY INCLUDING LATER DEVELOPMENT OF SYSTEMS FOR INVESTIGATION OF ADVERSE EVENTS IN CHILDREN

SYNOPSIS

1) SUMMARY OF THE CLINICAL ILLNESS AND TREATMENT AT ERNE.

2) Lucy was admitted age 17 months to the Erne hospital with rotavirus gastroenteritis on 12 April 2000. It is evident from her records that she was moderately severely dehydrated. She was given an excessive volume of number 18 solution intravenously. The blood sodium level which had been normal on admission fell over a period of 4 ½ hours during therapy. A water overload from the excessive volume of No 18 solution used and the associated fall in the blood sodium probably contributed to or caused cerebral oedema. When Lucy suffered a seizure and respiratory arrest 4 ½ hours after the No 18 solution started she was given a further excessive volume as 250 -500 mL of normal saline over one hour which was not indicated for her clinical condition and may have caused deterioration in her state.

3) Lucy was found to have signs of brain death and transferred to the Royal Belfast Hospital for Sick Children (RBHSC) in the early hours of 13 April 2000 where, after a number of tests confirming the cerebral oedema, she was declared dead on 14 April 2000.

4) Number 18 solution was in widespread use throughout the United Kingdom at the time for maintenance intravenous therapy. In some units it was used for maintenance/ replacement therapy in mildly dehydrated children with initial normal blood sodium levels on the basis that it was expected that the infusion would only be used for a few hours while trials of oral rehydration, the preferred therapy, were given. No 18 solution was not indicated for *resuscitation* volumes when 0.9% normal saline was indicated nor for more severe degrees of dehydration especially with increased losses e.g. from continuing diarrhoea when maintenance/replacement fluid to be used was 0.45% or normal saline.

5) In the course of the Trust review it became evident that Lucy had received between 400 and 500 mL of number 18 solution given at 100 ml /hour over 4-4 ½ hours after the intravenous infusion was set up. By the standards of practice at the time such a large volume should only have been given if it included a *resuscitation* volume of 91-182 ml of normal saline (i.e. 22% and 45% of the total) given at the start of the IV therapy over less than 1 hour and followed by 0.45% saline at rate of around 70-80ml per hour.

6) THE ERNE REVIEW APRIL TO JULY 2000

7) After Lucy's death following concerns about the fluid regime used raised both by Dr O'Donohoe, consultant in charge of Lucy's care, and the nursing staff, the Trust recognised the unusual incident of the death from gastroenteritis occurring in a 17 month old child. It responded appropriately by setting up a review. The review process included seeking an opinion from an external paediatrician.

8) In 2000 there were no existing guidelines on how to proceed with such a review. Principles of common sense and the use of skills available e.g. within clinical practice on how to assemble facts or accounts, analyse them and reconcile them into a conclusion were available within the Trust.

9) These principles were not applied. Factual accounts of events were requested verbally from the medical staff, and in writing from the nursing staff with opinions on Lucy's care not being sought from or offered by the doctors. No attempts were made, while memories were fresh, to analyse or reconcile the accounts, properly to assemble a chronology or to summarise the fluid administered and to clarify any uncertainties.

10) The involved consultant anaesthetist Dr Auterson formed a view at the time that the fluid regime used and the hyponatraemia had contributed to a potentially avoidable death. His opinion was not sought in writing or in interview or offered by him. He was not shown a copy of the review report. [1]No information was sought from the treating clinicians at Belfast on their opinions on the cause of death. There is no documentation that Trust staff knew of the information included in the death certificate issued in Belfast. The Trust received no discharge letter from Belfast and this should have been sought by the review.

11) The review concluded that documentation and prescription of fluids had been substandard. Information provided by the external paediatrician, Dr Quinn was misleading and essentially wrong. He advised that the volumes of intravenous fluid used up to 3 AM were not grossly excessive and advised that the use of number 18 solution in these volumes was appropriate. This opinion is outside general guidance on intravenous fluid therapy for moderate or severe dehydration at the time. Dr Quinn drew attention to his uncertainty about the volume of normal saline used in the later respiratory resuscitation and advised this was excessive. He expressed "surprise" that the volumes used could have led to the cerebral oedema. He did not draw attention to how hyponatraemia and the rapid fall in blood sodium could have contributed to the cerebral oedema nor that it could have been caused by high volumes of number 18 solution. In his November 2012 witness statement he confirms that he had knowledge of these relationships at the time. Dr Quinn advised the Trust that he had no explanation for the cerebral oedema which caused Lucy's death.

[1] WS-274/1 Q 8, Q16

12) The review report recorded the finding that there was no explanation for Lucy's death. It concluded that none of the defects in documentation or prescription had contributed. Recommendations made included improvement in documentation and issue of guidelines for use on the children's Ward. Some additional training was provided for the nursing staff but not for the doctors. Guidelines on fluid prescription were provided for the Ward. There is no evidence of any audit process set up to monitor implementation or improvement in these points. Although the review recommended that parents should be interviewed to share the findings of the review, this was not initiated by the Trust.

13) The report of the review should have been shared with Trust staff who had contributed to it seeking their views on its conclusions and its recommendations. It was not.

14) **WHSSB and Erne Trust** : In my opinion the Trust should have set up a further external review of Lucy's death and obtained advice on how to do so. Dr Quinn states that he advised them to do so. Dr McConnell from the Western Health and Social Services Board (with responsibilities for commissioning) was consulted and kept appraised of the review process and its findings. He reports in November 2012 that he had advised the Trust, after receipt of the preliminary internal review in July 2000, to widen its enquiry into Lucy's death. This advice is not mentioned by Dr Kelly or Mr Mills and there is no contemporary documentation of this advice. At the time Dr McConnell concluded that the review was of satisfactory standard. I have limited understanding of Dr McConnell's role and advise that an opinion is obtained from a specialist in public health medicine. Dr McConnell sets out his responsibilities and role and reports that as commissioners WHSSB would wish to be aware of the death of Lucy and to work with WHSSB Board management to which he reported and with professional colleagues to ensure that the Erne Trust were taking all appropriate steps to investigate the surrounding events.[2] Dr McConnell expected to receive updated information on the progress of the reviews. Dr McConnell indicates that Trusts were independent entities and were responsible managerially to DHSS(PS) and responsible to WHSSB primarily regarding commissioning services. Dr McConnell held discussions with Dr Kelly about the process of the reviews. Dr Kelly in turn had reported to him as a matter of clinical governance. [3] On receipt of the reviews, Dr Kelly sent copies to Dr McConnell who reviewed them and held discussions with him[4] Dr McConnell's role appears to have elements of clinical governance.

15) Although WHSSB expected the Trust to report the death to DHSSPS Mr Mills, Chief Executive at Erne considered this was the responsibility of WHSSB or at least expected WHSSB to ask the Trust to do so. A wider review was not requested by Erne Trust and, given the explanation of the role of WHSSB by Mr Frawley General Manager in ensuring

[2] WS-286 Q 6-8

[3] (036a-046-098).

[4] WS-290/1,P7 & 036a-29-070.

that appropriate processes were in place in the Trust for investigation , it is of note that it must become evident to WHSSB in June 2001 that a wider review of Lucy's death had not been requested by the Trust.

16) Even if the Trust assumed that a Coroner's inquest was in hand, the delays in this process were known to be such that for safety and quality purposes any lessons that could be learned should have been investigated by an external review in 2000 in order to be assured that any safety and quality issues found were addressed for care of children being admitted to the hospital while awaiting Inquest findings.

17) After completion of the Internal review (informed by an external consultant paediatrician's opinion) in July 2000, the Trust took no further action or arranged a formal investigation of Lucy's death. In my opinion, the Trust should have reported the results of the Trust review at that time to the Coroner in order to assist his enquiries as they assumed that an inquest was being planned. Had they done so the Trust would have discovered that no inquest was planned.

18) **Review by RCPCH 2001**. The Trust requested a review of Dr O'Donohoe's practice from the Royal College of Paediatrics and Child Health. Dr Moira Stewart for RCPCH conducted a review which was not an investigation of Lucy Crawford's death although her case was included amongst others in the RCPCH review. Dr Stewarts's opinion of April and June 2001 identified that the fluid regime used in Lucy was inappropriate and that cerebral oedema could have resulted from rapid electrolyte changes found in her.

19) The RCPCH review was shared with Dr McConnell in June 2001 when it was evident that his recommendations that a further external review of Lucy's death had not been followed in 2000. Three weeks after its findings were available to the Trust Medical Director, Dr Kelly and to Dr McConnell, both became aware of a death in a child from use of number 18 solution and associated hyponatraemia. It is not evident that either made a linkage of this event with Lucy Crawford although Dr Kelly issued a letter to the paediatric department advising them to review their fluid regimes. In my opinion the Coroner in Belfast should have been informed of Dr Moira Stewart's opinion to assist in his enquiries as the Trust still assumed that an inquest was planned. The Trust only became aware in October 2001 that no inquest was planned.

20) **Reports in 2002.** In 2002, the Trust received two clinical reports, one from Dr John Jenkins acting for the Trust's response to litigation initiated by Lucy's parents, the other a second RCPCH review requested by the Trust on Dr O'Donohoe's practice. Both reports advised that the fluid regime in Lucy had been inappropriate and that it was likely to have contributed to her death. The Trust did not refer to the Coroner then and in my opinion had a responsibility to do so.

21) Lucy's death was referred to the Coroner in February 2003 by Mr Millar who had acted as an advocate to the parents from the summer of 2000 and made a link between Raychel Ferguson's and Lucy's death. This led to the inquest in 2004 when cause of

death was listed : I (a) cerebral oedema; (b) acute dilutional Hyponatraemia; (c) excess dilute fluid . II gastroenteritis.

22) <u>ROYAL BELFAST HOSPITAL FOR SICK CHILDREN (RBHSC)</u>

23) Lucy was transferred on 13th of April 2000 under the joint care of Dr Crean, consultant in the intensive care unit and Dr Hanrahan, consultant in paediatric neurology in Belfast.

24) After investigation, Dr Hanrahan found no clear explanation for the cerebral oedema and brain death present on admission at Belfast. It was known to Dr Crean at the time that acutely developing hyponatraemia could cause neurological deterioration and was aware of the problems associated with the infusion of hypotonic fluids in children and the potential for dilutional hyponatraemia to develop.[5] It is not evident that he shared this knowledge with Dr Hanrahan, nor that a discussion was held about how hyponatraemia and cerebral oedema could have developed in Lucy during her treatment in Erne hospital. Both were aware that she had been treated there for gastroenteritis. Dr Hanrahan considered that the level of blood sodium found was not low enough to cause cerebral oedema. Dr Crean has stated " *with the knowledge available to me at the time of Lucy's admission to PICU, I do not think I would have considered a sodium level of 127 to be low enough for me to have formed the opinion that dilutional hyponatraemia had caused Lucy's acute collapse*. " [6]It appears that neither Dr Crean nor Dr Hanrahan took account at the time of the excessive volume of number 18 solution and normal saline which had been used in Lucy at Erne hospital. Knowledge about hyponatraemia resulting from use of hypotonic intravenous fluid was included in the standard paediatric textbook current at the time (5th edition Forfar and Arneil) and other sources. According to Dr O'Donohoe , Dr Crean had questioned the volumes given to Lucy at Erne in a telephone call on 13 April and thus appears to have reviewed the fluid regime used. [7] The Erne notes faxed to RBHSC record that 500ml normal saline was started at 03:00 to be run freely and was given over 60 minutes [8]. The record does not show when the second blood sample showing the low sodium was taken (this was known to the Erne Review and Dr O'Donohoe) so that its relationship to the infusion of saline was not evident to RBHSC.

25) **Coroner referral :** In view of his uncertainty about the cause of Lucy's death, on 14 April 2000, Dr Hanrahan reported it to the Coroner's office and, after discussion with Dr Curtis, of the state pathologist department to whom Dr Hanrahan had been directed by the Coroner's Office, a decision was made that a Coroner's referral was not indicated but that it would help understanding to carry out a hospital autopsy. The content of the

[5] WS-292/1 Q8(b) & WS-292/2 Q5

[6] WS-292/2 Q5

[7] WS-278/1

[8] Dr Malik's note 061-017-048

discussion was not documented and neither Dr Hanrahan nor Dr Curtis can recall its content. Dr Curtis states that if he had been informed of the hyponatraemia he would have advised referral to the Coroner. He also suggests when referring to Dr Hanrahan's depositions to the Inquiry, that Dr Hanrahan was not aware of the significance of the rapid fall in the blood sodium and thus that the possibility of the fluid management problem was not mentioned. Dr Curtis states that he would have referred to the Coroner "*had the hyponatraemia been mentioned alongside dehydration.* " [9]

26) MR LECKEY SENIOR CORONER FOR NORTHERN IRELAND

27) Mr Leckey explains that an informal arrangement had evolved seeking from time to time advice and guidance from the state pathologist's Department if it was unclear to either the Coroner or the staff it was appropriate for a death certificate to be issued by a reporting doctor or if there should be a post-mortem examination. The Coroner's office would normally be advised of the outcome of the discussion held between state pathologist and reporting medical practitioner. The medical practitioner was not bound to accept the opinion of the pathologist. Mr Leckey emphasises that the Coroner's role is reactive rather than proactive and only deals with deaths which are reported and comments on lack of " quality assurance " of items entered on death certificates.[10]

28) **AUTOPSY AND DEATH CERTIFICATION**

29) **Hospital autopsy:** The autopsy request form completed by Dr Caroline Stewart on behalf of Dr Hanrahan listed *Vomiting and diarrhoea; Dehydration; Hyponatraemia; Seizure and unresponsiveness leading to brain stem death* The preliminary autopsy report on 14th of April 2000 confirmed cerebral oedema and thus provided Dr Hanrahan with no further information than had been listed on the autopsy request form but, notably, the autopsy report omitted hyponatraemia- a possible cause of cerebral oedema. Features consistent with fluid overload (pulmonary oedema and weight gain) were found but this was not specifically highlighted by the pathologist Dr O'Hara.

30) **Death certificate:** On the 4th of May 2000 a death certificate was issued by Dr D O'Donoghue SHO at RBHSC on behalf of, and following instructions from, Dr Hanrahan who listed cause of death as cerebral oedema, dehydration, gastroenteritis.

31) In the light of the uncertainty on cause of death, not resolved by the preliminary autopsy report, in my opinion Dr Hanrahan should have referred again to the Coroner and thus not issued a certificate. The items listed in the death certificate were not logical unless Dr Hanrahan considered that it was <u>treatment</u> of the dehydration which had caused the cerebral oedema. If so then he should have referred to the Coroner and thus not issued a certificate.

[9] WS 275/2 Q3 P3

[10] WS-277/2

32) Dr Hanrahan saw the parents to discuss his views on 9 June 2000, before he had the final autopsy report. The content of the discussion was not recorded. This should have been in keeping with good practice guidance at the time. [General Medical Council *Good Medical Practice 1998*- applicable at the time revised 2001- Para 3.] He advised parents to see Dr O'Donohoe at the Erne hospital to seek further information. It is not evident from the records whether he had concerns about the treatment at Erne hospital but his statements in 2012 suggest that he had concerns about the fluid regime used although it is not evident when he developed these.

33) The final autopsy report was available on 13th of June 2000. Dr O'Hara, the pathologist ,met the parents together with Mr Millar on the 16th June 2000. Notes made by Mr Millar suggest that Dr O'Hara raised concerns about aspects of the care at Erne. If he had formed these views, it is arguable that he should have referred Lucy to the Coroner but I advise that a pathologist's opinion is obtained on Dr O'Hara's responsibilities in this situation.

34) In Dr Hanrahan's latest statement, he explains he considered the hyponatraemia was not severe enough to result in dilutional hyponatraemia and coning and thus did not identify it as severe enough to be causative of Lucy's demise. However he reports he was not aware of the large bolus of normal saline which been given. [11]

35) Dr Hanrahan confirms that he did not clarify events at the Erne hospital when meeting parents on 9th of June 2000 but suggested to parents that they do so and that they put their concerns in writing (061-005-012). He states *"notwithstanding the express concern about the IV fluid administered to Lucy, the exact cause of death was not obvious at that stage in view of the seemingly modest drop in sodium measurement".* Comment: if at this stage Dr Hanrahan remained uncertain of the cause of death despite the post-mortem having been conducted, it is arguable that he should have considered reporting again to the Coroner or at the very least reviewing the post-mortem final report which was provided on 13th of June 2000. Yet he confirms that he did not review the final autopsy report although he had requested the autopsy specifically to resolve his uncertainty about the cause of Lucy' death. His omission in not seeking this information is striking and compounded by his absence at the audit mortality meeting held in August 2000. If a review of the case notes been conducted for this meeting, it may have become clear from the Erne hospital records which were held by the RBHSC and included the fluid balance charts (but not the Erne ICU/HDU chart), that high volumes had been given and more attention is likely to have been given to its role in Lucy's death in view of the lack of full explanation. A discussion with Erne hospital raising questions about the timing of the blood sample and the normal saline solution could have revealed the potential for presence of a lower blood sodium than 127 mm/l at the of the arrest.[12]

[11] WS-289/2 Q7 & Q 12 & Q 2 & Q 11 (d), & Q 9

[12] WS-289/2 Q 11

36) Dr Hanrahan indicates that he was unsure of who was in charge of Lucy's care when she was a patient in PICU and this is confirmed in responding in respect of the lack of provision of a discharge letter because it was "*not clear to me that I was the responsible consultant and I may have believed that I was only involved in a consultative role"* and does not *"recall formally assuming responsibility "*[13] In my opinion as set out in Para 569-580, Dr Hanrahan was responsible for diagnostic care in life and after death was responsible for the processes. The intensive care consultants Dr Crean and Dr Chisakuta were jointly responsible with Dr Hanrahan during Lucy's life.

37) Child mortality/audit meeting held RBHSC August 2000.

38) This meeting offered an opportunity for a review of the records, including those from Erne, and to recognise during the preparation of the case presentation the excessive volume of fluids used in Erne before admission to PICU, to question whether the level of hyponatraemia recorded at Erne could have been more severe because (in fact but not in the Erne record) the blood sample was taken after a large volume of normal saline had been given at the time of the collapse, and identified that the autopsy report did not properly identify the cause of the cerebral oedema nor of the death. The death certificate issue and content could have been reviewed and challenged because the logical sequence of pathogenesis was flawed.

39) The result of the audit could have been either to initiate a wider and more detailed assessment of the fluid balance, to report concerns to Erne and to refer again to the Coroner. Had any of these steps been taken the contributing factors to Lucy's death might well have been identified before the end of the year 2000.

40) The audit processes in place in Belfast appear to have a number of shortcomings and were not in keeping with guidance current at the time. The meetings were not adequately minuted, identifying significant outcomes of discussion, there does not appear to be a process of aggregating and analysing trends and the IT support for the process was limited. Audit annex F provides a review of standards and practice in clinical audit in the NHS at the time. And I provide detail commentary on RBHSC audit in Para 680-735.

[13] WS-289/2 Q 2

41) LUCY CRAWFORD ERNE HOSPITAL

42) LUCY'S ILLNESS AND CAUSE OF DEATH

43) Lucy died from cerebral oedema associated with rotavirus gastroenteritis at the age of 17 months on 14 April 2000 after admission to Erne hospital under the care of Dr Jarlath O'Donohoe, consultant paediatrician, on 12 April 2000 and transfer to RBHSC on 13 April.

44) Lucy had previously been a healthy child. She developed an acute gastroenteritis in the course of which she had vomiting and refusal to take fluid producing dehydration and later developed diarrhoea some hours after admission. She was treated with intravenous fluid over 4-4 ½ hours and on 13 April developed a seizure 7 ½ hours after admission and 20 minutes later had a respiratory arrest and was resuscitated but then was found to have evidence of brain death. She was ventilated and transferred to the Royal Belfast Hospital for Sick Children (RBHSC) where, after a period of further investigation and treatment, ventilatory support was withdrawn and she died on 14 April 2000.

45) Immediately after Lucy's death the Erne Hospital Trust set up an investigation by an internal review in the course of which an opinion on her care was sought from Dr Quinn, a consultant paediatrician in a neighbouring hospital.

46) INFORMATION ON CAUSE OF DEATH

47) **Autopsy:** A **Provisional** Anatomical Summary from the autopsy was provided on 17 April 2000 [061-009-033] which included the following :

(9). Relatively little congestion with some distension of large and small intestine with gas and free fluid, patchy pulmonary congestion, pulmonary oedema

(10). Swollen brain, with generalised oedema, brain to be further described following fixation

48) **Autopsy: The final Anatomical Summary** from the autopsy was provided on 13 June 2000 included :

(4).Extensive bilateral bronchopneumonia

(6). Swollen brain with generalised oedema and early necrosis

49) <u>THE CLINICAL ILLNESS IN LUCY AND TREATMENT IN ERNE INCLUDING THE FLUID REGIME USED</u>

50) **<u>Note on Gastroenteritis</u>** This is an infection caused by a number of organisms. In United Kingdom approximately half of all cases are caused by rotavirus. It can lead to dehydration in the following ways:

- Reluctance to drink with reduced fluid intake.
- Vomiting
- Loss of fluid into bowel lumen before being passed as diarrhoea
- Diarrhoea.

51) Dehydration can lead to loss of blood electrolytes including sodium. It can also lead to reduction of the circulating blood volume with poor perfusion of the body's organs including the skin with pallor, reduced capillary return on examination, dry mouth, lethargy and increased blood urea or creatinine. With advancing dehydration the circulation empties with a threat to loss of blood pressure (imminent or evolving shock) and the actual loss of blood pressure (established shock). Both of the latter conditions are heralded by an increase in pulse rate and respiratory rate, and decreased capillary return before the loss of blood pressure. In practice clinicians grade dehydration as mild (2.5% of body weight), moderate (5% to 7.5%), or severe (9%-10%) in the latter there is a risk or presence of shock (circulatory collapse).

52) With mild to moderate illness, treatment is attempted with oral fluids but if these are not taken or are vomited then intravenous fluid is required: (a) to *replace* the fluid already lost and (b) to provide the daily *maintenance* fluid required and, (c) In more severe illness *circulatory resuscitation* may be required for circulatory collapse to restore the blood pressure or prevent loss of it -that is treatment for imminent or established shock. Thus when used, intravenous fluid is given in a few children for resuscitation but in most children only for replacement/maintenance.

53) **<u>Note on the term resuscitation.</u>** In the discussions which follow it may be helpful to consider resuscitation in two ways : circulatory and / or respiratory

- *Circulatory* resuscitation. The circulation can be resuscitated by intravenous infusion of normal saline, plasma or blood or artificial colloid to fill the circulation (either as fast bolus or high rate infusion) and, if necessary, administration of drugs to support the cardiac pump action.
- *Respiratory* resuscitation needed for respiratory insufficiency or arrest is by administration of oxygen and artificial forms of ventilation such as the use of a bag and mask or, after intubation, artificial ventilation using a ventilator.

54) Note on "shock"*:* in this report and those of other clinicians various terms used include shock, circulatory collapse or circulatory failure. In relation to Lucy it may be useful to consider shock in the following ways**:**

a. **Established shock/circulatory collapse:** this is the advanced stage when the circulation is unable properly to perfuse the brain, lungs, kidneys and the heart and other tissues. Consequently these other organs begin to fail. It is manifest in a child by an inability to maintain posture, impaired consciousness to a mild or severe degree, increased pulse rate-often substantially so although in the later stages with inadequate perfusion to the heart, the pulse rate may become low. The respiratory rate is increased and there may be an acidosis arising from poor perfusion of the tissues. The blood pressure is often low and the pulse volume is low and "thready". There is very prolonged capillary return and metabolic features on blood tests showed an increase in urea and acidosis. Advanced Paediatric Life Support Manual (APLS)Phase 2 ("Uncompensated ") shock.

b. **Imminent or evolving shock:** a clinical condition which leads to poor filling of the circulation which is manifest as an increased pulse rate and respiratory rate, the blood pressure until late is often maintained, there may be biochemical abnormalities as above. The capillary return is prolonged. This is sometimes also described as circulatory failure (in contrast to circulatory collapse). APLS Phase 1 ("Compensated ") shock

c. **Potential shock:** clinical condition in which shock is likely to occur without therapy such as moderate to severe dehydration. The pulse rate may be high normal, capillary return may be a little prolonged and respiratory rate may be normal. (It is likely on balance that Lucy was at this stage before her infusion was set up although Dr Moira Stewart has placed Lucy in the category of circulatory failure as in the 2nd group above).

55) Note on deaths in childhood gastroenteritis

56) Deaths in childhood related to gastroenteritis in United Kingdom are now very rare (in contrast to the developing world where gastroenteritis forms one of the commonest causes of childhood death). In Annex B I provide some information upon the scale of this but in summary gastroenteritis is very common in children, a proportion are admitted for treatment to hospital and it is the commonest recorded diagnosis in paediatric admissions in England and Wales. Between 10% and 25% of the 44,000 or so children admitted each year with gastroenteritis receive intravenous fluid therapy. There were approximately 2-4 deaths a year in children aged over 12 months in the years 1999-2000 which were coded as associated with gastroenteritis in England and Wales. In my own experience of being involved in acute emergency general paediatric practice over 36 years before retirement I have been involved in the care of some 70,000-80,000 acutely ill children of whom approximately 5000-8000 have had gastroenteritis. I can recall only one death from viral gastroenteritis and that was associated with difficulty in

treating the shock and not related to cerebral oedema. Many children over the years in UK with gastroenteritis will have been treated with hypotonic IV fluids and some with higher volumes than indicated for their condition. Deaths from cerebral oedema associated with gastroenteritis have been reported but are exceptionally rare.

57) **Note on Deaths in childhood :** These can be categorised for discussion as follows

58) An **Expected/anticipated** death. (Such as in a child with a chronic life-threatening/life limiting conditions such as severe cancer, chronic disorder of brain, kidney, heart etc.)

59) An **Unexpected** death which may be either explained or unexplained

- *Explained* such as death following an acute onset of an illness with a high mortality such as meningococcal disease.

- *Unexplained* such as death occurring in the course of a disorder in which there is low mortality and in which the mechanism for death is not identified leading to questions about how this had resulted as the outcome.

60) In April 2000 Lucy 's death was unexpected and from the Erne hospital viewpoint was at that time unexplained and at RBHSC the "explanation " which was included in the death certificate issued by them was not logical.(*1 (a) cerebral oedema; 1 (b) dehydration; 1 (c) gastroenteritis.)*

61) Cerebral oedema: relevant summary description

62) Described simply, cerebral oedema is swelling of brain tissue in the brain cells with or without interstitial brain tissue swelling. Cerebral oedema may occur either in otherwise normal brain cells or, in cells which have been damaged by an illness. In the first instance if a normal brain cell is surrounded by fluid which has a more dilute chemical concentration than the concentration within the brain cell-such as when the extra cellular fluid is hypotonic as in hyponatraemia, then fluid moves from the low concentration surrounding fluid into the higher concentration intracellular fluid and brain swelling results. In the 2nd instance when cerebral oedema follows damage to brain cells as a primary event from a process such as encephalitis, or after shortage of oxygen or poor perfusion of the brain, the chemical concentration in the cell becomes higher than normal and the brain cell can swell even if the surrounding extracellular fluid is of normal chemical concentration. If the cell membrane or function is damaged by such a disease process of the brain cell, then fluid movement into the cell may be even higher. The swelling then becomes worse if at the same time the surrounding fluid is hypotonic for any reason. In Lucy it is probable that the first mechanism applied because there is no suggestion from the autopsy that there was a brain cell damaging illness present such as encephalitis. This process has been challenged in evidence given to the Inquiry and I refer to this in Para 616-679.

63) **Note on the probable mechanism resulting in cerebral oedema in Lucy and cause of death**

64) Lucy suffered respiratory arrest resulting from cerebral oedema and irreversible brain damage 7 ½ hours after admission to hospital. The cerebral oedema and pulmonary congestion which were found in Lucy on autopsy suggest the presence of excessive fluid in the body as did the increase in body weight between admission and death. Lucy had a normal blood sodium on admission and this level had fallen by the time of the collapse (from 137 to 127 mmol/l) and possibly to even a lower level because the second blood test was done after a high volume of normal saline had been given rapidly which could have increased the sodium level on the test. Hyponatraemia can be due to excessive water present in her body. In a child who had presented with a degree of dehydration, as in Lucy, and who was not taking an excess of dilute fluid orally, the only way that fluid could be introduced over the 7 ½ hours she was in hospital before her collapse would be through an intravenous infusion.

65) During her admission at Erne hospital Lucy was given intravenous (IV) No 18 solution used wrongly both in volume and rate because the regime used was inappropriate to her clinical condition before her respiratory arrest; and, then the situation was compounded by adding an excessive volume of normal saline during the respiratory resuscitation on 13 April.

66) No 18 solution used in Lucy was given at 100 ml per hour until the respiratory arrest. This is at a rate and volume used for circulatory resuscitation and No 18 solution is not correctly used for that indication. It is hypotonic with a low sodium content and can thus dilute the blood sodium if used wrongly. The volume given was well in excess of current practice and guidance for the time

67) Hyponatraemia can occur in any dehydrating illness from sodium loss in fluids (diarrhoea and vomiting) but is then usually found at presentation. Lucy had a normal blood sodium on admission and hyponatraemia developed following IV fluid therapy. Hyponatraemia can be associated with a dilutional effect either by excessive administration of low solute fluid or by excess secretion of antidiuretic hormone (the so-called syndrome of inappropriate ADH secretion) or a combination of both. The reduced level of blood sodium at the second test was of mild degree but the rapidity of fall in blood sodium (regardless of the absolute level) was a risk for development of cerebral oedema known to some extent at the time. It is probable that Lucy developed SIADH and the fluid regime used combined with this response to dilute the blood sodium and thus contributed or caused the cerebral oedema which occurred which may also have been exacerbated by the high volume of 0.9% (normal)saline run in at the time of the seizure.

68) CLINICAL CARE RECORDED IN THE ERNE CASE NOTES

69) On admission at 1930 hours Lucy was assessed by the senior house officer - Dr Malik - diagnosing a viral infection and planning blood investigations and intravenous fluids. Dr Malik was unable to establish a drip (intravenous infusion) but was able to send blood tests at 20:30 hours (lab report timed 20:50 hrs[14]) which showed an elevated urea of 9.9 mmol/l and a normal blood sodium at 137 mmol/l with a slightly reduced carbon dioxide consistent with a metabolic acidosis of mild degree. Lucy passed urine at 8pm (fluid balance chart[15]) or 9pm (nurse record[16]). (The fluid chart also records damp nappy at 11 pm).

70) Dr Malik, called in the consultant Dr J O'Donohoe who attended the Ward at a time not clear from the records. Dr O'Donohoe did not record his clinical assessment of Lucy. A venous cannula was inserted by Dr O' Donohoe (from the nursing record 2230 hours was reported as the start of the infusion[17]). The infusion may have started later because on the fluid chart [18] it is listed as starting at 23:00 hours although it is possible to interpret this chart as that Lucy had received 100 mls by 11 PM. Dr O' Donohoe has made a case note entry -which is probably retrospective -that (approximately symbol) 2300 hours "*line inserted".*[19]. The intravenous fluid set up was 0.18% saline in 4 % dextrose (No 18 solution) , and was run at a rate of 100 mls per hour. Two days later Dr O'Donohoe recorded in the case notes on 14 April 2000 that on 13 April 2000 he had told Dr Crean, who was enquiring from RBHSC on the telephone, that he had ordered 100 mls per hour for the first hour to be followed by 30 mls per hour of 0.18% saline in 4 % dextrose and also that he asked that Lucy be given 100 ml orally before the infusion started whilst waiting for the local anaesthetic cream to act. [20]. But the nursing staff and Dr Malik understood the instruction to be that 100 mls per hour be given IV until Lucy passed urine.(recorded in the nursing record as " *commenced at 2230 at 100 ml /hour to encourage urine output* "[21]). Up to 03:00 when Lucy had a respiratory arrest she was treated with 100 ml/h of No 18 solution.

71) Whilst on the intravenous infusion, Lucy vomited at 00:15 hours on 13 April and was sleepy. At 02: 30 hours Lucy passed a large volume offensive stool and was moved into

[14] . 027-012-031

[15] 027-019-062

[16] 027-027--058

[17] 027-017-058

[18] 027-019-062

[19] 027-010-022

[20] 027-010-024)

[21] 027-017-058

a side ward. At 02:55 hours Lucy had an apparent seizure, the nurse attended followed shortly afterwards by Dr Malik who prescribed rectal diazepam 2.5 mg which was followed immediately by another large loose stool. Dr Malik called Dr O'Donohoe to attend to reassess and before Dr O'Donohoe arrived at around 03:20 (03:20 Dr Malik note[22]) Lucy experienced breathing difficulties requiring bag and mask resuscitation. Intravenous fluid was changed to normal saline. The time the saline started was not recorded in the records and the intravenous rate was not recorded other than "*running freely*". The case record fluid chart shows 500 ml entered at 03:00 [23]. The admission nursing record from Erne HDU/ICU shows that 250 ml of normal saline had been given by 04:00 [24]. Dr Malik's entry into the case record states " *NaCl 0.9%.500ml given over 60 mins".* Dr O' Donohoe reported to the Coroner in December 2003 " *my recollection is that Dr Malik had started the intravenous normal saline before calling me and that the 500ml given was virtually complete before I arrived " (013-018-066).* Thus at least 250ml of normal saline was given over 1 hour and possibly 500ml.

72) A second blood sample was obtained during the respiratory resuscitation but the case notes do not identify the sample time nor who obtained it or whether it was taken before or following the start of the normal saline infusion. But Dr O' Donohoe reported to the Trust review that it was taken by him and thus after the saline was running[25]. This sample showed a fall in the blood sodium i.e. hyponatraemia with a fall in total protein also indicative of dilution of body fluids see Table

[22] 027-010-024

[23] 027-019-062

[24] 027-025-076

[25] 033-102-292

73) Table : Summary of Erne Biochemistry results (based on 027-012)

Time	Na*	K*	Cl*	Urea*	Cr	Total protein (63-79)	CO2* (22-28)
20:50 (12/4)	137	4.1	105	9.9	45 umol/l	67 g/l	16
03:57 (13/4)	127	2.5	104	4.9	28 umol/l	46 g/l	18

*mmol/l

74) The nursing record – but not Dr O'Donohoe or Dr Malik- notes 2 failed attempts by Dr O'Donohoe at intubation[26]. Dr O'Donohoe called the consultant anaesthetist Dr Auterson who attended and intubated Lucy. The timing was not recorded in the records.

75) During this respiratory resuscitation, and before Dr Auterson attended, Lucy was given a high volume of normal saline (250 -500 ml over 1 hour) which is a rate only appropriate to circulatory resuscitation. But Lucy was not shocked at that time and did not need circulatory resuscitation. This is evident from the case record made by Dr Malik who recorded " *Heart rate above 100 during the whole time BP 90/65- on average. Did not develop cyanosis O2 saturation 85-100%".* [27]. Dr O' Donohoe made an entry timed (by an insertion in the record) at 0330 [28] *" capillary refill < 2 sec, pulse easily felt, pupils dilated and unresponsive*". Followed by "*Dextrostix (approximate symbol) therefore (symbol) normal saline*."

76) <u>Fluid summary before admission to ICU Erne Hospital</u>

77) Lucy was transferred to the HDU/ICU in Erne Hospital arriving there at 04:30 to 04:40 hours. The timing from Dr Malik's note is 04:45 hours[29] and from the nursing record [30] at 04:40 and from the note [31] at 04:35. The nursing record shows that Lucy was on 30 ml

[26] 027-017-057

[27] 027-010-024

[28] 027-010-023

[29] 027-010-024

[30] 027-015-036

[31] 027-015-038

/hour IV of saline by then and that 250 ml of normal saline and 25 ml of mannitol had been given. [32]

78) Thus after 250 -500ml ml normal saline was given at high rate it was followed by 30 ml /hr of saline – possibly- from 04:00 hours although the high rate saline may have continued for longer. Thus by 04:40 over 5 ½ hours (IV starting at 23:00) or over 6 hours (IV starting at 22:30 hours) after IV set up before admission to Erne ICU/HDU a further 30 ml should be added to the calculation as a minimum.

79) <u>Fluid summary before admission to PICU RBHSC</u>

80) Between 0440 and admission to PICU RBHSC an additional 30 ml / hour for 4 hours was given = Total= 120 ml. On this basis the estimated total volume received IV before PICU admission would be in the order of 800-1050ml.

81) The recorded weight gain between admission Erne and RBHSC was 9.8 kg – 9.14 kg = 660 g equivalent to 660 ml fluid positive balance.

82) <u>IV FLUID THERAPY</u>

- **Fluid requirement in Lucy**
- **Fluid given to Lucy**

[32] 027-025-076

83) **<u>FLUID REQUIREMENT IN LUCY (see Annex C)</u>**

" There are many regimes in use but there is little substantial difference between them "

(Forfar & Arneil 5th edition P1295)

84) ***Maintenance*** **fluid required** : By the standards of the time No18 solution was used for maintenance.

85) Volume calculations are based on the conventional formula used (APLS and other sources see Annex C) as follows:

Body weight	*Fluid requirement per day*	*Fluid requirement per hour*
First 10 kg	100 ml/kg	4ml/kg
Second 10 kg	50 ml/kg	2ml/kg
Subsequent kg	20 ml/kg	1ml/kg

86) ***Replacement*** **fluid requirements:** Fluid volume requirements in dehydration are calculated using the following formula:

a. Percentage dehydration x Weight in kg x 10 = fluid deficit (ml).

87) The deficit is added to the daily requirement in order to calculate the 24 hour volume needed and the infusion rate. Any volume given at higher rate initial administration (e.g. as bolus circulatory resuscitation) is sometimes deducted from the following volume over 24 hours from start of infusion. However many clinicians calculate the 24 hours from after the initial bolus has been given although views on this point differ and advise inclusion of the bolus and thus reduction of the subsequent IV rate and volume unless continuing fluid losses occur (as they did in Lucy when she developed diarrhoea and had further vomiting). *Replacement fluids* should be either 0.45% or normal saline and advised practice for the time (APLS and Forfar & Arneil see Annex C) was to use 0.45% saline when using IV fluid for replacement/maintenance in the early part of the fluid regime in dehydration. In 2000 it was still the case in a number of units in a child with vomiting and failure to drink with no or mild dehydration to use No 18 solution in the presence of a normal blood sodium level at the start of the infusion (see RBHSC 1997 First Edition) and to alter the fluid sodium content dependent on the clinical condition, for instance, if voluminous diarrhoea developed an increase in the sodium content to 0.45% or 0.9% would be indicated. Initial hourly volumes in more severe dehydration or with continuing high losses volumes could be given up to 10-20 ml/kg/hour for up to 4 hours (in Lucy thus 91-183ml/hour) but only then using normal saline. No 18 solution was often

used in later period of fluid regimes if IV therapy was still required e.g. in a child reluctant to drink.

88) ***Circulatory resuscitation*** **bolus requirement:** When IV fluid is used as a bolus for management of circulatory failure either in established shock or when trying to prevent evolving shock/ circulatory failure then normal saline is indicated (or sometimes colloid such as plasma). A bolus of fluid can be used at the start of an infusion either by "pushing" in over 10-20 minutes or by arranging for the amount to be given over the first hour. After a bolus has been, given the hourly rate and volume should be reduced to that required for replacement/maintenance although initially higher hourly volumes could be used.

89) A bolus can be used in established shock of 20 mL/kilogram of body weight (and if necessary repeated up to 2 times) but when trying to prevent evolving shock (such as arguably in Lucy) then a volume of either 20 mL/kilogram or 10 mL/kilogram could constitute the bolus. Initial hourly volumes in more severe dehydration or persistent signs of circulatory failure could be given as up to 10-20 ml/kg/hour given for up to 4 hours (in Lucy thus 91-183ml/hour).Fluids with low sodium content including number 18 solution, should not be used for bolus or hourly high volume purposes.

90) **Requirement for Lucy's clinical presentation and disorder**:

91) **Replacement/maintenance**:

92) Lucy required replacement of between 5% and 7.5 % dehydration. She weighed 9.14kg and thus required an intravenous infusion of either 57 ml /hr (5%) or 67 ml /hr (7.5%) for replacement and maintenance. By the standards of the day for replacement and maintenance Lucy should have been treated with 0.45 % saline in dextrose.

93) The hourly rate is often higher for the first few hours than the 24 hour calculation and may need to be increased again if there are further large losses such as voluminous diarrhoea.

94)

Requirement for 9.14 kg	***24 hour volume total***	***Hourly rate ml/hour***
Maintenance	914	38
Replacement ***7.5%*** dehydration	686	29
Maintenance + Replacement ***7.5%*** dehydration	1600	67
OR :		
Replacement ***5 %*** **dehydration**	457	19
Maintenance + Replacement ***5%*** dehydration	1371	57

95) Circulatory resuscitation bolus requirement:

96) Whether Lucy required a bolus of fluid is debatable.

97) It is arguable that no bolus volume was required because the mucous membranes were moist (the nursing record noted she had moist mucous membranes) [33]and Lucy was passing urine; her pulse rate at 140 was at the high end of the normal range for her age (APLS age < 1 year up to 160/min and age 2-5 years up to 140/min), her respiratory rate was slightly elevated at 40 (APLS age < 1 year up to 40/min and age 2-5 years up to 30/min), Dr Malik on admission noted :

   a. *Vomiting everything she eats and drinks. "She passes stool normally" "now for the past 12 hours she is very sleepy" ; conscious and pink. Weight 9.14 kg. Temperature 38°. Heartrate 140/minute. Respiratory rate 40/min;Capillary refill > two seconds,chest clear. CVS S1+ S2+0. Abdomen soft bowel sounds +.CNS NAD Diag (triang) viral illness*

98) Thus from the records it is possible to conclude that on admission she had at most moderate dehydration and that she was not in established shock to the extent of being hypotensive nor was her pulse rate increased out of range: a sign of more severe circulatory insufficiency/ failure, nor is there any description consistent with this from the nursing notes.

[33] 027-017-056

99) Dr O' Donohoe for the review reported [34] that after he had set up the infusion at around 23;30 hours " *I looked into the treatment room a few minutes later and Lucy was standing on the couch in front of her mother and looking better. I was next called at approximately 03:00 hours because Lucy had what sounded like a convulsion. My initial presumption was that this was a febrile convulsion."*

100) On the other hand her capillary return was prolonged at > 2 seconds (normal < 2 sec) suggesting a degree of poor perfusion and the blood urea was elevated. Without prompt treatment Lucy could have progressed to established shock. It was therefore within the range of responsible practice, based upon advice available at the time, to use a bolus of at least 10 mL per kilogram and up to 20ml/kg but this should only have been given as normal saline.

101) Grading of degree of dehydration by clinicians

102) The clinicians (including Dr Quinn) who have reviewed her records differ in their assessment of the degree of dehydration present. In his written report Dr Quinn does not commit himself on this point but calculated differing options although it is noted from his meeting with Dr Kelly and Mr Fee on 21 June 2000 that he considered Lucy to be moderately dehydrated [35].

103) Dr Moira Stewart concludes Lucy had moderate to severe dehydration on admission and calculates fluids on the basis of 7.5 % dehydration and considered that a bolus of 20ml/kg, as normal saline should have been given for *"a degree of shock" (see below).* Dr Jenkins in his medical legal report for Erne Trust concludes dehydration as mild as does Dr Sumner for the Coroner in 2004 (5%) and Dr Evans for the parents estimates 7.5 %., and calculates, as Dr Stewart, on the basis of a weight of 10Kg[36].

104) My opinion based on review of the notes is that an estimate 5%-7.5% would be reasonable and that there was no convincing evidence of imminent or established shock.

105) But assessment of dehydration is subjective and not consistent between observers. (see *Armon K, Stephenson T, MacFaul R, Eccleston P, Werneke U, An evidence and consensus based guideline for acute diarrhoea management. Arch Dis Child 2001; 85:132-142).*

106) All opinions (save Dr Quinn in his report) are agreed that Lucy required IV treatment with fluid with a higher sodium content than the Solution 18 (0.18% sodium) which was used.

[34] 033-102-292

[35] 036a-047-101

[36] 013-010

107) When reporting in April 2001 for the Trust Dr Moira Stewart (following her assessment of the clinical record) noted that :

a. "*Clinical examination as documented, was essentially normal, but she did have prolonged capillary refill time indicating a degree of shock."*

b. and *….*

c. *. " These results indicate moderate-severe dehydration with a degree of pre-renal failure. The low CO2 suggests compensated metabolic acidosis (we do not have arterial or venous astrup results). The plan was to encourage feeding, and commence intravenous fluids after cannulation. Given the symptoms and signs, and the prolonged capillary refill time (>2 secs), it would be appropriate to give an immediate fluid bolus of up to 2Oml/kg (N Saline, or less commonly, colloid) and then reassess "*

108) Dr Sumner reporting to the Coroner reported [37]

"It is difficult to judge exactly how dehydrated Lucy was on admission to hospital. A capillary refill time in excess of 2 seconds is one sign of approximately 5% dehydration, however, this sign is likely to be hard to interpret in a febrile child At this level of dehydration, mucous membranes are dry, but it was noted that Lucy's tongue was moist. I think on balance that she was mildly dehydrated - perhaps somewhat less than 5% and involving a fluid deficit of approximately 350 ml.

"An appropriate fluid would be normal (0.9%) saline with a potassium supplement (eg 10 mmol in 500 ml Hartmann's or lactated Ringer's solutions. This could have been as an initial bolus of 100-150 ml over the first hour, to cover approximately half the calculated deficit then the rest of the deficit plus normal maintenance fluids could be given over the next 12-24 hours. The deficit by this stage would be approximately 2O0ml (8ml per hour) plus normal maintenance of 5ml per kg per hour - a total of approx 50ml per hour, most of which should be saline, though some could be a dextrose containing solution."

[37] 013-036-136

109) The following table shows the ***maximum*** volumes ***indicated*** for Lucy based on Dr Stewart 's opinion with IV starting 22:30 and running for 4 ½ hours up to 03:00hours.

	Weight 9.14 kg	***Weight 10kg (used by Dr Stewart)***
Bolus 20ml/kg on admission	183 ml	200 ml
7.5 % deficit	686 ml	750 ml
Maintenance	914 ml	1000 ml
TOTAL over 24 h	1783	1950
TOTAL by 03:00 including initial bolus	**467 ml***	**510 ml**

110) Note * After the bolus had been give this would have been at 63 ml/hour

111) Fluids that were given to Lucy based on information available in the records

112) **Solution 18 given:** From the records it is possible to see that by the time of the seizure at 02:55 or 03:00 hours Lucy had received IV Solution 18 at a rate of 100 ml /hour and that the total received by 03:00 hours was either 500 mls, 450 mls or 400 mls.

113) The IV infusion was recorded as started at 22:30 in the nursing record[38]. The range in total volumes in the previous paragraph estimated as given results from the confusing fluid balance chart from the children's Ward starting 12/4/2000[39]. This shows 100 mls of number 18 solution given by 11 PM when the nappy was recorded as damp. It then shows a recording that 100 mls was given at 12 midnight, 100 mls at 1 AM, 100 mls at 2 AM but then the entry at 3 AM simply has 500 mls recorded (this being the start of the normal saline see below). Thus it is possible that between 2 AM and 3 AM a further 100 mls of Solution 18 was given. This would represent a total of 500 mls of Solution 18.

114) **Normal (0.9%) saline given :** During the respiratory resuscitation started at 03:00 normal saline was given running " freely" and the records suggest either a total of 250 ml or 500ml had been given by 04:00 hours. Lucy probably had at least a total of 650 or 750 ml (plus a further possible 250ml) infused over the period between the setup of the intravenous infusion and her admission to the high dependency/intensive care unit at Erne. Based on the clinical note made by Dr Malik and Dr O'Donohoe, Lucy

[38] 027-017-058

[39] 027-019-062

did not need circulatory resuscitation with normal saline at the respiratory arrest at 03:15 hours yet the volume of normal saline given was appropriate only for that purpose.

115) **FLUID CALCULATION** The following table shows volumes which could have been received by 03:00 hours using regimes proposed by Dr Stewart and Dr Sumner in their reports. It assumes that the intravenous infusion started at 22:30 hours; **body weight of 9.14 kg** ;there was either 7.5% or 5 % dehydration on start of IV fluids.; that " push " bolus was given over 15min and thus was given by 2245hr after which the rate was 67 or 57ml/h

Option	***Bolus over ¼ hour***	***Dehydration***	***Hourly rate ml***	***By2330***	***Hours 2330-0300***	***Total by 0300***	***Fluid % saline***
Dr Stewart	20ml/kg= 183	7.5%	67	0.75x67=50 +183=233ml	3.5x67 =234ml	467	183 ml *0.9%* + 284 ml *0.45%*
DrSumner	10ml/kg=91	5%	57	0.75x57=43 +91=134	3.5x57= 200ml	334	91 ml *0.9%* +243 ml *0.45%*
Dr O'D intended	100ml (over 1 hr)[40]		30	130	3.5x30= 105ml	235	335ml *0.18%*
No bolus option	none	7.5%	67	67	4.5x67= 301ml	302	302 ml *0.45%*
Given			100	100 (or 150)	4 ½	450 (400-500)	450ml *0.18%*

116) Note: Dr Evans for parents proposed bolus of 180 ml followed by 70ml/hour of 0.45% saline and commented *" infusion of a too large a volume, most of which was too dilute"*[41]

117) ***Volume by 03:00h :*** From above Table it can be seen that volume of No 18 solution given used was far too high. From guidance if this volume was to be given, 40% should have been as normal saline and 60% as 0.45% instead it was all given as No 18 solution.

[40] 027-010-024 " *bolus of 100ml over 1 hour followed by 0.18% NaCl/Dextrose 4% at 30 ml/hour"*

[41] 013-010

118) **Hourly rate:** After an initial bolus, the hourly rate given was too high at 100 mL per hour in contrast to the volume indicated of 67 mL per hour. (In severe shock up to 20ml/kg/hour - in Lucy 183 ml/h- may be needed but then only as saline and for less than 4 hours. Lucy was not severely shocked).

119) **Fluid type:** There was an excessive volume of number 18 solution given. Any bolus of fluid administered over the first hour should, by the standards of the day, only have been normal saline and the remainder as 0.45% saline over the first hours. On this basis there was an excess volume of number 18 solution given over a period of 4 ½ hours (and possibly a further additional 50ml). The compounded by a very large and excessive volume of 250-500ml of normal saline over one hour.

120) Conclusion

121) By the standards of the day as set out in the guidance shown in the Annex C (Forfar & Arneil 5th Edition – current in 2000 and Advanced Paediatric Life Support Manual (APLS) it is evident that an excess volume of number 18 solution was given up to the respiratory arrest at around 03:00 hours and the treatment profile used in terms of the rate of 100 mL per hour was incorrect.

122) At the time of the respiratory arrest, normal saline was run in-"running freely" and the volume appears to have been 250 mL -500ml administered over one hour. This is a grossly excessive volume and there was no evidence from the clinical records that this bolus was required.

123) In sum the records show that a grossly excessive volume of fluid had been given to Lucy IV at Erne Hospital of the wrong type for her clinical condition. The regime contributed to or caused the cerebral oedema.

124) THE ERNE HOSPITAL/SPERRIN LAKELAND TRUST REVIEW INCLUDING DR QUINN'S REPORT

125) Lucy died on 14 April 2000. On that day the treating consultant at the Erne Hospital, Dr O'Donohoe, informed Dr Kelly the Medical Director of the event. The Medical Director advised Mr Mills, the Chief Executive and Mr Fee, Director of Acute Hospital Services. On the same day a clinical incident report was initiated by the Ward Sister, Ms Traynor, reporting that the senior house officer had been unable to set up an intravenous infusion and expressing concern about the fluids prescribed and administrated, and reporting that Lucy had *"collapsed unexpectedly. Cause unknown*"[42].

126) On 14 April 2000, Mr Mills advised Dr McConnell, Director of Public Health of the WHSSB of Lucy's death. In turn Dr McConnell undertook to inform Martin Bradley,

[42] Ref:036a-045-096 to 036a-045-097

Director of Nursing WHSSB.[43] That day, Mr Fee agreed with Mr Mills to coordinate a detailed case review together with Dr Anderson (Clinical Director Women and Children's Services).[44]. Mrs O'Rawe , Director of Corporate Affairs at Sperrin Lakeland Trust was also informed by the Chief Executive who became aware of press interest on 17 April. On that day Mr Mills reports that the Chairman of the Trust was informed.[45].

127) On 17 April 2000 Mr Fee advised Dr Hamilton at the WHSSB of Lucy's death[46] - a medical consultant at WHSSB who commissioned acute hospital services . [47]

128) On the 17th and 18 April Dr Anderson and Mr Fee met Dr O'Donohoe, Dr Malik the Senior House Officer, Sister Edmondson (Night Manager at Erne Hospital), Staff Nurse McManus, Enrolled Nurse McCaffrey and Staff Nurse McNeill. A record made in appendix 6[48] of the review report notes that the staff were offered support and were advised of the intent to conduct a review.

129) On 19 April Dr Anderson and Mr Fee met to agree an action plan which is set out below. Mr Mills met with Martin Bradley WHSSB and also noted that Dr McConnell WHSSB was advised that circumstances were still being examined. Dr Anderson and Mr Fee agreed to seek a written report from the consultant anaesthetist Dr Auterson which was provided on 20 April. On that day it is noted by Mr Mills that Mr Fee had reported to him that there was some uncertainty about the instructions given to staff about the rate of flow of intravenous fluid, and that when the child collapsed, anaesthetic support prescribed more fluids.[49] However, there is no documentation of this action by the anaesthetist nor is it reported by Dr Auterson. The Trust was aware that the Belfast post-mortem reported cerebral oedema

130) The Action plan:[50]

131) Dr Anderson and Mr Fee by met on 19/4/2000 to review the case records and agreed:

[43] Ref: 030-010-017

[44] Ref: 030-007-012

[45] Ref: 030-010-017

[46] Ref: 033-102-286

[47] WS 287/1

[48] Ref: 033-102-285

[49] Ref: 030-010-017

[50] 033-102-285

132) That staff who were listed attending meetings on 17th/18th of April with Dr Anderson and Mr Fee: (namely Dr O'Donohoe, Dr Malik, Sister Edmondson, Nurse McManus, Nurse McCaffrey and Nurse McNeill) and Dr Auterson Consultant Anaesthetist would be asked to provide a factual account of the sequence of events from their perspective.

133) That the case notes/copy of case notes would be made available for reference to those concerned. Dr Anderson agreed to get a copy of the case notes made and to have both a copy in the original retained in Mrs Millar 's office for the immediate future.

134) Dr Anderson is to speak to Dr O'Donohoe and request that he share with staff concerned, in confidence, the verbal report of the cause of death received.

135) Mr Fee is to seek an appropriate method of advising Lucy's parents that "we will arrange an opportunity to share with them information on the nature of Lucy's illness, the treatment given, and the cause of death, addressing where possible, any questions they have, when we have established necessary information and facts".

136) Mr Fee will speak to Ms Murphy, Health Visitor Manager, to establish what support is being given to the family and if it is possible to make this offer through the health visiting service.

137) Mr Fee is to establish from the infection control service, the nature of rotavirus infection.

138) It was agreed that Dr Anderson and Mr Fee would need an external expert paediatric opinion on the management of Lucy's care. Mr Fee is to test the source of such an opinion with Mr Mills.

139) Dr O'Donohoe and the staff concerned are to be encouraged to consider creating an opportunity to talk through the issues and emotion surrounding this case. Mr Fee and/or Dr Anderson would facilitate such a discussion.

140) Mr Fee and Dr Anderson gave consideration to whether or not the work arrangements require modification for any of the staff involved. In the absence of an expert opinion on the likely significance of the care given having contributed to the deterioration in Lucy's condition and the unlikely event of a recurrence of a similar outcome of the child presenting with this type of condition it was decided that no alteration to the work arrangements for those concerned would be appropriate at this stage.

141) On 20/4/2000 Mr Mills agreed to Mr Fee's request that he needed advice from a paediatrician and that he, Mr Mills, would arrange this. And they also considered whether Dr O'Donohoe should continue to see and treat patients. Mr Anderson and Mr Fee expressed the opinion that he should continue to do so. Mr Mills discussed how

best *"we should communicate with the family to advise the circumstances were still being examined".*

142) Involvement of nursing staff in the review process.

143) On 21/4/2000 letters were sent to Nurses McNeill and McCaffrey[51] stating:

144) "*The purpose of this review is to try and gain a clearer understanding of Lucy's deterioration and identify if there are any lessons to be learnt. I would ask that you provide me with a factual account of the sequence of events, in relation to Lucy's care where you were involved*."

145) In a different letter to Nurse McManus[52] specific responses, including opinion , as well as fact, were requested as follows:

a. *What advice/recommendations do you believe Dr O'Donohoe gave in relation to the volume and type of fluids to be given?*

b. *Over what period was it to be given?*

c. *To whom were these instructions given?*

d. *Are such instructions/prescriptions normally written?*

e. *Would this volume be consistent with the volume normally given to a child of this age and weight?*

f. *Can you clarify from the fluid balance chart for me the actual volume administered over the period 11.00pm on 12 April until 3.00am on 13 April 2000?*

146) The report to the review from Nurse Jones[53] dated 18 May 2000 confirms entries made on the fluid balance chart at 1.00am, 2.00am and 3.00am were made by her.

a. "*The amounts of fluid is noted to the left of each box give a complete and accurate record of all intravenous fluid dispensed during that period. However I do note that the running total is indicated to the right of each box has not been tallied correctly* ."

147) But this information is insufficient to clarify how much fluid was given. I consider this inadequate response simply compounded the difficulty in interpreting the fluid balance chart and should have been subjected immediately to an enquiry made of her

[51] Ref: 033-102-297 to 033-102-298, and 033-102-302 to 033-102-303

[52] Ref: 033-102-299 to 033-102-300

[53] Ref: 033-102-320

by Mr Fee and Dr Anderson and a summary made of the fluid that had been administrated.

148) On 26 April, Nurse McManus wrote in response [54] by raising questions about the status of the request. She refers to the fact that the nursing documentation should be sufficient and her wish to able to seek some outside advice to ensure that what she reports is done correctly. Subsequently Mr Fee spoke to her and made notes dated 27 April 2000[55].

149) "Mr Fee spoke with Staff Nurse McManus on the telephone regarding the contents of her letter. She confirmed she had no direct involvement in the administration or recording of fluids to Lucy Crawford."

150) Whilst awaiting reports, Mr Fee recorded that on 27 April 2000 he discussed the case with Sister Traynor and Nurse Swift. The appendices to the review produced in July do not include a written report from Sister Traynor nor from Sister Edmondson.

151) Sister Traynor offered her opinion and this was noted by Mr Fee as follows:

152) "Mr Fee spoke with Sister Traynor who commented that the fluid replacement volume was not unusual in a child of this age given her condition. She also stated that there did not appear to be evidence of overload of fluids. We reviewed the notes again. Sister confirmed that the rate to be administered would normally be recorded on the fluid balance chart along with the type of fluids. Mr Fee spoke to Staff Nurse Swift who confirmed that she and Dr Malik were present when the fluid regime was commenced by Dr O'Donohoe. She states they were advised to administer 100ml per hour until Lucy had produced urine. Nurse Swift was not involved in recording the 2.00am or 3.00am record of the fluid balance chart. She suggested that it was possibly Nurse Jones. Nurse Swift agreed to provide a report."

153) **Ms Traynor** explains she was formerly the Ward sister on children's ward Erne hospital when Lucy was admitted but was not involved in Lucy's care as she was not on duty. Sister Traynor made a statement to the police[56] that she had no role in Lucy's care and came on duty on the children's ward Erne Hospital on the morning of 13 April. She raised an incident form because of Lucy's sudden death and lack of detail recorded on the nursing notes and fluid balance chart. Ms Traynor indicates that she met Mr Fee and Nurse Swift on 27th of April 2000 to discuss the concerns raised and reports that she was preparing a draft policy protocol on fluid management for the paediatric Ward. Ms Traynor does not recall having seen the statement attributed to her by Mr Fee that she did not consider there was a fluid overload as the note had not been shared with her.

[54] Ref: 033-102-314

[55] Ref: 033-102-295

[56] Ref: 115-020

Ms Traynor believes her comment as documented to be inaccurate and she could not have given a fully informed answer to Mr Fee's question and in relation to a question about the 100 mL/hour volume used she had responded that this could be the case in older children as they were admitted up to age 16. Comment: this failure to share Mr Fee's note included in the review report is a further example of the deficiency of the review process in failing to share it with those who had contributed in order to clarify any incorrect entries and to reconcile different accounts. [57]

154) Nurse Swift wrote a report enclosed as an appendix in the review. Ms Swift answering explains that she does not recall meeting Mr Fee and Sister Traynor on 27th of April 2000. But that she had no concerns about the fluids prescribed or administered to Lucy or how it was done. [58]

155) Mr Fee's role in the review

156) Mr Fee explains[59] he had a background in nursing and that in the Sperrin Lakeland Trust he was Director of Acute Hospital Services with the following responsibilities

- Corporate responsibility with fellow Directors of the Trust to manage the Trust as an organisation.
- A member of the Trust Board and an Executive Director of the Trust .
- Operational director for acute hospital services which included the Women's And Children's Directorate. Dr Anderson was the Clinical Director and Mrs Millar was the Service Director for that division.
- The professional nursing lead across the Trust .

157) And that he had some prior experience in conducting reviews as an NHS manager.

158) Actions taken with the Review report

159) Mr Fee reports that when the review was completed it was sent to the Chief Executive and to Dr Kelly.[60] In November 2012 he recalls that the report was given to Mr Mills Chief Executive. Subsequently Mr Fee was not involved in any discussions with regard to the findings of the review to the extent that they concerned the cause of the deterioration and death of Lucy. Mr Fee explains that he expected that the death would

[57] WS-310-1 Q2 P 3; Q3 P4 ; Q3 P6 (h), & (e)

[58] WS-311/1 Q1 P 2

[59] Ref: 116-031-001 to 116-031-003

[60] 116-031-001 to 116-031-003 .

have been reported to the Coroner in Belfast. He explains that he did not recall having had sight of copies of Dr Moira Stewart reports or those of Dr Jenkins or Dr Boon in 2002 and he does not recall any discussion relating to them.[61]

160) Mr Fee did not receive any formal steer about further action after July 2000 although he was aware that the Chief Executive briefed the Western Health and Social Services Board and that Dr Kelly had briefed Dr McConnell and written to him. He was aware that Dr Kelly had discussed the review with the lead clinician for paediatrics Dr Halahakoon. He, Mr Fee, did not see it as his role to discuss the review with Dr O'Donohoe nor did he discuss the findings with Dr O'Donohoe or Dr Malik[62] .

161) Although Mr Fee was the lead nurse for the Trust he was not aware that anybody spoke to individual nurses about improving their documentation for prescription nor as it turns out was this step taken with Dr Malik. However an aide memoire relating to how to prescribe fluids was constructed for the Ward by the nursing staff..

162) Mr Fee was not aware that a death certificate had been issued. He only became aware that no Inquest was planned during the litigation process. Mr Mills stated that this was in October 2001. [63]

163) Medical Contributions to the Review and my comments

164) **Dr Malik**, Senior House Officer, produced a report 5 May 2000.[64] He does not mention that he was present at the time the fluid started yet it was he who signed the prescription which was inadequately completed simply stating number 18 solution, countersigned by nurse Swift and without stating the volume or rate to be given. He made no reference to his recording that 500ml normal saline was given. in his case note entry made after the respiratory arrest. . Given the uncertainty about the fluids, he should have been asked to provide a report on those specific points.

165) **Dr O'Donohoe.** The report by Dr O'Donohoe is undated but probably attached to the letter he wrote to Dr Anderson, which oddly is dated 5/3/2000.[65] he gives an account of what he did when he attended, and states that while strapping the cannula in situ " *I saw Dr Malik writing as I was describing the fluid regime i.e. 100 mL as a bolus over the first hour and then 30 mL per hour"*. And he describes his intention as follows *"to cover the possibility that the cannula might not last very long and the succeeding rate was relatively slow since I had seen her taking oral fluid well and presumed the rate of fluid*

[61] WS 287/1 Q 47 P 18; & P20 & Q49

[62] 0116-034-011

[63] WS 293/1 Q36P19.

[64] Ref: 033-102-281 to 033-102-282

[65] Ref: 033-102-292 to 033-102-293

needed was relatively small." He reports Lucy standing on the couch in front of her mother and looking better a few minutes later. This is an important contribution because it suggests that Lucy was not shocked and thus to continue 100 ml per hour was inappropriate. There is no entry in the case records by Dr Malik recording Dr O'Donohoe's instructions. Dr O'Donohoe describes that it was he who took the blood for repeat urea and electrolytes and that he had noted the blood sodium falling to 127. Also that he accompanied Lucy to Belfast and "*I was unable to make a diagnosis for her deterioration prior to transfer".* He makes no mention of the infusion of normal saline which must been running at the time he took a blood sample. There were thus a number of questions which rose from this inadequate report and an interview should have been held with him or alternatively a letter written to clarify the points. Dr O'Donohoe reports that he was asked to set out the facts in his report for Dr Anderson and that before or after he had submitted the report for the review he was not interviewed by anyone in the Trust in relation to the contents of his report. Also he had not raised a question within the Trust or RBHSC about the large volume of normal saline which had been given nor had discussed the results of the post-mortem.[66] Nor did he report to the review his telephone discussion held with Dr Crean nor that the normal saline volume started by Dr Malik had been almost run through when he attended as he reported to Dr Kelly in August 2003.[67]

166) **Dr Auterson:** Dr Auterson confines his contribution to the review to a factual statement. (he recalls in his November 2012 WS that he was only asked by Dr Kelly or Mr Fee - verbally - to provide a factual statement surrounding his part in the incident). [68] It is not evident whether he was informed about the scale and purpose of the review. His opinion on Lucy's care was not sought and he offered no opinion to the review or Trust then about the fluids used. He telephoned the Belfast hospital with the results of the low sodium and was clearly concerned about this. He recalls that he received the lab results during his resuscitation from a nurse who took the result by telephone from the laboratory. He reports that after Lucy's death a review of the fluid balance chart and lab results led him to believe that hyponatraemia played a significant part in Lucy's deterioration and death and he believed that the quality of care was less than satisfactory in regard to the prescription of intravenous fluids and record of subsequent administration. He thought the death was probably avoidable. Dr Auterson believes he discussed the case informally with Dr O'Donohoe, Dr Anderson and his anaesthetic colleagues in the 24-48 hours after the event.[69] Nobody at the Trust asked him to assist in understanding the cause of Lucy's death and/or whether medical treatment received may have contributed to it. In his deposition to the Coroner [70]Dr Auterson states that the

[66] WS-278/1 Q12 P7; Q15 P9; Q12 (c) P12

[67] 047-053

[68] WS-274/1

[69] WS-274/1 Q 11 (d) , (i), Q8 , Q15

[70] 013-005-093

hyponatraemia occurred to him when he got the lab results. He agreed that Lucy had received too much of the wrong fluid. In my opinion Dr Auterson should have reported his concerns about the fluid regime to Dr Kelly at the time and arguably to the review. The extent to which colleagues reported what they judged to be substandard care provided by colleagues at the time was variable. This issue was addressed in *GMC Good Medical Practice 1998* para 23 & 24 which was applicable in 2000. In a later response Dr Auterson confirms the previous information. He did not specifically report his concerns about Lucy's fluid management because he thought a report was the responsibility of the paediatric team for the clinical incident review and that it was an obvious problem to him because of the sequence of events, the clinical observations and the laboratory results. [71] Comment : if the review process had taken the step of reconciling the deficiencies of information provided and in particular had interviewed Dr Auterson or requested specific responses, this issue would have come to light much earlier.

167) <u>DR QUINN'S REPORT</u>

168) When Dr Anderson and Mr Fee decided that an external expert paediatric opinion was required Mr Mills, Chief Executive telephoned Dr Quinn, consultant paediatrician in Altnagelvin Hospital, and agreed that he would review the case records and report his opinions. Dr Quinn has later stated that he placed some constraints on the report that he would produce although these are not documented at the time by Dr Quinn, Mr Mills, Dr Kelly or Mr Fee. Mr Mills explained in his police interview[72]that he selected Dr Quinn because he was well respected and points out that he did not do clinics in Erne Hospital at the time. Also that he wanted a view independent of that from Belfast because the Belfast hospital had been involved in Lucy's care. Dr Kelly and Dr Anderson reported in their police interviews[73] that they were not involved in the selection of Dr Quinn and did not know him.

169) On 21 April 2000 Mr Mills noted that he had asked Mr Fee to contact Dr Quinn to advise him of the main issues "*we need to examine"* and to forward the case notes to him. On 27 April Mr Fee informed Mr Mills that he had spoken to Dr Quinn. There is no record made of any restrictions imposed by Dr Quinn on his report or any question raised about the status of what he was being asked to do.[74] The status of the internal review being conducted was not clarified other than as *"initial".*

[71] WS-274/2

[72] 116-049-008 and 116-049-009

[73] 116-044-003 and 116-031-009

[74]

170) Mr Fee wrote to Dr Quinn after this telephone discussion as follows[75] including a wide brief

a. *"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.*

b. *These were*

c. *The significance of the type and volume of fluid administered*

d. *The likely cause of the cerebral oedema*

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

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

171) The items listed are explained in 2005 by Mr Fee in his interview with the police because when meeting with Dr Anderson to look over the clinical notes, they identified issues around the level of prescription of fluids and fluid balance and it appeared on the surface that Lucy had received approximately 400 ml of intravenous fluid. Neither he nor Dr Anderson knew whether that was correct and concluded that external help was required.[76] In Mr Mills' note[77] which appears to be a note of a conversation on Thursday 20 April between Mr Mills and Mr Fee, this volume is also mentioned in terms of Lucy being given 100 ml per hour for 4 hours. Mr Fee reports that he was only aware that an excess of fluid had been given at the Inquest in 2004.[78]

172) Telephone discussion between Dr Quinn and Mr Fee 2 May 2000

173) On 2 May 2000 there was a telephone discussion between Dr Quinn and Mr Fee. Notes of this were made by Mr Fee who listed the following relevant points.[79]

a. *Difficult to get a complete picture of the child*

b. *Type of fluids appeared appropriate. The amount given will be dependent on the level of dehydration but would expect up to 80 ml per hour.*

[75] 033-102-296

[76] Ref: 116-031-005

[77] Ref: 030-010-017

[78] WS 287/2

[79] Ref: 033-102-287

c. *When the fluids are divided over the length of stay child received approximately 80 ml per hour.*

d. *There is no clear instruction on the volume of fluid is intended nor the volume for normal saline after it was commenced.*

e. *The volume taken over the seven hour period appears reasonable.*

f. *Question why was the child floppy*

g. *Did the child have a seizure or was it rigid, a symptom of coning?*

h. *2.5 mg of Valium given does not appear excessive. She could have been given up to 4.5 mg of the Valium.*

i. *Was the resuscitation adequate?*

j. *How much normal saline was run in?*

k. *If 500 mls was given this may have affected the level of cerebral oedema experienced at post-mortem.*

l. *Was the child rigid at the time the mother called the nurse or was there an event that was in advance of the mother calling the nurse?*

174) There is then a ***footnote*** added: *"nursing staff advise that normal saline was commenced at 3:15 AM and 250 ml had been administered by 4 AM. The dose was then reduced to 30 ml per hour for the next two hours."*

175) It is not established whether the information about the start of the normal saline and the volume administered was given to Dr Quinn. Dr Quinn reports no recollection of this telephone call or its contents.[80] It is not evident either where this information comes from. Dr Malik recorded in his handwritten record that 500 mls should be given over an hour. The timing of Dr Malik's entry into the case records is not clear. The entry is followed by a handwritten note written on 14 April by Dr O'Donohoe on the same page. It is not present in any of the written reports attached to the July 200 Erne review as appendices. In his report para 13 Dr Quinn raised queries about the volume of saline but the fluid balance chart [81] which appears to be written at the time of admission to the Erne HDU shows that 250 mls normal saline in total was given.

[80] WS 279/1 Q 16

[81] 027-025-076

176) **Meeting held between Dr Quinn, Dr Kelly, Mr Fee 21 June 2000**

177) A verbal report from Dr Quinn was given on the 21 June 2000 at a meeting with Dr Kelly and Mr Fee.[82]. The points are listed below. There are no notes which indicate that Dr Quinn was reluctant to provide a written report which was provided the following day, 22 June 2000. In both this meeting and in his written report he refers to the fact that fluid was given by the anaesthetist. A note of this meeting was made by Mr Fee :

a. *Dr Quinn provided his opinion on the notes and PM report*

b. *Fluid issue: Choice of fluid correct*

c. *Resuscitation volume higher than normal.*

d. *Dr Murray outlined his expectation of fluid replacement*

e. *Maintenance 40 mL/hour*

f. *10% dehydrated 80 mL/hour*

g. *"This child was probably moderately dehydrated. Urea 9"*

h. *Fluid replacement 4 hours at 100 mL provided was greater than normal but not grossly excessive.*

i. *Dr Quinn does not feel that the extra fluids caused the brain problem.*

j. *Dr Quinn notes that there was further fluids administered after the resuscitation- 250 mL N-saline . Again "choice of fluid by anaesthetist was reasonable but volume high. Could after an hypoxic incident this have produced the cerebral oedema. Events remain unclear."*

k. *Could there have been earlier seizures resulting in hypoxia for 15-20 minutes prior to catastrophic "seizure event?*

l. *Did significant coning occur and when?*

m. *"What role the B'pneumonia"*

n. *Reviewing the PM report Dr Quinn feels it does not help us to piece together why this child died. There was rotavirus present to cause the diarrhoea but this does not appear to have been very significant.*

o. *The B'pneumonia. Dr Kelly asked is there an issue of missing this chest problem?. Dr Quinn stated he had no great concerns on this issue as it would*

[82] Ref: 036a-047-101

common for this to happen-the diagnosis can be very difficult in this size of infant until a chest x-ray is performed.

p. *Dr Kelly asked is there an issue of incompetence-should consideration be given to temporary suspension. Dr Quinn stated that he saw no reason for suspension. The issues raised by the case are more about recording fluid prescriptions carefully and ensuring clarity of instruction.*

178) DR QUINN'S WRITTEN REPORT 22 June 2000

179) I provide this report in full in Annex A and comment on each section taking account of the November 2012 Witness Statement WS 279/1 from Dr Quinn for the Inquiry.

180) Limitations on the information available to Dr Quinn when drawing up his reports

- Information on Lucy's clinical course at Erne Hospital is available from :
- the case records,

and information provided *later* :

- during the course of the internal Trust review
- from Witness Statements to the Coroner.
- from Witness Statements to the Police Service Northern Ireland

181) Dr Quinn produced his report based only on a review of the case records. Whether further information was given to him on the telephone by Mr Fee is not established. Dr Quinn reports that he did not receive and did not seek other information from the Trust before the 21/6/200 meeting with Dr Kelly and Mr Fee. He is unable to recall the telephone conversation held with Mr Fee on 2 May 2000. [83]

182) Further information was available to Mr Fee when writing his review and subsequently even more information has come from witness statements long after these events. I list some of this information below when commenting on Dr Quinn's report. I understand Dr Quinn did not see the review report or its appendices.

183) The Trust review report was not available to Dr Moira Stewart for her first review of the notes when reporting to the Trust on 26/4/2001 although she was given a copy of

[83] WS-279/1 Q9(c) P12

Dr Quinn's report. Dr Quinn only saw a copy of Dr Moira Stewart's report when preparing the November 2012 witness statement .[84]

184) I do not know whether Dr Jenkins had seen either the review or Dr Quinn's report before he produced his report on 7/3/2002 for the Trust for medico legal purposes in response to the litigation claim made by parents.

185) Information from the records would have been available to Dr Evans who provided a report for the solicitors dealing with the litigation claim on behalf of the parents. I am not sure whether the report of the Trust review was made available to Dr Evans which would then presumably be the version sent to the parents on 10/1/2001 (which was altered from the 31 July 2000 signed copy[85] of the Trust review in the recommendations section and had no appendices attached ; and thus (probably) without Dr Quinn's report).

186) <u>Information available later to Erne review or Coroner but (probably) not to Dr Quinn</u>

187) A witness deposition 17/2/2004 by Nurse Swift for Coroner [86]states that Dr O 'Donohoe asked her to give Lucy diorylate and this was given at 22:00 hours.

188) Information confirming who ordered the saline and when it was started has only become available from Coroners witness statement and appears was probably started on Dr Malik's instruction at 03:00 from the statements made by nurse Thecla Jones [87] and staff nurse McManus[88] thus preceding the attendance by Dr O'Donohoe.

189) Dr O'Donohoe reports that he took over bagging from Dr Malik and then noted that Lucy's pupils were dilated and fixed. The timing of this important clinical sign is not recorded in the records but Dr O' Donohoe 's report to the Trust internal review[89] states it was when he took over bagging from Dr Malik. And (in a probably retrospective entry) inserts the time of 0330 hours. He also stated that he took the second blood sample.

190) Dr Auterson's note to the internal review (of 20/4/2000) states he was called at approximately 03:40 and arrived shortly after 03:50 and took over ventilation from Dr O'Donohoe when he noted the pupils were fixed and dilated and unresponsive to light.

[84] WS-279/1 Q30(a)P30

[85] 015-028-136

[86] 013-026

[87] 013-028-103

[88] 013-027-100

[89] 033-102-293

He intubated Lucy with some difficulty experienced in accessing equipment and then continued mechanical ventilation.

191) Information provided by nurse Thecla Jones to the review does not assist in clarifying the amount of fluid given[90] from the fluid balance chart. She reports that the entries made on a fluid balance chart were by her at 1 AM, 2 AM and 3 AM. *"The amounts of fluid is noted to the left of each box give a complete and accurate record of all intravenous fluid dispensed during that period. However I do note that the running total is indicated to the right of each box has not been tallied correctly ."*

192) Additional information which would not be available to Dr Quinn unless it had been given verbally by Mr Fee was as follows:

193) Nurse Swift account [91]provided for the internal review in appendix *states "I connected the IV line and I was instructed by Dr O'Donohoe to run the fluids at 100 ml /hr until Lucy has passed urine. In the presence of Dr O'Donohoe, Dr Malik, myself , Mrs Crawford and Lucy I connected the line to Lucy's (indecipherable) and took Lucy and Mrs Crawford to cubicle six medical side. Lucy vomited +++ bile coloured fluid she remained sleepy but her temperature settled. Mrs Crawford and Lucy sister then came into the cubicle. This was approximately 10:45 PM and after this point I had no direct contact with Lucy.".*

194) However this account suggests the IV fluid was running from at least 10:45 PM. The vomit at 2245 hr mentioned in this report was not recorded on the fluid balance chart.

195) Later information from Dr Donohoe 's report to the review [92]indicates that the blood sample showing the low sodium must have been taken whilst the saline was running because the sample was taken by him when he attended. It was received in the laboratory at 0357 [93]witness statement of biochemistry technician.

196) In his report for the review Dr O'Donohoe [94] stated that he gave 100 mls per hour for the first hour to cover the possibility the cannula might not last very long. He then intended to give 30 mls per hour until Lucy passed urine.

[90] 033-102-320

[91] 033-102-290

[92] 033-102-292

[93] 115-043-001

[94] Ref 033-102-293

197) Information available to Dr Quinn from the case records

198) **In summary**:

- On admission Lucy was dehydrated to a moderate degree.
- Lucy was given 100 ml/hour IV of Solution 18 up to 0300 from either 22:30 hours or 23:00 hours- probably the former.
- The total volume given of Solution 18 was between 400-500 ml
- Saline 250-500 ml was given IV by 04:00 hours and started before Dr O'Donohoe arrived and from 03:00 hours or thereabouts
- Dr O'Donohoe arrived at around 03:15 to 03:20 [95]
- The first blood sodium was reported before the IV infusion was set up at 20:50 hours and was normal 137 mml/l
- The second blood sodium was taken after the respiratory arrest and showed a fall to 127 mmol/l
- The second set of electrolytes were consistent with haemodilution
- Lucy was not in established circulatory collapse on admission nor at the time of the 03:00 respiratory arrest.
- The anaesthetist arrived later than Dr O' Donohoe

199) COMMENT ON DR QUINN'S REPORT

200) Comment on the notes of the meeting 21 June 2000

201) Dr Quinn expresses his view about the degree of dehydration rating this as moderate. He does point out that the fluid administered was of unusually high volume. But he mentions this in the context of resuscitation without drawing attention to the inappropriate use of number 18 solution if used for resuscitation purposes.

202) "The fluid replacement 4 hours provided was greater than normal but not grossly excessive."

203) "Dr Quinn does not feel that the extra fluids caused the brain problem"

204) *Dr Quinn notes that further fluids had been given "after the resuscitation- 250 mL normal saline"* and his opinion that the "*choice of fluid was reasonable but volume high".*

[95] Ref 027-010-024

205) (The reference made by Dr Quinn to resuscitation here probably refers to the respiratory resuscitation at 03:15 hours**.**)

206) There was discussion of the autopsy report of bronchopneumonia and it appears Dr Quinn was informed of the final post-mortem report. Dr Kelly in explains that he had *"Received post-mortem report dated 13th of June 2000 via Mr Fee"* [96]*,* before Dr O'Donohoe received his copy on 23 June. Dr Kelly reports he received another copy via Dr O'Donohoe on 26/6/2000. Comment : It is not clear how Mr Fee obtained the autopsy report and confirms that he had a copy but does not explain how it was obtained. [97]

207) Comment on the written report 22 June 2000

208) The key points are in this paragraph :

209) "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 3.00 a.m. when she had the possible seizure she got a total of 550mIs. This would include 150 mls oral and 400 mls i.v. as the intravenous drip was running at 100 mls per hour over a 4 hour period. Calculating the amounts over that period of time this would be about 80mIs/hr. I have calculated the rates of fluid requirements. If she was not dehydrated she would have required 45 mls /hour. If she was 5% dehydrated it would have worked out at 60 ml /hour and 10% dehydration works out at 80 mls/hour. I would therefore be surprised if those volumes of fluid could have produced gross cerebral oedema causing coning."

210) This information given indicates to the Trust that the use of N 18 solution, even in the high volume used, was not a significant issue in Lucy's deterioration, cerebral oedema and death. No emphasis is given to the wrong choice of fluid for Lucy's condition used in the volume and rate given nor to its likely contribution to the hyponatraemia which developed.

211) <u>Dr Quinn response to brief point [1] : The significance of the type and volume of fluid administered</u>

212) In paragraph 9 of his report Dr Quinn reported that treatment was given with Solution 18 *"which would be appropriate".* He calculated volume of fluids over the 7 hour period between admission and 3.00 a.m. when Lucy had the possible seizure.

213) The volume used by Dr Quinn to assess the appropriateness of the regime used was 80 mls per hour which equates to 210 mls per kilogram of body weight per day. The treatment for 10% dehydration in Lucy who weighed 9.14 kg would have been 200 mls per kilogram per day: 75-76 ml /hour. Yet there was no suggestion by Dr Quinn that Lucy had severe (10%) dehydration. In my opinion Dr Quinn is wrong in calculating the

[96] WS-290/1 P6

[97] WS 287/2 (Q6)

fluid given divided by the entire length of stay before the seizure. Rather it should have been calculated from the time of start of intravenous infusion and the volume prescribed then should take account of any preceding deficit including a delay in setting up an intravenous infusion which in Lucy was about 2 ½ hours.

214) There is a small error made by Dr Quinn in the calculation of the fluid requirement if not dehydrated when he estimates need at 45 ml/hr. Lucy was 9.14 kg, daily maintenance requirement thus is 914 ml = 38 ml/hour. His other estimates are rounded up and correct.

215) The information available in the case records to Dr Quinn was sufficient for him to be able to determine that the volume given IV to Lucy by 03:00 (when she experienced her seizure) had been 400 or 450 mls (or even 500 ml) all with 0.18% saline. And at a rate of 100 ml per hour which Dr Quinn had noted. Yet he erroneously concludes this volume was not grossly excessive and the fluid used was appropriate.

216) It is arguable that the overall volume given was not grossly excessive if the maximal therapy option was used as proposed by Dr Stewart which included a 20ml/kg bolus of normal saline. But by guidance of the time 40% should have been normal saline and 60% should have been 0.45% and , not as No18 solution.

217) But Solution 18 should not have been given at the rate of 100 ml/hour over 4 hours because at that rate and volume Solution 18 is not appropriate. Dr Quinn stated that the volume taken over the seven hour period "*appears reasonable*." It was not appropriate for treatment of Lucy's condition as evident from the case records and Dr Quinn acknowledges this. [98]

218) The fluid balance records that a further volume of 0.9% saline 250 mls was given IV between 03:00 and 04:00 hours and Dr Malik's record was of 500ml given. Dr Quinn comments "*If 500 ml* (of saline) *was given this may have affected the level of cerebral oedema experienced at post-mortem."* The case records contain information about the saline and even if 250ml only had been given this is clearly an excess volume with the same potential effect.

219) Although Dr Quinn raises a question about an excessive amount of fluid used at the time of the respiratory resuscitation at 0315 when saline was given he does not take full account of the high volume given before as No 18 solution.

220) The nursing record makes it clear that nurses were giving 100 mls an hour until urine was passed. Lucy had passed urine at either 8pm see fluid balance chart [99]which also records nappy was damp at 11 pm or 9pm nurse record[100] and a urine sample was

[98] WS-279/1 Q23 (c);(d);(e)

[99] 027-019-062

[100] 027-017-058

sent to the laboratory received at 21.03h[101] Dr Quinn does not remark on the damp nappy at 11 PM/23:00 hours after the infusion was running.

221) Dr Quinn points out that no prescription was written for the fluid used. This is the case both when the saline infusion was first set up and the earlier prescription written for the No. 18 solution at 2230 / 2330 hours on 12/4/2000 which simply records *"SOL 18 IV"* (signed Malik and Swift) [102].

222) Dr Quinn makes no comment on the substandard observation recording. Lucy's observation record only notes the (elevated) temperature at 1930,2030,2230,2330 hours but does not record the pulse rate until 03:15 when it was 163 (outside range).

223) **In summary it was evident from the records that by the time of the seizure Lucy had been given too high a volume of the wrong fluid for Lucy's condition. As fluid management is a fundamental part of consultant general paediatric expertise Dr Quinn should have recognised this and emphasised this in his report.**

224) Dr Quinn response to brief point [2]: The likely cause of the cerebral oedema and [3] The likely cause of the change in the electrolyte balance i.e. was it likely to be caused by the type of fluids, the volume of fluids used, the diarrhoea or other factors.

225) Dr Quinn had been asked to consider factors related to cerebral oedema.

226) In his written report, Dr Quinn does not set out any explanation of causation of the cerebral oedema. He confined his comments to :Para 9 : *I would therefore be surprised if those volumes of fluid could have produced gross cerebral oedema causing coning.* And Para 14 : *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.*

227) Dr Quinn makes no reference as a potential cause of the cerebral oedema to the rate of fall of the blood sodium, the possibility of a syndrome of inappropriate ADH or the contribution which a high volume of low solute fluid infused intravenously may have made.

228) Despite these deficiencies Dr Quinn makes a number of relevant points in his witness statements of November 2012 :

229) He reviews his clinical experience over 30 years and knowledge of fluid management during training before taking up consultant appointment and subsequently over many years as a consultant paediatrician. Dr Quinn states he refers to text books including Nelson and Forfar & Arneil and other guidance on fluid management. He

[101] 027-011-028

[102] 027-019-063

indicates (witness statement questions 2-4) his knowledge of hyponatraemia and inappropriate ADH secretion amongst other causes. And, his awareness that fluid overload can cause cerebral oedema and that in certain circumstances dilute fluid can cause it. [103]

230) Dr Quinn comments that the note made of the meeting with Dr Kelly and Mr Fee 21st of June 2000 does not accurately record what was discussed in that:

231) "Appears, to the best of my recollection, to be an accurate summary of what was discussed save for the following points:

232) I note that this document states that Lucy's urea was 9. In fact from the clinical records the urea was 9.9. I am unable to recall whether a reference was mistakenly made to a urea of 9 at the meeting or whether this was mis-recorded

233) The document states- "Dr Quinn does not feel that the extra fluids caused the brain problem". I do not consider that this is an accurate summary of my views, which was that I did not consider the amount of fluid that was recorded as having been administered before 3 AM was sufficient to cause such a degree of cerebral oedema as to lead to coning.

234) The document refers to 250 ml saline being administered after the resuscitation. I have no recollection of being informed of this figure.

235) And also that in his recollection of the discussion held at that meeting in respect of possible causes of cerebral oedema he states : :

- "*I did discuss the possible causes of cerebral oedema. As I recall this included:*
- *1 the use of N/5 (0.18%) saline and the volume given*
- *2 the possibility of hypoxia-at the time of the fit/collapse.*
- *3 the possible large volume of N saline given (0.9%)*
- *4 The efficiency of the resuscitation.*
- *5 the possible apnoea, and therefore hypoxia as a result of the rectal diazepam.*[104]

236) But Dr Quinn does not challenge the accuracy of the portion of the note of 21 June 2000 meeting with Dr Kelly and Mr Fee which recorded his view that " *Fluid issue: Choice of fluid correct; Resuscitation volume higher than normal."*

[103] WS-279/1 Q23(g) & (h) P25.

[104] WS-279/1 Q9 (d) P12 & Q9(g) P 14

237) Dr Quinn explaining his view about the fluids *"I felt this total volume (550 mL) given over the recorded period of time should not have been sufficient to provide such a degree of cerebral oedema that Lucy coned and had irreversible brain damage."*[105]

238) Dr Quinn explains why he would have been surprised if the volume of fluids received by Lucy could have produced gross cerebral oedema causing coning. Based on clinical experience of administration of fluid to children over the years. "*I did not feel the volume given over the timescale should have so rapidly resulted in a gross cerebral oedema."*[106]

239) Asked whether any consideration had been given to the intravenous infusion of solution 18 at a rate of 100 mL/hour having contributed to gross cerebral oedema he responded *"I consider the solution 18 administration could have contributed to the cerebral oedema but it cannot be considered in isolation. All of the fluids given to Lucy could have contributed to the cerebral oedema including whatever proportion of 500 mL of N saline was given at the time of the collapse around 3 AM."*[107]

His later view about the about what had caused the drop in sodium was :

- *"Consideration was given to the use of N/5 saline at around 100 mL per hour for 4 hours. I was also specifically asked by Mr Fee what part the diarrhoea could have had as a cause of the sodium loss. I considered inappropriate ADH as the cause of the decreased sodium. My conclusion was that all 3 could have contributed."*

- *"I had been aware of rapidly falling serum sodium being a risk factor for cerebral oedema since my early paediatric career, particularly in relation to hypernatremia dehydration treatment."*[108]

240) Dr Quinn also confirms here that he was aware of the result showing a sodium of 127 mmol per litre taken in the early hours of 13th of April 2000 but would not be able to see from the case records when this had been taken and its relationship to the normal saline infusion.

241) COMMENT on Dr Quinn's response to the question posed him by the Trust on causation of cerebral oedema.

242) The omission in his report of proper consideration of the relevant mechanisms in generation of cerebral oedema in Lucy is a major shortcoming. Nevertheless from the Trust's point of view, he did not provide a satisfactory explanation for the cerebral

[105] WS-279/1Q9(q) P15

[106] WS-279/1 Q 23(i)P25

[107] WS-279/1 Q23(l)P26

[108] WS-279/1 Q25(b) P27 & Q25(c) P27

oedema when he reported in his report Paragraph 14 that :" *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."*

243) In my opinion, this should have been followed up by the Trust as it was an indication for further investigation and assessment. It was relevant to consider whether the seizure was associated with imminent coning-if so then the high volume low solute fluid given by 03:00 would be implicated. If on the other hand, the fatal coning was later than 03:00 i.e. at the respiratory arrest some 15-20 minutes later, then the volume given up to 03:00 would be compounded by the additional volume of normal saline which started at around 03:00. It is at least arguable that the additional volume given a rate of 250-500 ml /hour of saline triggered the coning which was manifest as the pupil fixation seemingly noted for the first time by Dr O'Donohoe at the time he took over bagging from Dr Malik. Dr Malik does not record the time in the case records but Dr O' Donohoe inserts a note (possibly retrospectively) that it was present at 3:30 hour. There was thus sufficient information on the case records for Dr Quinn to conclude that the volumes used were excessive and he had raised questions about the volume of saline used in the resuscitation. Pupil fixation is an important signal of cerebral oedema and thus was of great importance to determine the time it was noted and the relationship to the fluid volume given.

244) Given that the death occurred from cerebral oedema and associated coning the assessment of the contribution of the fluid type and volume became critical. Dr Quinn does not fully address this point because he did not relate the excessive volume of low solute fluid and the observed hyponatraemia and other features of haemodilution with the brain oedema. These factors can be causative and it is arguable that he should have concluded this or at least raised suspicion.

245) Dr Quinn's report was worded in a way which was potentially misleading about the appropriateness of the fluid regime used. Dr Quinn now expands his views in his November 2012 witness statement and indicates that he had mentioned the role which the regime may have played in causation of the cerebral oedema. Thus at the time he provided potentially incorrectly reassuring advice to the Trust at least in the written report.

246) Dr Quinn failed properly to evaluate the information from the records although I can see these were confusing and do require a degree of interpretation.

247) He did not offer an assessment of the degree of dehydration in his written report although this is to an extent implicit in the calculations he made.

248) Dr Quinn did not suggest communication with RBHSC to determine other clinicians' views on the problems encountered in Lucy. On the other hand his report was only to be based on a case note review. He should, in my view, at least have suggested in his written report that this step should be taken.

249) I understand from his statements made later that Dr Quinn had undertaken this review within a framework of limitations and constraints. These were not set out in the letter sent to him by Mr Fee nor by Dr Quinn in his report. He should have included these constraints as an introduction to his report and also that he was not producing a report fully in response to the brief given to him.

250) Dr Quinn has later emphasised that his report was intended to be a verbal one based only on a case note review (rather similar to the case note review and verbal report provided in the case of Claire Roberts by Prof Young in Belfast in 2004). Also that he had advised that the Trust carry out further checks on the volumes given.

251) Dr Quinn states later that he had advised the Trust that additional specialist reports should be obtained. If this is the case, it is arguable that he should not have provided a written report or when doing should have included this in his report. Dr Quinn states that had had advised Mr Mills Chief Executive to do so but there is no record of this.: *"As far as I can remember, this recommendation was made to Mr Mills by telephone. To the best of my recollection I had 2 telephone calls with Mr Mills and I believe that the recommendation with regard to obtaining a consultant paediatrician from outside the Western board area was made during the second call. I am unable, however, to recollect the dates of either of those telephone calls."*[109]

252) Given the circumstances in which he interpreted that a case note review was all that was being requested, it was not inappropriate that he chose not to interview staff himself or to interview the parents. These latter two steps were appropriate for a more formal Trust review. It is not evident that Dr Moira Stewart took these steps when carrying out her review in 2001 nor that Dr Jenkins did so when preparing his medico legal report in 2002. The responsibility for doing this, in my view, lay with the Trust process.

253) Dr Quinn made no remark on the incorrect entry in the case records made by Dr O'Donohoe reporting the fluid used to Dr Crean.

254) Dr Quinn's warnings to the Trust

255) Despite his report's shortcomings Dr Quinn had advised the Trust that:

- He was not confident about the cause of the cerebral oedema
- The fluid volumes used were high
- He was not clear about how much normal saline had been given at the time of the arrest.

[109] WS-279/1 Q8(i) P11

256) The Trust should have taken further action in the form of setting up further investigation process, seeking an opinion from another expert review. As the Trust expected the Coroner to be involved (as did Dr Quinn from his 2012 witness statement) they should also have taken steps to inform the Coroner of Dr Quinn's views and also the findings of the Trust review report to assist the Coroner in his enquiries.

257) THE REVIEW REPORT

258) The draft review report was enclosed in a letter from Mr Fee to Dr Anderson on 5 July 2000 inviting him to comment and/or amend it.[110] Mr Fee suggests to Dr Anderson that a meeting be arranged with the family to be attended by Dr Anderson, Dr O'Donohoe and Sister Traynor. He reports that a meeting had taken place between Lucy's parents and Dr Hanrahan at RBHSC and that the autopsy report had been shared with them by Dr O'Hara (Royal Trust pathologist) and Mr Stanley Millar from the WHSSC in a meeting on 16 June 2000. It is not evident that Mr Fee determined what was stated at that meeting nor how he knew that meeting had taken place.

259) Dr Anderson replied to Mr Fee on 17 July 2000[111] commenting on the report. He does not appear to propose to alter the wording of the draft report. The final version of the review report is dated 31 July 2000. Dr Anderson points out he had not seen the post-mortem report and wrote

"I found that the report by Dr Quinn, whilst being helpful in the sense that it ruled out any obvious mismanagement on the part of our medical/nursing staff at the hospital, was also evidence of the fact that there was no clearly obvious explanation for the child's sudden deterioration"

Lessons to be learnt

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

The importance for standard protocols to be ready available in the ward against which treatment can be compared

"There was a mistake in the calculation of the on-going cumulative fluid which the patient received……..

recommendations

"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

[110] Ref: 034-029-082

[111] Ref: 033-102-262

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 showed them the measures that have been taken to analyse the care of Lucy's admission."

260) **Dr Anderson's Role [112]and my comments**

261) Dr Anderson's was Women and Children's clinical director and reported to Mr Fee for management and administration, and to Dr Kelly for professional staff personnel matters. He held meetings monthly with Mr Fee, Mrs Millar clinical services manager for paediatrics and Dr Halahkoon, the Trust lead consultant paediatrician.

262) Dr Anderson's contribution to the review seems to have been limited to writing his recommendations. He did not meet Dr Quinn. Dr Anderson states that Mr Fee wrote to Mr Mills incorporating Dr Anderson's recommendations but there is no copy in my papers. Dr Anderson stated that he *"compiled information and considered matters for the purpose of the report "*which implies he had taken an analytical approach but this is not evident. Rather he focused on process issues only. Dr Anderson states he had no further involvement after completion of the review report and took no further action or initiative although he received updates in the regular monthly meeting with Mr Fee and discussed Lucy's care informally with the lead paediatric consultant Dr Halakahoon.

263) Dr Anderson's aim was to determine if there had been any obvious mis-management on the part of the staff and to identify any lessons to be learnt and he stated there was no formal structured process within the Trust at the time for investigating adverse events. Comment : Yet "The Procedure for Acute Hospital Service Directorate 1998" includes a flow chart setting out processes including feeding back to staff. [113]and there was a special incident form in use in the directorate which suggests a process was in place. He reports that Mrs Elspeth Millar set up formal training for the nursing staff (but Mr Fee was seemingly unaware because he did not report this in his police interview).[114]

264) MY OPINION ON WHAT SHOULD HAVE BEEN DONE DURING AND AFTER THE REVIEW

265) Aim of the review. It was conducted to determine if

[112] Ref:116-038

[113] 319-041-002

[114] WS 291/1 Q45 P16

266) *There is any connection between the Trust's actions, omission and treatments and the progression and outcome of Lucy's condition. And whether there was a need for change in the care of patients.*

267) **What should have been done (and was not)**

268) When medical and nursing staff were asked to provide reports, their brief should have included a request for them to report any aspects of Lucy's care which concerned them. The requests made to the nursing staff were in writing and quite specific. They were being asked for the facts and two were asked for, or at least offered , their opinion. In contrast Doctors O'Donohoe, Malik, and Auterson were not invited in writing to contribute to the review process and specific points sought were not set out. This has been a major flaw because Dr Auterson later asserts that he was concerned that the fluid administered had been too much and the wrong type[115]. Dr O'Donohoe refers to a report he made in August 2003 to Dr Kelly [116] in which he raised a question that the 500ml saline was almost complete by the time Dr O'Donohoe had arrived. If this information was available to the reviews or reported to Dr Quinn it would have been evident that a grossly excessive rate of saline was used. Mr Fee and Dr Anderson should have interviewed Dr O'Donohoe or sought more information from him at the time of the July 2000 review. [117]

269) Dr O'Donohoe has stated that Dr Hanrahan told him (presumably on the telephone) on the afternoon 14 April 2000 that he –Dr Hanrahan- had notified the Coroner who had agreed that a hospital PM be carried out and that an Inquest was not being considered. Dr O' Donohoe is unable to recall whether he told anyone in the Erne Trust of this. [118] If he had been asked to discuss his report this might have come to light.

270) An analysis of responses should have been carried out to reconcile differences or omissions.

271) A summary chronology should have been included. There remained missing information particularly in relation to the timings and the amounts of fluid given. These were not resolved at the time when memories were fresh which is a failure of this review process. One example is the inadequate information relating to the fluid balance chart and its interpretation. At the very least one would have expected a tabulation of the fluid given.

[115] Ref:013-025-094 and WS-274/1 pages 4 and 5

[116] 047-53-148

[117] WS 278/1 Q7 10 (b) ii

[118] WS 278/1 Q3 & Q 10 (b) ii

272) Interviews should have been conducted with the medical and nursing staff involved as part of the process.

273) Communication with the regional centre was identified as a particular issue during the review. Although the Trust received the autopsy reports from RBHSC, they did not receive a discharge summary which would have been expected in a child transferred from the Erne hospital, especially one who had died. As a minimum this omission should have been corrected by approaching RBHSC.

274) Mr Fee noted that Sister Traynor had expressed her view that the regime used was not a problem but was not asked to produce a written report nor shown this note with which she disagrees.[119]. This was an opportunity to clarify the exact volumes that had been given.

275) When Dr Quinn's report was received it should have been shared with the medical and nursing staff seeking their comments. Equally Dr Quinn should have been shown a copy of the review and asked if it altered his views. He did not see the report from Dr Auterson. If it had been made available to Dr Quinn it might have helped him in his conclusions in respect of the fluid administered (high rate only appropriate to circulatory resuscitation). Dr Auterson did not receive a copy of the review report (or annexes) and was thus not able to comment on or correct Dr Quinn's account.

276) The information recorded in the notes given on the telephone on 14 April by Dr O'Donohoe to Dr Crean was wrong. Dr O'Donohoe did not report to Dr Crean, nor to the internal review the volume of normal saline or the total volume given of Solution No.18. The internal review did not identify this or follow it up.

277) The review reported that the nursing staff understood the instructions for Dr O'Donohoe to be to give 100 ml per hour until Lucy passed urine. And that the nurses considered this *"was standard practice at the time"*. Lucy had a damp nappy recorded at 11pm and the continued high rate should have been stopped then. This point was not raised in the review

278) To assist in interpreting the low sodium the timing of the blood sample should have been documented either from Dr O'Donohoe or from the laboratory technician who processed the sample or the nursing staff who would have been involved in taking it to the laboratory. This information was available (received in the lab at 0357hr) and was supplied later in 2005 in a police witness statement.[120]

279) The time of return of the result and the time at which the consultant anaesthetist attended should have been documented.

[119] WS-310/1

[120] Ref: 115-043-001

280) Dr O'Donohoe should have been asked to identify the time precisely when the pupil fixation was noted.

281) The review process did not document the concerns raised by Dr Asghar Staff Paediatrician on fluid management after he had reviewed the clinical records..

282) The Consultant Anaesthetist expressed concern about the lack of a paediatric ventilator and this has not been recorded as a requirement for action. There is no attention given to the need of the hospital staff to accompany Lucy to the regional centre. By this time-2000-a regional retrieval team for paediatric intensive care should have been in place and this should have been identified as a shortcoming if such an arrangement was not in place.

283) A final report should only have been assembled after these steps had taken place.

284) Review findings about Lucy's cause of death.

285) Dr Anderson had noted *"…there was no clearly obvious explanation for the child's sudden deterioration".*

286) The review findings stated [121] *"neither the post-mortem 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 around 2:55 AM on 13th of April 2000 or why cerebral oedema was present on examination at post-mortem".*

287) **Recommendations and what was done / not done** .

288) There were a number of recommendations made in the review. These included need for :

a. improvement documentation of prescribed orders;

b. "standard protocols in the Ward against which treatment can be compared"

c. A team meeting with staff involved in Lucy's care for "*discussion of report/findings"*

d. A meeting with the family *"to appraise them of all 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 are being taken to analyse the care of Lucy's admission."*

[121] 033-102-265

289) As the Clinical Director Women's and Children's Services, Dr Anderson should have conducted the meeting with the team, ensured that the meeting with the parents was set up (and attended it), that protocols were set up, that documentation was improved and set up audits to monitoring change over the ensuing few months. Some of these tasks could have been delegated with a lead member of staff identified to implement them. Training of medical and nursing staff on prescriptions and documentation and use of Ward guidance should have taken place, Mr Fee reports[122] that guidance on fluid prescription was issued but was not aware of any actions taken in respect of nursing training despite the fact that he was the lead nurse for the Trust although Mrs Millar manager had taken some steps to do this [123].

290) In the final portion of his letter to Mr Fee[124] , Dr Anderson suggests that a joint meeting should be held with the team members and also with the family but he did not respond to the request in Mr Fee's letter [125]enclosing the review report that he arrange this.

291) The parents should have been told that a review was in hand and an approach made to them to share the findings.

292) Given the seriousness of the event, it was the responsibility of the Trust senior management to ensure that these processes were in hand and to have requested reports. Although governance in general was in its infancy in 2000, clinical audit was well embedded and there was much guidance available on how to complete the "audit cycle" and report to senior management/ directorates (Audit Annex F).

293) No steps were proposed to improve the identified poor communication with the regional centre.

294) <u>SUBSEQUENT ACTIONS BY ERNE HOSPITAL TRUST REGARDING LUCY'S DEATH</u>

295) From July 2000 no further action appears to have been taken with the review report or its recommendations. Lucy's death remained unexplained even if the Trust had taken reassurance from Dr Quinn's report relating to the fluid regime. Mr Fee recalls that the report was given to Mr Mills Chief Executive. Subsequently Mr Fee was not involved in any discussions with regard to the findings of the review to the extent that they concerned the cause of the deterioration and death of Lucy. Mr Fee explains that he expected that the death would have been reported to the Coroner by RBHSC.

[122] 116-034

[123] 116-039

[124] Ref:033-102-265

[125] 033-102-261

Subsequently Mr Fee states that clinical governance arrangements were not established until after the adoption of *Best Practice Care in Northern Ireland* in 2002.[126]

296) The review was sent to the senior management team-Chief Executive and Medical Director (Mr Fee was already part of the team as the lead nurse for the Trust). There is no record of the response to the review by the Medical Director or Chief Executive. No formal action appears to have been taken within the Trust management as a result of the review despite the fact that the cause of death had not been identified other than sending it to Dr McConnell WHSSB with whom Dr Kelly had kept in touch through the course of the review.

297) Although one of the main purposes of conducting the review was to prepare for communication with the parents, no contact with them was initiated after the review and they were unaware that a review was taking place. Lucy's parents were concerned about the cause of death as certified and in July 2000, supported by the Chief Executive Officer of the Western Health and Social Services Council ("WHSSC"), an approach was made to the local Coroner but the Omagh Coroner, Ms Colhoun, is unable to recall this contact. [127]

298) In a letter dated 1 November 2000 to Ms B O'Rawe, Director of Corporate Affairs of Sperrin Lakeland Trust, Mr Crawford asks the Trust to provide a copy of the review[128] but they did not receive this until January 2001 as the Trust initially declined to share it with them. The copy sent[129] differed from the 31 July 2000 version. Ms O'Rawe (now Mrs Rippey) explains in that responsibility for strategy and policy in clinical governance was the remit of the medical director to whom she provided support. A clinical and social care governance strategy document was presented by Dr Kelly for Board endorsement in September 2000 (033-011). She was complaints manager and refers to the involvement of Mr Fee in dealing with complaints raised by parents on 29/9/2000 and that she had requested him on 27/11/2000, in view of parents declining a meeting, to set out the key findings of the review in a letter to send to Mr Crawford. This was sent on 10/1/2001 enclosing a version of the report.[130]

299) The Trust management was aware that no satisfactory explanation had been found for Lucy's death and thus Lucy's death remained unexplained. The Trust was aware of the diagnosis of cerebral oedema and bronchopneumonia and of the

[126] WS 287/1 Q 47; Q49;& WS 287/2

[127] WS-303/1

[128] Ref: 033-033-064

[129] Ref:015-028-132 to 015-028-136

[130] WS-309/1 Q 1(e); Q2

hyponatraemia found during the course of Lucy's treatment in the Erne hospital. At this stage they should have should have taken additional steps including:

- An approach to RBHSC to determine their views about Lucy's illness and cause of death.
- Submission of the review report from the Erne Trust to the Coroner's office in Belfast. The Trust senior management believed that Coroner's investigations were in process. In my opinion it was their responsibility to ensure that the Coroner was fully informed to assist the Coroner in his inquiry as there were concerns about the quality of care received by Lucy and the Trust had no explanation of her death.
- An external review by a clinician of the aspects of Lucy's care should have been set up in order to identify safety issues relating to continuing care of patients in the Erne hospital at the same time as information sharing with the Coroner.

300) What was needed for investigating Lucy's death was a focused detailed structured analysis and report on her care. If this been done- even with the deficiencies of the records- it was possible to conclude that an excessive volume of low solute fluid had been given and, that it was likely that this caused or contributed to the cerebral oedema and death. Furthermore, had it been set up quickly it would have been possible to have clarified the uncertainties which were present in the records and to which Dr Quinn had referred.

301) Dr Quinn states that he had advised the Trust in a telephone call with Mr Mills to obtain further expert paediatric opinion. Mr Fee however has no recollection of this it when it was discussed in the police interview.[131]. Dr McConnell also states that he advised a wider investigation but there is no documentation of this and Dr Kelly makes no reference to this advice. [132]

302) No attempt was made during the review process to obtain information from the treating clinicians in RBHSC and the Trust remained unaware of the death certificate diagnoses and the lack of intent to hold an inquest. On 10 August 2000 [133] Lucy's death was discussed in the mortality section of an RBHSC audit meeting chaired by Dr Taylor Consultant Paediatric Anaesthetist in the Paediatric Intensive Care Unit. There was no communication made by RBHSC to the referring hospital but had an approach been made by Erne hospital to RBHSC relevant information might have come to light. (Although it is possible that no useful information would have been available in view of the shortcomings in the RBHSC actions – see section on RBHSC below).

[131] 116-033-001

[132] WS 286/1 Q15

[133] Ref:061-038-123

303) Later actions

304) Rather than investigating the cause of Lucy's death, subsequent actions taken were focused on competency of Dr O'Donohoe after an approach from the staff grade paediatrician.

305) In September 2000 the RCPCH was asked to provide a review of Dr O' Donohoe's practice which included Lucy. A report was provided in April 2001 and another in August 2002. The Trust also received a report from Dr John Jenkins in 2002 as part of the Trust response to litigation initiated by the parents in 2001.

306) Although the Trust requested this from RCPCH in September 2000, there was a delay of 7 months before Dr Stewart provided her report in April 2001. Her report provided significant information about the relationship between the fluid management in Lucy and development of the cerebral oedema which led to her death. Had the RCPCH review been carried out sooner that information could have been acted on sooner. However in the event the Trust took no obvious action in 2001 in response to Dr Stewart's warnings.

307) In my opinion in 2001 the Trust should have reported the death to the Coroner in June when they received Dr Stewart's view that the fluid regime may have contributed to her death and especially later when the Trust learnt in October 2001 that an inquest was not planned.

308) From information received in the spring/summer of 2002 the Trust should also have made a referral to the Coroner.

309) Mr Fee states that he does not recall sight of copies of the RCPCH reports nor that from Dr Jenkins and does not recall any discussion relating to them.[134]

310) Other governance points

311) No records were made when the parents had a meeting with Dr O'Donohoe [135] on or about 5 May. One would have expected an entry in the case records and/or a letter written to the GP and to the parents to confirm the substance of the discussion. Dr O'Donohoe saw the parents before he had the final autopsy report. He did not have a copy of the discharge summary from RBHSC nor did he seek one.

312) A meeting is recorded 28 June 2000 between Dr Kelly and Dr O'Donohoe.[136] But no notes are available from that and it is not evident whether the review findings were shared with him.

[134] WS 287/1 P 20

[135] 030-010-018

[136] 033-102-261

313) Dr Moira Stewart's 26/4/2001 report on behalf of RCPCH. [137]

314) This report followed a request in a letter 14/9/2000 to RCPCH from the Medical Director for a review of professional conduct and competency issues associated with Dr O'Donohoe. RCPCH nominated Dr Stewart, R College Regional Advisor and Consultant Paediatrician/Senior Lecturer in Child Health at Queen's University Belfast.

315) Dr Stewart may have been involved in discussions before this because Mr Mills stated to the police that on 15/6/2000 he discussed a review with Dr Kelly who was enquiring into professional issues related to Dr O'Donohoe. He reported that he had already involved or engaged the regional adviser (presumably in paediatrics) and had discussions with Dr Halahakoon who was Dr O'Donohoe's colleague. Mr Mills also states that Dr Kelly had contacted the GMC helpline anonymously to get advice from them.[138] .

316) On 25.01.2001 [139]Dr Stewart wrote to Dr.Kelly referring to a letter to him of 9 November 2000 Dr Kelly from Patricia Hamilton (Secretary of Royal College of Paediatrics and Child Health) [140] on the 'external review' of the professional competence of Dr.Jarlath O'Donohoe and stated *"I think it would be helpful if I had an opportunity to go through the relevant case notes before meeting with the individuals involved … It may be necessary to ask a Paediatric Specialist for an opinion in one or more of the cases, should that case fall within the remit of a recognised sub-speciality".*

317) From the viewpoint of the Inquiry it is a matter of remark how long an interval passed before the RCPCH report was provided (7 months after approach was made by the Trust).

318) Lucy Crawford case was one amongst 4 others to be included in the review. Dr Stewart clarifies her role *"I was not asked to prepare a comprehensive medical report on any individual child but to comment on the overall management of the children by a general paediatrician, as documented in case notes provided by Sperrin Lakeland Trust . At the time I agreed to undertake the review, I was unaware of any details regarding diagnosis or outcome."* Dr Stewart emphasises her report was not commissioned or delivered as a medical report on Lucy's death and considered that *"further conclusions as to cause of death were more appropriate at inquest when all additional information could be considered*".[141]

[137] 036a-025-055

[138] 116-051-004

[139] 036a-015-030

[140] WS-290/1 P6

[141] WS-298/1 Q 6(d) P5;& Q10 (d)

319) Dr Stewart's report was based on a review of the case records, the post-mortem report and Dr Quinn's report and she held a telephone discussion with him. She concluded that her opinion on the management differed from that of Dr Quinn. She was under the impression that the Coroner was involved. [142]

320) She did not have sight of the internal Erne hospital review conducted in 2000. I provide detailed commentary on Dr Stewart's report in Annex A but here highlight the following relevant comments she made in regard to the fluid management of Lucy:

321) *"Given the symptoms and signs, and the prolonged capillary refill time (>2 secs), it would be appropriate to give an immediate fluid bolus of up to 20 ml/kg (N Saline, or less commonly, colloid) and then reassess.*

322) At 3.00 am, and after administration of 0.18% NaCl, the repeat sodium was 127, and potassium 2.5. Biochemical changes are often well tolerated and easily corrected with appropriate fluid replacement, although these results do show a change over a relatively short period of time

323) The fluid balance records between admission and the events at 3.00 am are incomplete. 0.18% saline was commenced at 10.30 pm, but the rate is not prescribed on the fluid balance sheet. My interpretation of the chart is that she received 100 mls/hr 0.18% saline until around 3.00 am when the adverse episode occurred.

324) The volume given, therefore, does not appear excessive. There is debate about the most appropriate fluid to use. **[this point appears to refer to the fluid to use in replacement and maintenance and not to resuscitation fluid]**

325) APLS guidelines: deficit should be replaced with normal saline and maintenance with 0.18% N saline.

326) *For convenience the 2 fluids are often combined and given initially as 0.45% NaCl in 5% dextrose, and the regimen altered on the basis of blood result. After the respiratory arrest at 3.l5am, the fluids were changed to N saline. The clinical notes state that 500 mls was given over the next hour.*

327) *there are deficiencies in the prescription and recording of volumes of fluids administered."*

328) <u>Comments on Dr Stewarts's 2001 report</u>

329) <u>Volume used in Lucy</u>

[142] WS-298/2 Q1

330) Dr Stewart emphasised in her report that normal saline should have been used to provide 20 mL/kilogram of the total volume given. In witness statements of November 2012, Dr Stewart explains her opinion on was it appropriate to treat Lucy with solution 18 at a rate of 100 mL/hour between 10:30 PM and 3 AM?:

331) "this was a clumsy attempt to reconcile volume of fluids Lucy received from 10:30 PM to 3 AM with recommendations for administration of IV fluids in a child presenting with shock and dehydration. Solution 18 would have been an inappropriate solution according to accepted practice at this time. Initial treatment with a bolus of normal saline at 20 mL/kilogram given over short period of time would have been appropriate treatment. Replacement dehydration (7.5%)-750 mls(estimated dehydration) Replacement fluid-100 mL/kilogram over 24 hours."

332) Dr Stewart emphasises that it is not possible to work out exactly what the intravenous fluid input was. *" As I recorded earlier, I did not find it possible to work out exactly what her intravenous fluid input was during the time from insertion of cannula until sudden deterioration. If she had been given a fluid bolus of 20 mL/kilogram to correct shock she would have received 180-190 mL fluid over a short period of time. Over the next four hours 11 PM-3 AM if she had received additional 60-70 mL/kilogram (maintenance and deficit) she would have received a further 240-280 mL altogether making 440-480 mL in total."…."..but I do not believe the volume was the major problem-given her initial presentation"*

333) In her 2001 written report Dr Stewart did not address the volume of normal saline given and later explains that her report "*does not specifically refer to appropriateness of fluid given after respiratory arrest. The emphasis was on the events leading up to the respiratory arrest*. " And she explains this further *" following her acute deterioration and respiratory arrest, other consultants were involved in her management, and their care was outside remit of the report requested"*[143]

334) Dr Stewart also states:" *it would have been inappropriate to give a further fluid bolus of 500 mL normal saline to a child who has had a seizure-like episode, respiratory arrest, fixed dilated pupils, increased BP (144/113*)"[144]

335) **Comment :**In my opinion it would have been preferable for Dr Stewart to have stated more clearly in her written report that an excessive volume of solution 18 had been administered because all of the 400-450 mls volume given up to 03:00 hours was that fluid. It is left to the recipient of the report to interpret her opinion. In regard to the volume of saline used the case notes record that the normal saline was started at around 03:00 hours by the paediatric SHO working for Dr O'Donohoe and thus before

[143] WS-298/1 Q11 (c) P9; Q12 (b)P8; Q 13 (d) P 10

[144] WS-298/1 Question 13 (c) P10

other consultants were involved. Consequently it would have been reasonable for Dr Stewart to have included comment on this volume.

336) **<u>After Dr Stewarts's 2001 report was submitted on 26 April 2001 a meeting held was held on 21 June 2001 with Dr Kelly Medical Director.</u>**

337) Dr Kelly recorded the following questions and answers: [145]

"Q1. Was the delay to IV fluids significant? Was there sufficient attention to fluid balance?

Q2. Was it reasonable to push oral fluids in the first hours of admission?…

Q4. Should a urea of 9.9 given rise to major concerns. It corrected to 4.9 within hours.

Q5. Do you really think that the electrolyte changes caused the seizure?

A1 – Capillary refill time, raised urea and C02 level point to circulatory failure. IV fluids were indicated earlier. Overall amount of fluids once started not a major problem but the rate of change of electrolytes may have been responsible for the cerebral oedema. RVH ward guidelines would recommend N-saline not 1/5th normal as the replacement fluids.

Other issues – Was this child bagged with mask for ~ 1 hour (?anaesthetist involvement)"

The conclusion was:

"There is insufficient sub-optimal practice to justify referral to GMC … Monitor, develop further work on guidelines and protocols and link with paediatrics at Altnagelvin advised"

338) Dr Moira Stewart comments on these notes respecting the accuracy of what was recorded. *" I met Dr Kelly. He asked me questions, took notes but I do not have copies so answers are based on recall a conversation. I presume he has a copy of answers to the list of questions he put to me. This is a brief summary of a much longer conversation. I do remember him asking me if I had really thought the electrolyte disturbances had caused the seizure (Q5) and I said an unequivocal "yes". From recall, I then went on to elaborate on guidelines for type of fluid for replacement of dehydration and for treatment of "shock"….."*[146]

339) Dr Stewart corrects the reference to RVH guidelines and recalls she made reference to APLS guidelines.

340) <u>Electrolyte change and cerebral oedema</u>

[145] Ref: 036a-027-066

[146] WS 298/1 Q14 (a) P11

341) The only comment made in the written report on the electrolyte change (hyponatraemia) was: "*Biochemical changes are often well tolerated and easily corrected with appropriate fluid replacement, although these results do show a change over a relatively short period of time. "* This point was addressed further during the meeting with Dr Kelly.

342) Answering seeking clarification about Dr Stewart explaining her reporting that the overall amount of fluids used not being a "*a major problem*", states that it was extremely difficult to work out from the notes what volume of fluid had been prescribed. *"My opinion is that the volume of at most 400 mL given to a child with evidence of shock over a four hour period, including resuscitation, maintenance and replacement fluids would not usually be excessive-but that the exclusive use of hypotonic fluids i.e. solution 18 led to a rapid fall in sodium and resulted in acute deterioration around 3 AM or thereabouts."* And that at the time and reviewing the notes now she still find it impossible to determine actual volumes of fluid given due to lack of documentation. [147]

343) "… *I explained the guidelines in general use of children presenting with shock and/or dehydration, and that use of hypotonic solution 18 would not have been indicated as the sole infusion fluid. I was and am aware of the problems associated with abnormal electrolyte levels in children and in particular, rapid changes in values."*

344) Dr Stewart considered that Lucy required management appropriate for shock and refers to her report (036a-025-055) where she used the term " *a degree of shock*". The factors which Dr Stewart identified as suggesting shock were "*increased respiratory rate (40/per minute), heart rate (140/minute)-upper limit normal, prolonged capillary refill time (> 2 seconds), reduced CO2, lethargy. "* Thus she advised the Trust that Lucy should have been given up to 20 mL/kilogram of normal saline or colloid as an initial fluid bolus managing as for shock.

345) Dr Stewart refers to the notes of the meeting with Dr Kelly and Dr Stewart recalling that she stated unequivocally that electrolyte disturbances had caused the seizure.: " *from recall, I am fairly certain that I said that the change in electrolytes resulted from administration of solution 18 as Dr Kelly alludes to this in his note." [148]*It is clear from her comments in the statement that Dr Stewart expected a Coroner's inquest to be carried out.

346) In my comment on Dr Quinn's report I considered it is possible, even with regard to the confusing entries, to calculate from the case records that a fluid overload was given when the normal saline volume given at the collapse is taken into account. And it

[147] WS-298/1 Q14 (b)(i) P 11; Q14(b)(iii) P11

[148] *WS-298/1 Q14(b)(iv) P12; Q11 (a) P 8; Q14 (a) P11; Q 14 (b) (vii) P12*

is possible that the volume overload itself was an additional factor in the causation of the cerebral oedema combined with the hyponatraemia. Dr Stewart had advised that a 20 mL/kilogram bolus of saline should have been used at the start of the infusion and 0.45% thereafter. It would have been preferable for Dr Stewart to have provided a specific explanation of how the hyponatraemia and the rate of change in electrolytes could have resulted from high volume used of low solute number 18 solution. In my opinion she should have set out how high volume of low solute fluid together with the saline overload could all have combined to contribute to or cause cerebral oedema and to explain more clearly in her written report how the hyponatraemia was produced.

347) Nevertheless in my opinion Dr Stewart gave sufficient information to the Trust in 2001 to identify that the intravenous fluid treatment given to Lucy could have contributed to her death.

348) Dr Moira Stewart and Dr Andy Boon reported to the Erne Hospital on behalf of RCPCH in August 2002

349) The RCPCH carried out a second professional competency review of the practice of Dr. O'Donohoe, arising out of a request made by Dr. Kelly on 7th February 2002 [149] following upon further concerns which had been raised by Dr. Asghar [150]

350) Dr Moira Stewart was again involved together with Dr Andrew Boon Consultant Paediatrician from Reading both being nominated by the RCPCH. They produced a joint report within the context of the protocol for external clinical advisory team visits of the RCPCH which importantly in paragraph 3 states *"the Trust should inform the NCAA if an individual doctor is being investigated."*

351) In paragraph 8 *"the ownership and circulation of report is agreed. The report is owned by the requesting Trust . Any action taken, or not taken, as result is the responsibility of the Trust . The College does not get a copy but requests a summary of the conclusion of the visit. Neither the report or any of its contents are circulated beyond the agreed stakeholders."*

352) The report [151]included a number of children and Lucy. The reviewers note that their brief was to *"……. To look into professional concerns about the clinical competency and professional performance of Dr O'Donohoe. Although the terms of reference were to deal with professional matters, we were aware of other matters which had been raised and previously investigated by Sperrin Lakeland Trust ."*

[149] Ref: 036a-129-273

[150] 036a-032-073.

[151] 036a-150-309 to314

353) The review team refer to documents relating to professional competence review including copies of correspondence between the staff grade and Mr Mills and listed staff interviewed.

354) Report : In paragraph (iii) the report states

355) Lucy was a child admitted with vomiting requiring IV fluids and suffered a convulsion followed by a respiratory arrest and subsequently died of cerebral oedema in Belfast.

356) The prescription for the fluid therapy for LC was very poorly documented and it was not at all clear what fluid regime is being requested for this girl. With the benefit of hindsight there seems to be little doubt that this girl died from unrecognised hyponatraemia although at the time this was not so well recognised as at present."

357) In witness statements, (see footnotes) Dr Moira Stewart points out that the conclusions drawn in August 2002 were after a long discussion between Dr Boon and Dr Stewart. They found the documentation of events following admission to be very poor, difficult to follow and notes *"we had not been asked to prepare a medical report on Lucy's cause of death.".*

358) Explaining what hindsight had enabled the reviewers to reach a conclusion that Lucy died from unrecognised hyponatraemia Dr Stewart's explains *"Dr Boon and I had access to documents not available at time of initial report and the opportunity to talk to other members of the Sperrin Lakeland Trust. We discussed possible factors which may have contributed to Lucy's death but decided we could stand over this statement."*

359) A question in WS 298/1 Q 15 (d) referred to a document WS-298/3 P4 Para 5(iii) in which the 2002 external review concluding that : *"more attention to detail of the fluid therapy might possibly have avoided this girl's cerebral oedema and fatal outcome" .* This statement does not appear in the report submitted to the Trust. And this is explained by Dr Stewart later in WS-298/3 Q1 from her memory, Dr Boon and Dr Stewart discussed the draft report and took account of their knowledge that a medico legal case was under way and that they had not been asked to contribute to the process and therefore decided they should not exceed the remit of their review which was to examine the professional competency of an individual consultant. They concluded that prescribing administration of fluids fell short of accepted practice and altered the wording in their final report made to the Trust.

360) I highlight below the text which was removed from the final version.

361) *The prescription for the fluid therapy for LC was very poorly documented and it is not at all clear what fluid regime was being requested to this girl. With the benefit of hindsight there seems to be little doubt that this girl died from unrecognised hyponatraemia although at that time this was not so well recognised as at present.* ***More***

careful attention to detail of the fluid therapy might possibly have avoided this girl' s cerebral oedema and fatal outcome.

362) Comment: The explanation given by Dr Stewart for the change in this wording is understandable but it might have been considered as a point to raise verbally with the Trust given their knowledge of the inadequate report provided by Dr Quinn with which they were familiar.

363) Dr Stewart explains who had not recognised the hyponatraemia, *"At that time there was less awareness among general paediatricians in the UK of the potential harm associated with use of hypotonic solutions in acutely unwell children, due to a lowering of sodium levels. "* and *"From the late 1990s and more especially in the early 2000s, there have been an increasing number of published scientific papers which reported adverse outcomes associated with hyponatraemia in acutely unwell children."*[152]

364) Dr Stewart refers to the fact that she cited hyponatraemia in her 2001 report and in her meeting with Dr Kelly and had *"commented on changes in blood sodium over short periods of time (036a-025-057)".* [153] .[154]

365) Dr Stewart explains why she was unable to reach the conclusion that Lucy had died of hyponatraemia when she submitted her first report in April 2001. Dr Stewart points out that this was an examination of case notes in 4 children and not intended to be a medical report or an expert witness report on any of the 4 cases. *"In addition the lack of documentation in Erne hospital notes meant that clear conclusions as to the interplay of factors leading to her death was outside the scope of the initial report. Dr Boon and I were sent additional documentation, prior to our external review visit Erne hospital, when we met with other members of staff over and above that given to me at the time of the initial report."* [155]

366) Dr Stewart has no record or recall of the additional documentation received in 2002 to other than a written report from Dr Asghar. [156]

367) Dr Stewart was surprised that the Coroner had not been involved.

368) Comment : The conclusion drawn by Dr Boon and Stewart in 2002 as documented in the draft version of their report raises questions about what might have resulted from a wider external review been carried in the year 2000. It is possible that a

[152] WS 298/1 Q15 (a) P12; Q 5(b) P12/13; Q 15(c) (i)

[153] WS-298/1 Q10(d)

[154] WS 298/1 Q (d) P13

[155] WS 298/1Q15 (e) P13

[156] WS-298/2 Q2

similar conclusion could have been drawn. However in 2002 Drs Stewart and Boon had the benefit of the alerts issued in 2002 in Northern Ireland about use of number 18 solution and risk of hyponatraemia and more published reports were available compared with 2000.

369) ROLES OF SENIOR MANAGEMENT TEAM

370) Dr Kelly

371) Dr Kelly a consultant geriatrician was Medical Director of Sperrin Lakeland Trust and a Board Member [157]. His responsibilities are listed in his job description (1999).[158] and include responsibility for clinical governance.

372) Comment on the part played by Dr Kelly in the 2000 Review

373) Dr Kelly reports after he was approached by Dr O'Donohoe on 14 April 2000, he informed him that in view of the concerns Dr O'Donohoe had raised about potential misdiagnosis, or wrong use of drugs or fluids, he advised him that there would be a full review of the case and that it was likely that the case would be referred to the Coroner. Dr Kelly did not take any steps to assure himself that this in fact was in hand. He made a report to Dr McConnell at the WHSSB-the first occasion that he felt this need in his role as Medical Director (1 December 1999- 1 December 2003.) In Dr Kelly's view the purpose of the Trust review was to investigate events surrounding the death of Lucy and to establish if there had been failings in the Trust 's treatment or care or whether areas of practice required improvement within the paediatric Department. He was not involved in interviewing staff members and the review was being dealt with by Dr Anderson and Mr Fee. He met Dr Quinn only because Dr Anderson was on leave and he wished to be satisfied that there were no issues relating to medical incompetence because he had received Dr Asghar's concern about the fluid management. He also records that Dr Quinn expressed reluctance to be involved in complex litigation processes.

374) In the 2005 PSNI [159]interview Dr Kelly reported no action on his part to ensure that the recommendations of the review were put in place nor that he identified any need to seek further paediatric expert opinion on Lucy's death after receipt of Dr Quinn's report. He agreed with the Chief Executive to obtain expert paediatric advice on Dr O'Donohoe only in the context of assessment of competence/discipline matters raised by Dr Asghar.

[157] Ref: 116-043

[158] Ref:115-056-014

[159] 116-043

375) During the review on 15 May 2000 Dr Kelly wrote to Dr McConnell [160] and informed him that the fluid regime was probably irrelevant and the cause of death is still not clearly established. He reports that a full analysis of the investigation is to be conducted by Dr Anderson and Mr Fee and that Dr O'Donohoe had told parents that results of the review will be shared with them. Dr Kelly stated he will give more information to Dr McConnell in due course and asks for his comments.

376) In my opinion Dr Kelly was part of the senior management team receiving the review report and to an extent quality assuring it and checking that its recommendations were carried out. In the PSNI interview Dr Kelly describes the review as involving external paediatric opinion and that such external opinion was quite rare for the time and included interviews and analysis as well as a post-mortem report. He expresses his opinion on the quality of the review and describes it as *"a reasonably comprehensive review of the situation".* And "*extremely comprehensive".*

377) In my opinion, Dr Kelly should have noted the omission by the review team in interviewing the doctors involved and redressed this himself. He conducted an interview with Dr Malik following the letter of complaint from Dr Asghar in November as Dr Kelly stated that he did not wish to interview him during the course of the review itself.

378) Dr Kelly confirms that the review report was sent to the Chief Executive of the Trust but that it was not considered at Trust board level and that after the review Dr Kelly had no discussions about Lucy until 2001.[161]

379) Dr Kelly[162] had the expectation that there would be an Inquest. It was his understanding that the Coroner was aware of the case and was surprised when he was informed during the litigation process in October 2001 that one had not occurred or was planned. In 2000 he took no steps to confirm whether or not an inquest was to be held or the content of the death certificate. Mr Mills recalls that about the time Lucy died, he was informed by either Mr Fee or Dr Kelly that an inquest was to be held.[163] Dr Kelly had obtained a copy of the final autopsy report in June 2000 both from Mr Fee and Dr O Donohoe. This is a hospital post-mortem and makes no reference to this being conducted on behalf of the Coroner as part of the processes by the Coroner's office. He might have considered raising this as a question at the time.

380) Dr Kelly states that he had no contact with the Royal either the pathology department or the paediatric Department as he considered it inappropriate to do so given that he had set up a review but he must have known that the review did not do so.

[160] 036A-046-098

[161] WS 290/1 Q 22

[162] Ref:116-045-003

[163] WS 293/2

Mr Mills noted that Dr Kelly was contacting the Royal on 4/5/2000 seeking information about tests related to the autopsy *"Dr Kelly advised he was asking for report on tests carried out in connection with post-mortem" .*[164] But Dr Kelly does not mention this action.

381) In the PSNI statement Dr Kelly referred to meeting with Dr Halakahoon on 23 June 2000 to discuss issues raised by the " LC case" and he shared the results of the PM and Dr Quinn's review and highlighted fluid prescribing and resuscitation issues raised. Later he met with Sister Traynor. Notes were made [165].

382) Dr Kelly confirms Mr Fee's and Dr Anderson's reports that there was no structured approach to investigating an incident such as a death in hospital at the time in 2000. He points out that clinical and social care governance did not come into Northern Ireland until 2001/2. (This is probably referring to the *Best Practice/ Best Care* consultation document 2001) a report which refers to an earlier one October 2000 of DHSSPS(NI) *Confidence In The Future* requiring doctors to be involved in audit which refers to the need to record adverse events-paragraph 5.10 onwards). Comment : all doctors were expected to be involved in audit by their professional bodies and the GMC from mid 1990s.

383) The Trust reported to Dr O'Connell and Mr Millar (acting on behalf the parents) that actions are being taken in keeping with the Trust's governance policy on adverse events and referred by Mrs O'Rawe on 22/11/2000[166] [167] writing to Mr Crawford stating in respect of the clinical review that "*this process is one which has been introduced by the Trust in the last two years or so and is in the main undertaken where there has been a sudden unexpected death*". The clinical incident form completed on 14 April 2000 is set out within a framework related to the evaluation of perinatal problems. This is understandable because the culture of perinatal morbidity and mortality reviews was of very long standing and preceded many other aspects of clinical governance dating from the late 1960s at least. The Erne Trust clearly aspired to, and reports that, it has a clinical governance process which included this involved processing individual major incidents by setting up an investigation, recording the findings and lessons learnt on a data base (Datix Web) and informing the Chief Executive Office and the Head of Clinical Governance. A flow chart was provided dated 1998. [168]

384) Dr Kelly 's later actions.

[164] 030-010-018.

[165] 036a-008-013

[166] Ref:033-028-057

[167] 015-024-126

[168] 319-041-002

385) Dr Kelly reported that when the Trust received the agreement of the Royal College to conduct the review of Dr O'Donohoe that it was *only to be on the issue of professional competency*.[169]. This confirms the limited role expected of Dr Moira Stewart's approach in terms of investigating the cause of death in Lucy. In his November 2012 WS-290/1 page 6 Dr Kelly reports *"post-16.07.00 telephone contact with Dr Moira Stewart, regional College adviser in NI for the RCPCH to seek independent external assistance in assessing the competence and conduct of Dr O'Donohoe".*

386) Dr Stewart's 2001 report gave Dr Kelly sufficient information for him to have appreciated that the fluid volume given to Lucy was wrong in being all number 18 solution up to 03:00 hours although in her opinion the volume was not excessive if a bolus of saline had been given, and thus there was an excessive volume of low solute fluid given. Furthermore Dr Stewart identified that a rapid fall in the blood sodium could have led to the cerebral oedema. In June 2001 the Medical Director had sufficient information to implicate therapy mismanagement with the death and should have taken further action. In my opinion a formal investigation of the death should have been set up for clinical safety purposes. Dr Kelly believed that the Coroner was involved and thus should have informed him of the identified issues.

387) Dr Kelly indicates that he became aware that hypotonic fluids administered to Lucy may have contributed to the cause of her cerebral oedema in and around June 2001 following the possibility of hypotonic fluids being an issue raised in a meeting Dr Moira Stewart on 31st of May 2001 and the discussion which occurred at the medical directors group meeting on 18th of June 2001. And in this information was confirmed when he received Dr John Jenkins report in April 2002. [170]

388) Three weeks after the 1 June 2001 interview with Dr Stewart, Dr. Kelly wrote on 21 June 2001 to colleagues at Erne enclosing a copy of *Halberthal et al Lesson of the week: acute hyponatraemia in children admitted to hospital : retrospective analysis of factors contributing to its development and resolution.BMJ 2001;322:780-782* to inform them that he had been advised at a Medical Directors meeting that a child had recently died after developing severe hyponatraemia leading to seizure activity and coning[171] **.** He also reported that the RBHSC had changed its guidelines and was no longer using Solution No.18 post surgery or for rehydration in paediatric medicine. He asked his colleagues to review the Erne's practice with regard to fluids.

389) Given his recent discussion with Dr Stewart, arguably Dr Kelly could have concluded that Lucy's death may have been similarly caused. On 24/7/2001 Dr Kelly discussed the report from the RCPCCH with Mr Mills and refers to Mr Mills notes of this

[169] 116-043

[170] WS-290/2 Q5 (a), (b), (c),(d)

[171] Ref: 036a-055-141

meeting[172]. On 27 June 2001 Dr Kelly forwarded Dr Stewart's report and his meeting notes, (which provided a signal that there may have been fluid mismanagement in Lucy's case), to Dr McConnell and asked him for his comments[173]. Dr McConnell advised the Trust either then or when Dr Kelly met Dr McConnell on 8/10/2001.and has explained that he did not link the case of Raychel Ferguson.

390) On 26 June 2001 at the Sick Children's Liaison Group at Antrim hospital Dr Taylor RBHSC gave a presentation about hyponatraemia in use of hypotonic fluids in children.[174] If Trust staff had been aware of this presentation there would have been an opportunity at this time for the Trust to reflect more closely on the management of Lucy. Dr Taylor explains the function of this group established in 1999 and it was attended by a consultant paediatrician and anaesthetist responsible for children from the main hospitals in each board area. Dr Taylor confirms that the purpose of the group was to agree best practice guidelines for improving stabilisation and transfer of children to PICU and not intended as a forum for the review or investigation of child deaths. It is relevant to determine how Erne hospital was represented and how feedback took place.[175] This group offered a mechanism for dissemination of adverse events which might trigger new guidance.

391) Erne understanding about a Coroner's Inquest for Lucy

392) Dr Kelly in 2001 and 2002 believed that a Coroner's Inquest was to be held.

393) Dr Kelly believed when he received Dr Stewart's 2001 report that the Coroner's office was fully aware of the case and would be scheduling an Inquest. Dr Kelly in explains when he received Dr Jenkins report in early April 2002 he asked DLS to clarify when a Coroner's inquest was due to take place and what the reason was for the delay. He had been advised by the DLS team after an approach in July 2002 that a lengthy delay before an inquest was not unusual for a Coroner's inquest to take place 2-3 years after the death.[176]

394) Mr Mills reports that from 12 October 2001, Dr Kelly, Ms O'Rawe and Mr Fee were made aware that an Inquest was not planned. [177] &[178]

[172] WS-290/1 P8

[173] Ref: 036a-028-069

[174] 093-035

[175] WS-280/1 Q7

[176] WS 290/1 Q31 P23 (b) (v); Q34(a) ;Q35(e) & WS Q23(f) P 19;

[177] Q15 of 2012 WS 293/2.

[178] WS-293/1. Question 36. P19.

395) Dr Kelly states that he approached DLS on 15th of November 2001 to contact the Coroner regarding the inquest. And again at a scrutiny committee meeting on 12th of April 2002. But he also confirms that it was known in November 2001 by him that an inquest was not yet scheduled. But it was only in 2002 that they learned that no inquest was planned. [179]

396) Comment: this appears to explain why the Erne Trust was still expecting that a Coroner's inquest would be held and why Dr Kelly did not make a referral himself to the Coroner. It still leaves the question about why Dr Kelly did not consider it his responsibility to refer to the Coroner when he became aware in 2002 that an Inquest was not to be held. If this delay was expected, clinical governance/safety requirements would be such that in the interval a hospital should become aware of any lessons to be learned from the management of the case such as a death from gastroenteritis. Such lessons could have been provided from an external review. It is difficult to understand why Dr Kelly made later requests of DLS rather than checking with the Coroner's Office especially after October 2001 and arguably in view of the information provided by Mr Mills reporting that the Trust was aware on 12 October 2001 that no Inquest was planned, in my opinion should have referred to the Coroner at the latest in October 2001.[180]

397) <u>2002</u>

398) Dr Kelly reports that a second College review was requested because Dr Asghar and Dr Halahakoon lead consultant paediatrician had raised concerns about further cases. Following advice given by Dr McConnell the Trust requested a further review from RCPCH February 2002 [181].

399) <u>Erne Trust response to 2002 reports of Dr John Jenkins and Drs Boon and Stewart</u>

400) The Trust received a report from Dr Jenkins as part of the litigation process in March 2002. Dr Jenkins for the Trust[182] and Drs Boon and Stewart for RCPCH each raised concerns that hyponatraemia contributed to Lucy's deterioration and death. These reports followed the publication in March 2002 of the Northern Ireland warning on hyponatraemia and number 18 solution.

401) In the end on 1 February 2003 Mr Stanley Millar being aware of and making the linkage with Raychel Ferguson's death reported Lucy's death to the Coroner in Belfast.

[179] WS-290/2 Q6

[180] WS-293/1 Q 36. P19.

[181] 036a-129-273

[182] 013-011-038 and 039

This led to the inquest in 2004. It was only at this point that the Trust set up a root cause analysis.

402) **Role of Mr Mills (Trust Chief Executive)**

403) In his 2005 PSNI interview[183] Mr Mills reports he saw the issue to be one related to potential disciplinary matters rather than an investigation of the cause of death or as a serious unexpected clinical incident. He felt that at the time that seeking an external opinion was exceptional. He would have discussed his approach with the chairman of the Trust[184], and with Dr McConnell but there were no established codes of practice in handling such issues at the time. However he states that the Trust did recognise that Lucy's death was a serious adverse event and that "*she died and we did not know why*[185]".

404) Comment: Mr Mills relied upon the Trust review process and also upon Dr Stewart's report even though he acknowledges that it was inconclusive in the sense of identifying a number of reasons why Lucy might have died. He states that the Trust was receiving information which was pointing to the fact that the fluids may not have been an issue including from their external paediatrician and from the paediatrician nominated from the RCPCH and states that this paediatrician agreed with Dr Quinn's report.[186]. This is not the case. Dr Stewart had raised concerns about the fluid management.

405) He also states that no other doctors than Dr Kelly and Dr McConnell and Dr Stewart had been given a copy of this report.

406) Mr Mills[187]states that the reporting of adverse clinical incidents was being introduced as part of clinical governance arrangements and that his reporting to the General Manager of WHSSB formed part of this process which was formalised in Autumn 2004. He understood that senior officers of the Board brought significant expertise and advice to this process and refers to a Circular P1/86 WHSSB on reporting to them Untoward Events/Unusual occurrence.

407) The Trust did not report Lucy's death as an adverse incident to DHSSPS and Mr Mills explains that he understood it was the responsibility of WHSSB to do this or to

[183] Ref:116-049

[184] Confirmed in 2012 answering Q 10 of WS 293/2

[185] Ref:116-049-011

[186] Ref:116-050-012

[187] WS 293/2 Q4(a)

request the Trust to do so.[188] Although he acknowledges that the line of accountability of the Trust was to the DHSSPS no-one in WHSSB made it clear that it was the Trust's responsibility to report Lucy's death to DHSSPS.[189]

408) Comment : These points are relevant to the questions which I have raised about the role of Dr McConnell as one of the senior officers of WHSSB in that Dr McConnell expected (and understood) that the Trust had reported to DHSSPS. The role of the WHSSB has been explained further by Mr Frawley in that Board officers were involved to some respect in clinical governance issues and were seen as a source of expertise in adverse events reporting and it appears were seen as such by Dr Kelly

409) Communication with parents

410) The parents were not informed of the Trust review process nor were they invited to attend a meeting to discuss its findings before they invoked a formal complaint process in September 2000. Then on 11 October 2000 Mr Mills wrote to Mr Millar acting on behalf of the parents offering a meeting with Mr Fee, Dr O'Donohoe, Dr Anderson and Sister Traynor[190].

411) Mr Millar, on behalf of the parents, writing to the Omagh Coroner on the 31 July2000 reports that he had a copy of Lucy's case notes from the Erne hospital which parents had obtained from the Trust. He also had a copy of the full post-mortem report and notes of conversation between parents and the pathologist.

412) A formal complaint was lodged with the Trust by parents in September 2000 and then the Trust offered to meet them but no written response to points raised was made until March 2001.

413) In a letter dated 1 November 2000 to Ms B O'Rawe, Director of Corporate Affairs of Sperrin Lakeland Trust , Mr Crawford asks the Trust to provide a copy of the review[191] . Mrs O 'Rawe replied 22 November 2000 [192]explaining why the review report was not being sent to them then. Parents did not receive a copy until January 2001. The copy sent[193] did not include the recommendations present in the 31 July 2000 version..

[188] WS 293/2 Q 4(c)

[189] WS-293/3 Q1(a) &(b)

[190] 015-019-119

[191] Ref: 033-033-064

[192] 015-024-126

[193] Ref:015-028-132 to 015-028-136

414) Mr Mills replying to Mr Crawford's letter of 16th March 2001 wrote[194] :

415) "Turning specifically to the point made in your most recent correspondence, the outcome of our review has not suggested that the care provided to Lucy was inadequate or of poor quality. As you will be aware, the Trust engaged an independent consultant, from another Trust , to review Lucy's case notes and to advise us on this very question. We do however accept and acknowledge that our review has flagged up issues which the Trust wish to address for the future. These include communication and written records and are referred to in Mr Fee's report"

416) Comment: The July 2000 review had identified concerns about the quality of care. *[195]*

417) <u>WHSSB</u>

418) **MR FRAWLEY GENERAL MANAGER WHSSB UNTIL 31/8/2000**

419) Mr Frawley explains that from March 1996, the Western Board ceased to have any operational, managerial or supervisory responsibility for the three Trusts established in the Western Board area on that date.[196] The primary focus of the Board was on commissioning health and social care for the resident population. from this time the Trust's primary reporting relationship moved from the Board to DHSSPS and the Trust had no explicit responsibility for notifying the Board of unexpected or unexplained deaths but when one was brought to the attention of the Board it would ensure that an investigation was initiated, the professional leads in the Board been advised and that any formal report was shared with the Board to enable it to consider whether it needed to initiate any action in the light of the report.

420) Mr Frawley sets out the expectation of the WHSSB as Commissioner in relation to clinical governance outlined at section 5 of the service agreement SA document 4.[197] He points out that the statutory basis of clinical governance was not implemented in Northern Ireland until 2003. However he reports that from mid 90s onwards a number of initiatives were taken to develop awareness of the value and importance of clinical governance. A paper was produced setting out the key elements of the framework and listing the challenges which include identifying and minimising risk, evidence-based practice, learning lessons from poor performance, identifying problems and ensuring

[194] Ref; 033-018-033

[195] 015-034-146

[196] WS-308/1 Q1 (d) & WS-308/1 Q1 (e)

[197] WS-308/1 Q4 (a) & WS-308/1 Q15

that lessons were learnt and, in supporting professionals in delivering quality care. This was used as a platform for the Western Board to develop its approach to ensure that Trusts were central to the introduction of clinical governance and put at the centre of the relationship between Trusts and the Board in a service agreement. He refers to document 4 SA with Sperrin Lakeland Trust 1999-2000. In this attention is given to clinical governance and that it will be a standing item featuring prominently in on-going discussions with providers.[198]

421) Comment: This document appears to confirm that clinical governance and its monitoring was part of the responsibility of WHSSB.

422) In a report Mr Frawley provides *"Sperrin Lakeland Health And Social Care Trust. Risk review of acute hospital service provision through the Sperrin Lakeland model" Health Care Risks Resources International. Date February 2000*. [199] The document- (P178) notes that there was significant audit activity which is to be commended with 120 audits reported in the previous year. It reports that clinical audit should be seen as a main component of clinical governance which is an essential requirement for medical staff and should involve all clinical staff. The Trust should be able to show demonstrable results in quality improvement and risk reduction from the clinical audit process. (P180). It identifies risk as a lack of clinical guidelines, policies and clinical procedures across the Trust.

423) ROLE OF DR MCCONNELL (WHSSB)

424) Dr McConnell was a consultant in public health medicine and Director of Public Health for the Western Board at the time of Lucy's death.

425) Dr Kelly's view about the role of Dr McConnell is provided explaining why the Trust was in communication with Dr McConnell about Lucy's death *"Dr McConnell was the senior medical doctor in WHSSB and carried responsibility for the safe delivery of services and performance of the clinical teams. Dr McConnell would have been involved in any areas of service and performance or quality of care issues".* And in subparagraph (l) states *"my understanding at that time was Dr McConnell, as the senior doctor within WHSSB would have a responsibility to be satisfied that the incident was been properly reviewed. That is services and quality of care delivery was safe and appropriate- disseminating any lessons learnt across the WHSSB and possibly the wider HPSSNI"*[200]

426) Constraints and limitations on my comments.

[198] WS-308/1 P65 et seq

[199] WS-308/1. P148. Document 10.

[200] WS-290/1 Q10 P12

427) It is difficult for me to form a clear view about Dr McConnell's professional responsibilities in respect of a child death within the Board area. In November 2012 Dr McConnell reports

428) It is very important for the Inquiry to be clear about the respective roles of DHSSPS, Trusts and the Health and Social Services Boards at the time of these events as there appears to be a clear danger of a misunderstanding that there was a direct managerial relationship between the Boards and Trusts which was not the case.[201]

429) My following comments are based on my experience as a consultant paediatrician working with colleagues in public health medicine within my Trust, at PCT/district level in Wakefield, in Yorkshire region and Yorks & Humber government office, and, at national level-the latter through contacts within college/faculty networks, collaborative working in the Department of Health and numerous contacts and collaborations in boards, committees, surveys and reviews (including work with regional director of public health medicine in production of the report 2005/2007 on the Health of The Nation's Children via the York Public Health Observatory).

430) It is my understanding (which is not well informed and I stress that Public Health Medicine is not within my range of expertise) that within a locality the Director of Public Health Medicine has responsibilities which include monitoring of infant and child mortality rates addressing any unusual patterns and would produce at times an annual report which included review of child and infant mortality. It is also commonly the case that a consultant in public health medicine amongst the team working with the Director of Public Health takes a lead on child health issues.

431) Dr McConnell states that he was a commissioner of services which included those from the Erne hospital. In the case of Lucy's death he was acting mainly as a conduit for reporting a death and subsequent events to the Western Board through Mr Bradley, Director of Acute Healthcare and, to the Board's Chief Executive Mr Frawley. [202]

432) Dr McConnell explains in answering that he would have reported Lucy's death to the healthcare committee of the WHSSB to debate, endorse and agree actions taken or proposed by WHSSB staff. But he has not been able to identify recording of the discussions around the time of Lucy's death.[203] Dr McConnell explains it was appropriate for the Trust to inform the Board through him about Lucy's death and he would have wanted to receive updated information on the progress of on-going reviews Trust investigation of Lucy's death in order to keep professional and managerial colleagues within WHSSB regularly updated on related issues. But he indicates this was as their

[201] WS 286/1

[202] WS 286/1 November 2012

[203] WS-286/2 Q1 (e) & (f)-(i) ; WS 286/1 Q8

commissioning body and that there was a need for the Trust to report to DHSSPS who were their primary management authority.

433) Comment : Dr Kelly indicates that the Department of Health and Social Services in Northern Ireland had not been notified that the review was taking place [204]although Dr McConnell understood the Trust had informed done so.[205]

434) Dr McConnell states that there was no formal requirement at that time to report the death to DHSSPS he *"would have felt that this would include, Lucy's death, the events, as known at the time relating to it, the action which was being taken by the Trust to examine related causes and any actions which the Trust would be taking in the immediate aftermath".* He believed from the information provided by Mr Fee and Mr Mills that Lucy's death had been reported to DHSSPS and therefore he had no need to take any further steps to ascertain this and he would have expected Mr Mills as Chief Executive of the Trust to have informed DHSSPS. [206]

435) Dr McConnell believes that he would have discussed the issue of obtaining a wider review on Lucy's death both with Dr Kelly and with Mr Mills.[207]

436) In July 2001 Dr McConnell brought up the death of Raychel Ferguson and related hyponatraemia and fluid balance problems at the Directors of Public Health meeting with the Chief Medical Officer. This is shortly after he had received his letter from Dr Kelly enclosing Dr Moira Stewart's report. Dr McConnell did not link Lucy's death with questions arising with Raychel Ferguson's death and explains that one was related to surgery and the other to vomiting and that the relevance of the fluid management issues was not so apparent to him as in Lucy's case at the time.[208]

437) Dr McConnell explains the role of WHSSB was in introducing and monitoring clinical governance and quality of delivery of care and that the introduction of clinical governance was in very early development in 2000. [209] This view contrasts to some extent with Mr Frawley's statement who indicated it had been in hand since late 1990s (see above)

438) Dr McConnell concluded in July 2000 in respect of the review conducted by the Trust that

[204] WS 290/1 Q14 P14,

[205] WS 286/1 8(c)

[206] WS-286/2 Q1 (e)& Q2 (b) (d) ,(e) & Q3 (c)

[207] WS-286/2 Q4

[208] WS-286/2 Q7 (f)

[209] WS-286/2 Qs 6-8

439) *1. That the range of issues explored was appropriate. 2. That the range of staff involved/contributing to the review was appropriate. 3. That issues of concern had been identified regarding unclear/poor documentation, staff communication and a lack of desirable/necessary protocols and were to be addressed. 4. That the specific Cause of Death and Cerebral Oedema were still unclear and that further work/review would be desirable to resolve this.*

440) Also that the Trust was responsible for the determining whether or not an inquest had been held. [210]

441) Dr McConnell states [211]

442) *"I have no recollection of the detail of any advice I may or may not have provided to Dr Kelly or other person within S/L Trust regarding their review although I am sure that there were regular conversations and discussions between staff of the Trust and the WHSSB, including me, about this….. although I am sure that I would have discussed with Dr Kelly, following their initial review, the need for S/L Trust to consider having a wider review involving experts from outside the span of our area and settings/clinicians involved in any treatment roles. "*

443) Yet 2001 when he received Dr Moira Stewart's report Dr McConnell was aware that the Trust had not followed up to his advice to the Trust to obtain further wider opinion on Lucy's death as it is clear from correspondence and from Dr Moira Stewart's opinion that she was not undertaking a review of the cause of Lucy Crawford death.

444) Dr McConnell's letter of 5 July 2001[212] to Dr Kelly focuses on personnel matters re consultant practice rather than concerns about Lucy's death.

445) *"Thank you for your letter of 27th of June 2001, and in the enclosed detail regarding the feedback from the Royal College of Paediatricians regarding their concerns raised about the performance of Dr O'Donoghue(sic). I have had a good look through this and would be more than happy to discuss with you the issues arising from it. Overall, initially, this seems to capture a range of the issues of which you and I have now become all too familiar. There are issues of system failures, communication failures and individual performance failures but I suppose the most pertinent comment that I am not sure that all of these are sufficiently clear and serious to form the basis of very definitive action in relation to Dr O'Donohoe. I do feel however that there is likely to be a need for the Trust to discuss these findings in some detail with Dr O'Donohoe and to get some sense from him of what program of corrective action he would propose to make in order*

[210] WS 286/1 Q15

[211] WS 286/1 Q 8(c)

[212] Ref: 036a-029-070

to be able to respond to the deficiencies identified. Obviously, you and I need to follow up on this with a more detailed conversation."

446) <u>RESOURCES AVAILABLE TO THE TRUST (and Dr Kelly) IN 2000 IN N.I. FOR FURTHER ENQUIRY INTO AN UNEXPLAINED CHILD DEATH</u>.

447) In July 2000 The Trust review had determined that Lucy's death was unexplained. Further investigation should have been set up even if a Coroner's Inquest was pending to clarify questions about her care which were unanswered. There were a number of resources for advice available in 2000 to have been considered by Sperrin Lakeland Trust or WHSSB for the investigation of Lucy's death. These include:

- By referral to the Coroner
- By requesting an external formal professional review.
- Using the expertise of the Confidential Enquiry Into Stillbirths And Deaths In Infancy (CESDI) or of the Confidential Enquiry into Perioperative Deaths (CEPOD)
- Seeking advice from the Medical Directors network or The British Association of Medical Managers (BAMM).
- Seeking advice from RCPCH
- NCAS advice available from 2001 onwards

448) **Referral to the Coroner**.

449) Lucy's death was sudden and unexpected and was an extremely rare complication of her underlying illness-gastroenteritis. There were very strong arguments for a Coroner's inquest and investigation. Lucy's death fell into the category clearly defined in Northern Ireland for referral as set out in the 2008 guidance in Northern Ireland (given in Annex D) which is based on section 7 of the Coroner's Act (NI) 1959 and thus applied at the time of Lucy's death.

- *in any circumstances that require investigation;*
- *the death, although apparently natural, was unexpected;*

450) The Sperrin Lakeland Trust recognised the requirement for investigation in a child whose death was regarded as natural but unexpected. The Trust review had identified criteria under which, arguably, Lucy's death could have been reported to the Coroner listed in the report for the Inquiry from Bridget Dolan :

- *"The death may be related to a medical procedure or treatment whether invasive or not"*
- *"The death may be due to a lack of medical care"*

- *"There are any other unusual or disturbing features to the case"*

451) The death was unexpected and fulfilled at least one of the criteria set out: in that it was arguably not "*apparently natural"* complicating a disease with a very low mortality and there were indications that it may have resulted, at least in part, from the therapy used. Mr Mills, Mr Fee and Dr Kelly have all asserted in their police interviews that they assumed that a referral to the Coroner had been made and that an Inquest was to take place. The RBHSC had referred to the Coroner and knew that an Inquest was not planned. It is my opinion that simple enquiry made of the RBHSC about their opinion and the Belfast Coroner's office should have determined what was in process and, in any event, there was a arguably a responsibility of the Trust to send information to the Coroner from the review that Lucy's death was unexplained.

452) **A Trust external formal professional review**.

453) In 2000 there were no routine formal systems set up for death enquiries in children in Northern Ireland (nor in England and Wales) for a death outside infancy : Confidential Enquiry into Maternal and Child Health (CEMACH) – an extension of the Confidential Enquiry into Stillbirth and Deaths in Infancy (CESDI) to all ages in childhood was not yet in place. See Annex E

454) **Use of the expertise of the Confidential Enquiry Into Stillbirths And Deaths In Infancy (CESDI).** The Trust could have sought advice and contacts from CESDI which had expertise in Northern Ireland for enquiry into infant deaths and the investigation of sudden unexpected death in infancy including deaths in hospital. Although their processes were for children under the age of 12 months, they could just as appropriately be applied in a child five months older. I have attached the 2001 Northern Ireland report of CESDI and extracted in Annex E some important quality issues raised in their experience in relevant to risk management in hospitals in N.I. at the time in question. The report was introduced by the Chief Medical Officer. CESDI used panels whereby clinical records are assessed by a multidisciplinary group of independent assessors. This system would have been well known to Dr Anderson as an obstetrician because much of the focus is on perinatal mortality and to Dr McConnell (and to Dr Quinn). Dr Kelly's understanding of the Confidential Enquiry Into Stillbirths and Deaths In Infancy is correct in that its formal structure only considered deaths of infants up to one year but their advice and use of their expertise by the Trust could have been used outside the CESDI formal process [213]

455) **CEPOD :** although Lucy did not die from an operation or an anaesthetic, an anaesthetist was involved. The expertise and experience of the CEPOD enquiries could have been used as a format for investigation of Lucy especially as fluids were implicated and fluid balance in children had been the focus of CEPOD reports in the past.

[213] WS-290/1 Q13(b)P13

456) **Director of Public Health/DHSSPS.** Alternatively the Trust could have asked Dr McConnell or DHSSPS for advice on how to set up an external enquiry.

457) **Medical Directors Network**. Trust medical directors met together on occasion. The reviews from the Royal hospital on clinical governance show that from 1997 onwards there was development of expertise and experience in clinical governance and this is confirmed by Mr Frawley[214]..Although Mr Mills stated he was reluctant to seek support from Belfast because Lucy had been treated there, the Royal hospital clinical governance review did not necessarily need to be involved with paediatricians who had treated her. In Dr Kelly describes the Medical Directors network *"as a new grouping meeting with the CMO to discuss strategy service change and re-design matters it is not a forum ever used to discuss adverse incidents or deaths…."*. [215].Comment- their meeting, in my opinion , was an opportunity for networking and seeking advice as referred to in Dr Kelly's answer to the question about the meeting in June 2001 when he informally discussed the case of Rachel Ferguson with a colleague and use of number 18 solution.[216]. The same point applies regarding British Association of Medical Managers (BAMM) - see Annex E - on which Dr Kelly comments he was not aware that they provided advice on managing individual cases but in my opinion could have been used informally for advice.

458) **RCPCH**: Professional advice from a college regional adviser or directly from the college was available and in the event the Trust did resort to this available support. This was in the context of concerns about professional competence rather than further exploration of the cause of Lucy's death. Nevertheless the College, if approached, might have been prepared to signpost Dr Kelly to paediatricians with appropriate expertise to assist an investigation on the part of the Trust into the death of Lucy or at least provided advice on what to do.

459) **The National Clinical Assessment Service (NCAS)**. The National Clinical Assessment Service (NCAS) was established only in April 2001 following recommendations made by the Chief Medical Officer for England in his report "*Supporting Doctors, Protecting Patients"* published in November 1999 and in "*Supporting Doctors, Protecting Patients"* of January 2001. The latter however was current as a source of informal advice even though not yet set up in N.I. at the time. The role of NCAS is described in Annex E

460) "*An Organisation With A Memory*" published in June 2000 identified 4 key categories of serious recurring adverse events and recommended that a new national mandatory system be established to record and analyse adverse events in healthcare.

[214] WS-077-2 and WS-308/1

[215] WS-290/1 Q 13 (b) P13

[216] WS-290/1 Q32 (a) P 24

The document refers to an additional resource-the NHS *Clinical Governance Support Team* which from February 2001 had a website (now defunct but available at the time to Dr Kelly and others). I can find no record that Dr Kelly contacted the NCAS, nor whether he considered doing so in 2001 or 2002 when RCPCH when was asked to conduct another review raised this as an issue with the Trust. Dr Kelly highlights that NCAS did not exist in 2000 and started in England and Wales in April 2001 and was introduced in 2004 to Northern Ireland. It is probably for this reason that Dr Kelly did not involve this authority, as the Royal College advised, in 2002 when Dr Boon and Dr Stewart produced their reports. [217]

461) With more recent attention to structured clinical governance in the late 2000s, there is much more information available than was available 2000/2001. But the basic principles remain the same that the investigators will collect evidence, interview the practitioner, weigh the evidence and identify the facts of the case. These were all relevant to the internal review conducted by the Sperrin Lakeland Trust.

462) **SHARING AND INTERPRETATION OF DEATH CERTIFICATES IN CHILDREN**

463) I am not familiar with the structure and processes in Northern Ireland for recording and processing of death certificate information in children. In my own district all child deaths were reported to the community child health service by the registrar of deaths. Following my appointment in 1978 with some responsibilities for community child health at the time, a system was set up by which the community child health Department would in turn inform me by telephone of every child death and the cause. This continued on an informal basis until the establishment of CESDI when it became a responsibility of a nominated consultant paediatrician in every district to be informed of every death under age 12 months and to complete a questionnaire set out by CESDI on each child and return it to the CESDI office. I note that CESDI was in place in Northern Ireland in 2000 when Lucy died. Had a similar system been in place in Erne, the Trust would have been made aware of the certificate entries of cause of death. From around 2006 Local Children's Safeguarding Boards (LCSB) were set up which reviewed every child death and a lead paediatrician was nominated taking responsibility for review of each death. From 2008 following the Children Act revision, copies of death certificates are sent to the LCSB.

464) LIKELY OUTCOME OF A STRUCTURED REVIEW OF LUCY'S DEATH

465) Any such structured review would have involved enquiries made of the clinicians who cared for Lucy in Belfast as well as the team in Erne and it is an outstanding deficit of the Sperrin Lakeland Trust review process that they did not seek opinions from Belfast on her care or raise questions about how she might have developed cerebral

[217] WS 290/1 Q 13(b)P14

oedema**.** Especially as the review had identified communication difficulties as an issue to be addressed.

466) A formal structured enquiry into the death of Lucy is likely to have reported by December 2000 and identified the factors summarised in the causation of Lucy's death.

467) Identification of the incorrect use in Lucy of number 18 solution in 2000/2001 may not have led to general concerns about its use in children. On the other hand a conclusion could have been drawn similar to that of Dr Boon and Stewart in 2002 that "*More careful attention to detail of the fluid therapy might possibly have avoided this girl' s cerebral oedema and fatal outcome".* It is arguable that a review could have drawn greater attention to the risk of use of No 18 solution as a replacement fluid and provided a caution for dissemination to colleagues but the specific risk for hyponatraemia and cerebral oedema was not well known in 2000 compared with 2002. In contrast both N18 solution and normal saline given to Lucy were wrongly used in Lucy. Any medication used incorrectly is associated with risks of adverse outcome and her death may have been regarded to be a result of that misuse with implications to report as a drug error and to make either a referral or report to the Coroner for the Inquest and without considering a need for wider dissemination. It may not have been recognised specifically as a dangerous medication. (This appears to be the conclusion of the review by the Medicines Control Agency in 2001 in their response to Dr Taylor)[218].

468) **SPECIFIC QUESTIONS ON GOVERNANCE ISSUES POSED TO ME BY THE IHRD BRIEF** *Responses in italics*

469) **Erne Hospital and Sperrin Lakeland Trust**

470) Whether the Erne Hospital/Sperrin Lakeland Trust ought to have had a risk management or clinical governance policy, and if so what should that policy have contained, the applicability of such a policy to Lucy's case, and the steps that ought to have been taken pursuant to such a policy

471) For the time in question many hospitals did not have a well-developed clinical risk or clinical governance policy. As a minimum however regular clinical audit should have been carried out and there should have been a clinical incident reporting process. This appears to have been in place in the women and children's directorate. It would be of interest to determine how well-developed that was and what documentation exists in support of it. Nevertheless the way in which the review was carried out and the way that the report was managed showed that whatever the intention, the process was seriously flawed.

[218] Ref: 007-032-059

472) Whether the Erne Hospital/Sperrin Lakeland Trust ought to have had an incident reporting procedure, and if so what should that policy have contained, the applicability of such a procedure to Lucy's case, and the steps that ought to have been taken pursuant to such a procedure

473) Lucy's death was regarded as a significant clinical event and the process was set up to investigated. This process in the end was flawed.

474) The factors that determine whether a death should be treated as an 'adverse incident' and whether those factors were present in Lucy's case so far as the Erne Hospital/Sperrin Lakeland Trust is concerned

475) Lucy's death remained unexplained and arguably could be seen as an adverse incident. To the extent it was subject to a review at Erne however flawed the review and reported it to WHSSB it was being handled as such. The lack of notification to DHSSPS by Erne appears to relate to confusion about this reporting pathway. Mr Mills Chief Executive expected WHSSB to do this or advise it and the latter expected Mr Mills to do it.

476) The steps that should have been taken when an 'adverse incident' occurs and, if applicable, whether those steps were taken in relation to Lucy's death

477) *see above*

478) The steps that should have been taken to examine the circumstances of Lucy's death and to reach an accurate conclusion on its cause, and the adequacy of the steps that were taken at the Erne by the Sperrin Lakeland Trust

479) *I have covered these points in my report in paragraphs 287-293*

480) The adequacy of the review which was undertaken at the Erne Hospital by the Sperrin Lakeland Trust to examine the circumstances and cause of Lucy's death, including the adequacy of its efforts to obtain all relevant information from staff or from elsewhere

481) *See above*

482) The information which was available to the Erne Hospital/Sperrin Lakeland Trust to enable it to investigate and explain the circumstances of and the cause of Lucy's death, the sources of that information, and the adequacy of the use it made of the information available to it, to include a consideration of the steps that were taken to obtain relevant information from nursing and medical staff

483) *See above.*

484) The factors that determine whether an external clinician (expert) should be asked to participate in an internal review, how the role of that clinician and the objectives of the

review are defined and explained to him and how that clinician should be asked to go about his task

485) At the time there was little formal guidance or process set out on how to conduct a review. Case note review forms part of the process of dealing with litigation or complaints and many clinicians have experience in this process. Common sense however applies. Clinicians have experience in analysing complex data and reporting on it. They have a responsibility when asked to conduct a review to do this scrupulously and accurately. If they have reservations about the information available to them this should be included in the review report. If they have reservations about the extent of the review or the purpose that the report would be put to this should be written in their report. The selection of an external expert however at the time was somewhat ad hoc. The investigation and opinion sought from a general paediatrician would however be generally appropriate where fluid management was in question particularly in a common condition like gastroenteritis and should have been within the competence of any experienced general paediatrician. Consequently the selection of Dr Quinn on this basis was not inappropriate if he was seen truly to be independent of the Trust . The selection however of an independent expert would often be conducted in consultation with the local director of public health or regional medical officer in England. Dr McConnell was informed of the selection of Dr Quinn and made no objection.

486) Whether the limitations which Dr. Quinn asserts that he placed around how he would perform his role were appropriate and acceptable

487) In the circumstances I believe this was acceptable. Dr Quinn was informed that his opinion was being sought as part of an initial process of review. It was reasonable for him to confine his opinion only on case records. A more formal structured enquiry would however require interviews with staff and parents. I suspect Dr Quinn relied on the internal review process to take these steps and that he was to some extent making a contribution to it. However his contribution was inaccurate even given the limitation of the information in the case records

488) Whether those limitations had any impact on the value of Dr. Quinn's role and the quality of his report

489) Dr Quinn stated he was not able to explain the cerebral oedema. This was the cause of death and should have been investigated further. He also raised questions about the accuracy and uncertainty of the quantities of fluid given. He made errors in his calculation of the time base over which the fluid was given and did not appreciate or advise that the high-volume of No 18 solution and rate used was not appropriate to the clinical condition of Lucy. The Trust appeared to rely on his report despite its limitations and failed to pursue an investigation to explain the cause of death.

490) The thoroughness and completeness of the report provided by Dr. Murray Quinn having regard to the information with which he was provided and the issues he was asked to address

491) *I have covered these points in my report in paragraphs 199-256 it would have been more helpful for Dr Quinn to have been appraised of the information which was assembled by Mr Fee. Dr Quinn raised questions identifying information he was lacking but these do not seem to have been incorporated as qualifying statements in the review conclusion made by Dr Anderson.*

492) Whether there should be provision for a follow up discussion between the external clinician providing the report and the Trust who is commissioning the report, and whether one should have taken place in Lucy's case

493) There should have been a follow-up discussion in the light of all the other information that was submitted to the review process. While there is no guidance at the time about this, common sense should rule. There is also experience both within and outside hospitals in conducting reviews of infant deaths-for example sudden infant death/cot death and in the perinatal period. The contributors to the review, including Dr Quinn, should have been shown the review and asked to comment on it before it was finalised.

494) Whether the parents of a deceased child should be informed that a review/investigation is being undertaken and asked to contribute to the review/investigation and the reasonableness of the decision not to include Lucy's parents within the review process

495) The parents should have been informed that a review was in hand and should have been invited to contribute. The results should have been given to them on completion of the review and their questions addressed.

496) The reasonableness of the conclusions reached in the Trust 's review, particularly around fluid management

497) The conclusions drawn that Lucy's death remained unexplained was a major trigger for further investigation. Dr Quinn and Dr McConnell assert that they had advised the Erne team to do so and this should have happened despite the shortcomings in Dr Quinn's report. The review was not conducted properly.

498)

499) The adequacy of the Trust 's response to implementing the recommendations of the review, and to disseminating lessons learned

500) *I have covered these points in my report in paragraphs 257-312*

501) What communication ought to have taken place and what information ought to have been exchanged between the Erne Hospital/Sperrin Lakeland Trust and RBHSC/Trust and vice versa, in relation to Lucy's deterioration and death, including the information that should have been given to the RBHSC at the point of transfer, and whether the communication that did take place and the information that was exchanged was adequate.

502) It was a critical omission that during the course of the review or subsequently, that no communication took place with RBHSC. A discharge summary was not received or sought and the content of the death certificate was not recorded. No attempt was made to determine whether an inquest was to be held and no attempt was made to inform the coroner of the results of the review. WHSBB was aware that this step had not been taken because the review report was shared and did not draw attention to this deficit. The review had determined that Lucy's death remained unexplained yet no attempt was made to communicate with the treating team at Belfast or the pathologist to discuss this or obtain their views the Erne Hospital/Sperrin Lakeland Trust ought to have advised the RBHSC/Trust of its decision to conduct a review, and whether it should have sought its participation in the conduct of that review

503) Whether the Erne Hospital/Sperrin Lakeland Trust ought to have advised the pathologist (Dr. O'Hara) of its decision to conduct a review and its outcome, and whether it ought to have advised the pathologist of the reviews conducted by the Royal College of Paediatrics and Child Health (in relation to Lucy) and their outcome

504) The Erne Hospital/Trust did not know (and made no attempt to find out) that a Coroner's inquest was not to take place. They did not determine that a death certificate had been issued or what its content was. In conducting a review however it was their responsibility-especially that of the medical director-to share the review as part of the information given to the Coroner if he thought that the Coroner was holding an inquest. Whether in this process they should have informed the pathologist depended on whether or not they thought the pathologist was conducting a Coroner's autopsy. They made no attempt to find that out. Further, they received a copy of the hospital post-mortem report in which there was no record that this was a Coroner's pm. They were aware that the pathologist had met the parents but made no effort to determine what the content of the meeting and his explanation was despite the fact that they recorded the death was unexplained. This is very difficult to understand.

505) As far as the reviews carried out by the Royal College of Paediatrics And Child Health are concerned, the findings should have been reported to the Coroner by the Trust rather than to the pathologist.

506) What communication ought to have taken place and what information ought to have been exchanged between the Erne Hospital/Sperrin Lakeland Trust and the Crawford family and vice versa and whether the communication that did take place and the information that was exchanged was adequate

507) The parents should possibly have been invited to contribute to the review and certainly made aware that the review was taking place. One of the recommendations (and one of the purposes of the review) was to meet parents and share the findings. This did not happen.

508) The significance of the role played by the Royal College of Paediatrics and Child Health in the reviews carried out under its auspices particularly in relation to the treatment of Lucy and the cause of her death, the adequacy of the action taken by the Trust on foot of the conclusions reached by the Royal College, and whether the findings of the reviews (to the extent that they related to Lucy) ought to have been disseminated, and if so, to whom

509) Reports should have been made to the Coroner. At the same time in order to provide sufficient information to the Coroner and for clinical safety and governance purposes, an external review specifically on cause of death should have been set up. The focus of the Royal College representatives visit was on the competency of a consultant not on the investigation of the death of Lucy. The RCPCH made this clear to the medical director.

510) What communication ought to have taken place and what information ought to have been exchanged between the Erne Hospital/Sperrin Lakeland Trust and the Western Health and Social Services Board and vice versa in relation to Lucy's deterioration and death and whether the communication that did take place and the information that was exchanged was adequate

511) The conduit for information sharing with WHSSB was via Dr Kelly to Dr McConnell and by Mr Mills to senior general management in WHSSB. The death was reported as was the process and findings of the review. The results of this fell short of what was required. WHSSB did not follow up to check that advice given to hold a more detailed review was being taken and they should have checked that DHSSPS had been informed- both appear to be part of their responsibilities as set out by Mr Frawley.

512) Whether the Sperrin Lakeland Trust ought to have reported the death of Lucy to the Coroner's Office and ascertained whether an Inquest would be held, and if so, when, and whether the Trust ought to have advised the Coroner's Office of the outcome of the internal review and/or the findings of the reviews carried out by the Royal College

513) Yes

514) The circumstances in which a Medical Director might be expected to challenge or query the findings of a post-mortem, and whether the Erne/Sperrin Lakeland Medical Director ought to have challenged the findings of the post-mortem in Lucy's case

515) *In order for a medical director to do this he would have needed information from a review of the clinical management either from his own team or from an external review unless the specialty concerned was within his area of expertise. The only information*

that the Trust medical director had was from the autopsy which reported cerebral oedema and bronchopneumonia but made no mention of hyponatraemia. Hyponatraemia had led to concerns within the Trust. The medical director Dr Kelly would have been aware of these concerns in respect of fluid and electrolyte matters by virtue of the framework within which the internal review was set up and on which it reported. However the paediatric opinion offered him, appears to have been taken as reassuring until Dr Stewart's report was available in 2001 followed shortly by the information about hyponatraemia occurring in Raychel Ferguson. In 2000 although Dr Quinn acknowledged in his written report that he has not been able to conclude why Lucy developed cerebral oedema, he did not recognise the fluid overload which would be a plausible explanation amongst others for cerebral oedema.. Nor did he comment on the significance of the low sodium in regard to its possible contribution to the cerebral oedema although he reported the finding. In 2001 action should have been taken in referring to the Coroner and/or setting up an expert review. Had Erne set up a wider clinical review the cause of death related to wrongly used IV therapy would have come to light probably by latest December 2000. However any drug used incorrectly can be dangerous and thus it may have not raised any generalizable concerns about No18 solution. Nevertheless it is also possible that that a conclusion could have been drawn similar to that of Dr Boon and Stewart in 2002 that "More careful attention to detail of the fluid therapy might possibly have avoided this girl' s cerebral oedema and fatal outcome". It is arguable that a review could have drawn greater attention to the risk of use of No 18 solution as a replacement fluid and provided a caution for dissemination to colleagues.

516) The Western Health and Social Services Board

517) What communication ought to have taken place and what information ought to have been exchanged between the Erne Hospital/Sperrin Lakeland Trust and the Western Health and Social Services Board and vice versa in relation to Lucy's deterioration and death and whether the communication that did take place and the information that was exchanged was adequate

518) See Paras 417-445

519) Having been advised of the circumstances of Lucy's death, what steps should the Board have taken to obtain further information

520) In my view an external report could have been set up in 2000 by the Trust as Dr McConnell advised. The WHSSB could have checked that one was to be established as part of the governance aspects of commissioning and could have reported within a matter of months. The RCPCH review was on clinical competence and not on Lucy's death. It reported 12 months after Lucy died.

521) Whether the Board had any general responsibility to ascertain and satisfy itself that the cause of the Lucy's deterioration and death was being effectively investigated by the Erne/Sperrin

522) In my opinion yes. It should have been evident that the quality of the review was poor. But I have limited understanding of the role of WHSSB and advise that an opinion of a specialist in public health medicine is obtained.

523) Whether the Board had any general responsibility to disseminate to others information regarding the deterioration and death of Lucy and the lessons to be learned from it, and if so, identify the persons/organisations to whom such information should have been disseminated

524) If general issues had been identified then they would have had such a responsibility. This happened after 2001. And might have happened after a proper investigation of Lucy in 2000.

525) The adequacy of the Board's response to Lucy's death in light of its particular role and responsibilities

526) *Both the Chief Executive and the Medical Director reported events. The main focus appears to have been on disciplinary matters with Dr O'Donohoe despite the fact that Dr McConnell had written to Dr Kelly (036a-029-070) on 5 July 2001 criticising systems failures and communication failures (i.e. governance matters.). But I have limited understanding of the role of WHSSB and advise that an opinion of a specialist in public health medicine is obtained and thus defer on this issue to an expert in public health medicine.*

527) LUCY CRAWFORD : EVENTS IN ROYAL BELFAST HOSPITAL FOR SICK CHILDREN (RBHSC) AND OTHER EVENTS, INCLUDING THE AUTOPSY AND ISSUE OF THE DEATH CERTIFICATE.

528) Lucy died on 14 April 2000 in RBHSC. After discussion with the Coroner's office a hospital autopsy was arranged. A **death certificate** issued on 4 May 2000 gave causes of death as *1 (a) cerebral oedema; (b) dehydration ;(c) gastroenteritis*.

529) **After the 2004 Inquest Lucy's death was certified as**

I (a) cerebral oedema; (b) acute dilutional Hyponatraemia; (c) excess dilute fluid

II gastroenteritis.

530) Summary of clinical events

531) Lucy was admitted after transfer from Erne hospital to the paediatric intensive care unit at RBHSC around 0830 in the morning on 13 April 2000. She was on intravenous normal saline and dopamine infusion. On admission to PICU she weighed 9.8 kg (having been 9.14 kg on admission to Erne) . Lucy was admitted under the joint care of Dr Crean consultant paediatric intensivist and Dr Hanrahan consultant paediatric neurologist. She was assessed by the paediatric intensivists and supportive therapy was continued by ventilator and intravenous infusion.

532) Lucy had been accompanied by Dr O'Donohoe from Erne Hospital during the transfer who provided a handwritten summary [219] to RBHSC. The only reference to fluid management is *"She had 1gm Claforan at (approximately symbol) 0500 and 5gm Mannitol (approximately symbol) 0500 (with a brisk diuresis)" ,* and a hand written list of observations which appears to be the transfer record where 500 mL NaCl 30ml/hr is entered. Timing from 6:35 AM up to 7:50 AM.[220].

533) It is not evident that Dr O'Donohoe informed the PICU staff of the low blood sodium level found at Erne and this information was provided later by Dr Auterson by telephone. On reaching the PICU on 13 April 2000 Dr O'Donohoe was met by Dr McKaigue who had accepted the transfer from Erne after telephone discussion with Erne hospital during which he gave advice on care after the respiratory arrest.[221] Dr McKaigue does not report any detail given him about the fluid management at Erne but recalls that a dextrose based solution was administered. Dr McKaigue then handed over to Dr Chisakuta and Dr Crean. Dr McKaigue recalls that *" at some point I became aware from*

[219] 061-014

[220] 016-016

[221] WS- 302/1 P 6

Dr Crean that there were issues around Lucy's fluid management ".[222] Dr McKaigue cannot recall when this was. The concern raised in particular was that the blood sodium could have been lower than 127 because the blood sample was taken after Lucy had been administered a large bolus of 0 9% saline during her resuscitation.[223]

534) The first RBHSC paediatric medical record entry was at 08:30 the initial admission record made by the senior house officer Dr McLoughlin. In the margin of the page (061-018-058) urea and electrolytes results at admission from Erne hospital are listed. It is not evident when this entry was made or how this information was obtained. At the end of the admission record Dr McLoughlin, wrote *"Erne hospital notes requested for further information".* Later there is an entry at 09:00 hours providing information from telephone call from Dr Auterson (anaesthetist at Erne hospital) that blood sodium level was 127 mmol/l per litre on repeat and this information is entered just above Dr Hanrahan's (10:30) entry[224].

535) An untimed type written record (061-018-065) by Dr Crean on 13 April 2000 summarises Lucy's clinical state. This includes a note that the sodium on Ward testing is 140 and that Dr Crean is awaiting faxes of her notes from the Erne hospital. There is no mention of hyponatraemia although the blood sodium level of 127 mmol/l was available in the handwritten case record at 09:00 hours. It appears that when Dr Crean saw Lucy, notes had not arrived by fax from Erne. Dr Crean explained that it was usual practice to obtain copies of the patient's notes from the referring hospital and this was the reason for obtaining the fax. He does not recall reviewing the notes but it would have been his usual practice to do so. There is some uncertainty about when the fax arrived and he states that it is likely he would not have seen it until after his Ward round. Dr Crean points out two separate times on the fax sheet 08:53 hours and 09:51 hours (061-017-054) he states that "*on review of the fluid balance chart now it is not clear to me how much fluid was actually administered to Lucy ."*[225]

536) Later on the 13 April 2000 there was a telephone discussion between Dr Crean and Dr O'Donohoe recorded in the Erne case records dated 14 April 2000 [226](which Dr Crean does not recall) noting:

537) *Yesterday Dr Peter Crean rang from PICU RBHSC to enquire what fluid regime Lucy had been on. I told him a bolus of 100 mls given over one hour followed by 0.18 % NaCl/dextrose 4% at 30 mls/hour. He said he thought that it had been NaCl 0.18%*

[222] WS 302/1 P7

[223] WS-302/2

[224] 061-018-059

[225] WS-292/1

[226] 027-010-025

Dextrose at 100 ml/hour. My recollection was of having said a bolus over one hour and 30 ml/ hour as above. Lucy had had 50 mls PO before I saw her I gave her 100 mls while waiting for the EMLA cream to take effect. Maintenance (approximately symbol)100 mls/kg (approximately symbol) = 1000 mls (- 150 ml PO+100ml bolus) = 750 =30ml/hr. Immediately pre-transfer I had discussed fluid rate for the journey. Dr Auterson. Calculated 40ml/hr (? 1000/24) but I thought continuing at 30 mls was appropriate. Signed Dr O'Donohoe

538) Dr O'Donohoe has no recollection of the contents of this conversation with Dr Crean. He concluded that the fluid was recorded as 100 mL per hour as suggested by Dr Crean after he had reviewed the notes following the conversation and that he became concerned about the quantity of fluids actually infused and discussed Lucy's management with Dr Kelly. [227]. He made no reference to this in his report to the Erne review. He does state that Dr Crean raised concerns about the fluids given and were *" different from those I had instructed to be given " .* This suggests that Dr Crean had reviewed and questioned Lucy's fluid regime in Erne on 13 April after review of Erne notes.

539) Dr Crean explains that Lucy was admitted under the care of Dr McKaigue and was jointly managed by the consultant anaesthetists in PICU and Dr Hanrahan consultant paediatric neurologist and he states that the clinician in charge of Lucy's case was Dr Hanrahan.[228]

540) At the time he had some concerns about administration of excessive volumes of hypotonic fluid and the lack of clarity about the volume of fluid infused. Nevertheless in respect of an explanation for Lucy's cerebral oedema, Dr Crean points out that it was only later that it became evident that, rarely, a child with a blood level in the order of 127 might develop hyponatraemic encephalopathy and usefully refers to the relevant literature. With the knowledge available to him at the time he would not have considered a sodium level of 127 low enough to have formed the opinion that dilutional hyponatraemia had caused Lucy's acute collapse.

541) Comment : Dr Crean did not take account of the effect of a rapid infusion of a large volume of normal saline on the blood sodium which might conceal a much lower level at the time of Lucy's collapse and indicates that Lucy's death was unexplained in April 2000. But the second blood sample was obtained during the respiratory resuscitation and the case notes do not identify the sample time nor who obtained it or whether it was taken before or following the start of the normal saline infusion. But Dr O' Donohoe reported to the Trust review that it was taken by him and thus after the saline was running in his report to the review.[229] Dr Crean reports that her death at that time

[227] WS-278/1 Q 8 ; Q 8 (b) (c); Q 8 (d) (e)

[228] WS-292/2 Q5

[229] 033-102-292

would not necessarily have triggered an incident form to have been completed although today an incident form would be completed for unexplained deaths. He also states the responsibility for presentation of Lucy at the audit meeting was that of Dr Hanrahan.[230] But Dr Crean does not report that by the procedure in RBHSC in 2000, he should have attended the mortality review in August.. I agree with Dr Crean about the lack of clarity in the fluid balance and other records about the fluid therapy in Erne (and timing of the second blood sample) because it has required a rather long and detailed look at the case records in order to determine how much fluid had been given to Lucy. In my view this would not have been straightforward to do during management of the clinical case in an acute ward setting yet it appears that he was concerned after reviewing the Erne notes and a quick review would indicate that 100ml/hour of 0.18 % saline and a large volume of saline had been given starting at the time of the arrest and Dr Malik's note (061-017-048) reports 500ml given in 1 hour , the nursing note states " *IV fluids changed to 0.9% saline and run freely* " (061-017- 050) and the fluid balance chart shows 500ml at 3am (061-017-056). The Erne ICU fluid chart showing 250ml saline had been given by 4am (027-025-076) is not included in the faxed papers in the Royal Hospital notes. Thus it would have been apparent a large volume of normal saline up to 500 ml had been given rapidly during the resuscitation between 03:00 and 04:00.

542) At 10:30 hours Lucy was assessed by the paediatric neurologist Dr Hanrahan who considered a differential diagnosis of infection, herpes, haemorrhagic shock encephalopathy, metabolic e.g. urea cycle disorder or cerebral oedema from other cause. *"No cause is clinically evident as yet"*.[231] A series of investigations was carried out including EEG, CT scan and liver function tests, ammonia , clotting screen, herpes titre, urine for organic acids and toxicology. It was noted that stool cultures had been sent from the Erne Hospital. Dr Hanrahan reports he did not have access to the Erne notes when he reviewed Lucy at 10:30 and is unable to state why or when he first had access to them or the fluid chart. He cannot recall a conversation with Dr Crean before reviewing Lucy. In answering he reports that the source of his information that Lucy had been given 0.18% saline came from the fluid balance chart from the Erne hospital from 11 pm to 3 am. He did not document any concern in relation to the appropriateness or otherwise of administering 100 mls per hour to Lucy or whether it caused or contributed to her collapse. In his witness statement Dr Hanrahan notes that he considered that 127mmol/l represents only mild hyponatraemia.[232]

543) At 17:45 hours Dr Hanrahan recorded that the prognosis in Lucy was hopeless and indications were that Lucy was brain dead.

[230] WS-292/2 Q1 &2; Q9; Q4 (c);Q5 ; Q 10 (f); Q 11

[231] 061-018-063

[232] WS-289/1 Q 7

544) The parents were counselled throughout the time Lucy was in RBHSC and this was recorded. The second brainstem tests were carried out at 1030 on 14 April 2000.

545) At 11:30 on 14 April 2000 Dr Caroline Stewart (specialist registrar) recorded that Dr Hanrahan had contacted the Coroner and discussed the case with Dr Curtis. *"Coroner's PM not required, but hospital PM would be useful to establish cause of death and rule out other diagnoses*."

546) Ventilatory support was withdrawn at 13:00 hours and Lucy was pronounced dead at 13:15 hours on 14 April 2000.

547) The paediatric intensivists and consultant paediatric neurologist had the same information from the records as was available to Dr Quinn limited though it was. Dr Crean knew that Lucy had cerebral oedema and that hyponatraemia had developed at Erne by the time of the second blood sample (sodium 127 millimoles per litre).

548) The clinical management of Lucy at RBHSC appears to have been of high standard. On admission the clinical assessment confirmed that Lucy was irretrievable and the responsibility of the team was to support her while confirming findings of brain death and pursuing investigations to attempt to identify the cause of her coma and the resultant coning which had occurred. The range of investigations was appropriate and results of brainstem testing confirmed the initial impression. Parents were supported through this time. The clinicians recognised that a post-mortem examination would be necessary and a hospital post-mortem arranged after Dr Hanrahan took advice from the Coroner's office.

549) Dr Stewart obtained consent from the parents for this although I cannot find a consent form completed. Dr Caroline Stewart made a note that she obtained written consent does not recall whether written consent was given or whether she or Dr Hanrahan obtained consent from parents. She points out that it was within the range of practice at the time for verbal consent only to be obtained. Dr Stewart recalls that the conversation with the Coroner's office was held by Dr Hanrahan because Lucy had an acute event which led to her death and the cause of this was not clear.[233]

550) <u>Post death Actions</u>

551) Records show Dr Caroline Stewart spoke to the pathologist Dr O'Hara and completed an autopsy request form. A handwritten entry by Dr Caroline Stewart [234] notes consent (written) by parents is accompanied by a tick. The autopsy request form notes "*parents wish organs to be used for donation*." [235]

[233] WS-282/1 Q6 (d)P5

[234] 061-018-068

[235] 061-022 -074

552) The autopsy request form included information that Lucy had been given intravenous fluids *" (No18 and N-saline)."* The section on clinical diagnosis records dehydration and hyponatraemia. Dr Stewart lists the clinical problems in order of importance:

- *Vomiting and diarrhoea*
- *Dehydration*
- *Hyponatraemia*
- *Seizure and unresponsiveness leading to brain stem death.*

Completion of the death certificate

553) The preliminary autopsy on 14 April 2000 reported a swollen brain with generalised oedema and pulmonary congestion and oedema.

554) On 4 May 2000 Dr Dara O'Donoghue (Clinical Fellow Intensive Care at RBHSC) noted *"contacted by "* (incomplete entry) re-death certificate and that he had spoken to Dr Caroline Stewart who had been waiting for PM result. "*PM result in front of chart. Spoke with Dr Hanrahan." "Cause of death Cerebral oedema; dehydration; gastroenteritis*

555) A death certificate dated 4/5/2000 was completed by Dr Dara O'Donoghue with cause of death written as 1a,1b and 1c as in the order listed above. Dr Dara O'Donoghue is unable to recall specific details of the discussion with Dr Hanrahan but Dr O'Donoghue had contacted him to advise on completion of the death certificate. A representative of Lucy's family had contacted and as Dr Hanrahan was the consultant who had been caring for Lucy he asked for his advice. The notes indicate "*that he advised me the cause of death for the MCCD should be 1 cerebral oedema 2 dehydration and 3 gastroenteritis. I believe Dr Hanrahan asked me if I would complete the MCCD and I believe I agreed to do this".* Dr O'Donoghue also explains that he cannot recall any training advice or instruction on the completion of medical certification of death but he had previously completed a number of death certificates.[236]

556) The issue of the death certificate appears to have followed discussion between the clinicians. Dr Caroline Stewart answering reports she understood the working pathogenesis had been agreed with the paediatric intensivists Dr Crean and Dr Chisakuta (and Dr McKaigue). [237] Dr Crean reports his understanding that the death

[236] WS-284/1 Q 10;

[237] WS-282/1 Q22

had been reported to the Coroner but was not party to any discussion regarding completion of death certificate although he thought it possible that discussions did take place. He does not disagree with anything Dr Stewart recorded on the autopsy request form. [238] Dr Chisakuta , Consultant in paediatric anaesthesia and intensive care RBHSC , recalls [239]that he and Dr Hanrahan had agreed hospital post-mortem examination was indicated *"to get some answers"* because Lucy's death had been sudden and unexpected but he was not involved in discussion of death certification.

557) The Northern Ireland guidance on death certification issued later but in principle applicable in 2000 –see Annex D- sets out the requirements for a doctor completing a medical certification of death and acknowledges that in a hospital team the consultant under whom the patient was admitted is finally responsible for arranging for the death certificate but recognises that this can be a delegated function to a doctor who is fully trained. The guidance is clear that a death certificate should only be signed by a doctor who has been involved or attending in the care of the patient.

558) " There is no clear legal definition of "attended", but it is generally accepted to mean a doctor who has cared for the patient during the illness that led to death and so is familiar with the patient's medical history, investigations and treatment. The certifying doctor should also have access to relevant medical records and the results of investigations. There is no provision under current legislation to delegate this statutory duty to any non-medical staff.

559) In hospital, there may be several doctors in a team caring for the patient. It is ultimately the responsibility of the consultant in charge of the patient's care to ensure that the death is properly certified. Any subsequent enquiries, such as for the results of post-mortem or ante-mortem investigations, will be addressed to the consultant. "

560) Dr D O'Donoghue had been involved during the care of Lucy having prescribed some intravenous fluid and a drug dose and had seen Lucy after her death.[240]

561) Comment. In my opinion Dr Hanrahan was responsible for the issue of the death certificate but it was appropriate to delegate its completion to Dr O'Donoghue on his instruction.

562) Dr Hanrahan's opinion on potential causation of death in 2000

563) It is not evident that a detailed discussion was held between Dr Hanrahan and Dr Crean to consider causation of death before or after referral to the Coroner. In my opinion, if that had taken place Dr Crean could have shared his experience and

[238] WS-292/1 Q 9 10

[239] WS-283/1

[240] WS-284/1

knowledge and thus drawn attention to the hyponatraemia as a significant factor in the production of cerebral oedema because the Erne records show it had developed rapidly over a period of 6 ½ hours and after 4 ½ hours of IV No 18 solution.

564) Dr Hanrahan did not consider then that fluid overload might have been responsible for the hyponatraemia which might have been the cause of the cerebral oedema. Dr Hanrahan explains that in his experience hyponatraemia was a very common biochemical finding and could not recall hyponatraemia severe enough to lead to cerebral oedema; and, in his opinion the level in Lucy was not sufficient to cause cerebral oedema. Although the fluid balance chart was available to him at some time during the course of Lucy's admission, it was not available to him at the time of his initial review. He had not appreciated the high volume of low solute fluid given to Lucy at Erne hospital. He was aware of the fall in the blood sodium but he considered this only to be of mild degree. He was not apparently aware of the significance of a rapid fall in the blood sodium and how this could be related, regardless of the level, to causation of brain oedema.[241] It was known amongst the paediatric intensivists that a rapid fall of blood sodium could cause neurological problems and Dr Crean explains that he was aware at the time that acutely developing hyponatraemia could cause neurological decompensation. [242]This point is also included in publications available at the time and later in a text book by Dr Gale Pearson. (*Handbook of Paediatric Intensive Care 2002 Saunders* which states ***"****page 88 hyponatraemia causes ileus, listlessness and ultimately cerebral oedema and convulsions, the extent of the symptoms is more closely related to the rapidity of change in serum sodium than the absolute value. Hyponatraemia can occur in the face of normal total body sodium (SIADH, glucocorticoid deficiency, water overload et cetera) raised total blood sodium (heart failure, cirrhosis, nephrotic syndrome and sequestration) or decreased total body sodium (increased losses, diuretic therapy, inadequate intake). "*

565) Hyponatraemia from use of low solute IV fluids including No 18 solution is documented in paediatric texts (e.g. Forfar & Arneil 5th edition current in 2000 Page 412 see Annex C) and it is a deficiency that a review of the fluid regime used at Erne was not conducted while considering cause of death at this time. But the fluid balance charts available from Erne hospital are not easy to analyse and required a focused piece of time to do so.

566) Dr Hanrahan states that the fluid management did appear inappropriate that it is not clear whether this was a contemporary view or whether this is something he has concluded subsequent to the publicity given to the issues. The grounds on which this judgement was formed are not clear.[243]

[241] WS-289/1 Q4

[242] WS-292/1 Q 8

[243] WS 289/1 Q14(h)

567) In later correspondence-17/6/2003 in a letter from Dr Hanrahan about Lucy to Mr Walby associate medical director litigation management Royal Victoria Hospital, he states he did not have access to the fluid chart at the time he saw her. And *"subsequent events transpired indicate that cerebral oedema probably related to hyponatraemia related to gastroenteritis, was the cause of death*." He also stated that he was aware that Lucy had suffered from rotavirus and post-mortem had shown cerebral oedema and bronchopneumonia. [244]

568) LEAD CONSULTANT FOR LUCY

569) My views about the split of responsibilities in the paediatric intensive care unit are based upon experience working with colleagues in intensive care in Pinderfields hospital and later with regional intensive care units as part of my clinical practice; in my role as Honorary Secretary in setting up through the Council of the British Paediatric Association, (which later formed the RCPCH) the BPA review of paediatric intensive care published in the mid-90s; my membership of the Department of Health national review of paediatric intensive care and involvement in production of the national report PIC " *Framework for the Future* " 1997; in setting up and chairing the multidisciplinary working party and writing the Department of Health guidance on level I intensive care (high dependency care) in district general hospitals; in drawing up the specification, awarding the contract and sitting on the steering committee of the paediatric intensive care audit network PICANET (up to my retirement from the Department of Health).

570) Consultants in paediatric intensive care were largely paediatric anaesthetists who worked either full-time in PICU (only a few) or, (most) who mainly worked on a sessional /rota basis, the rest of their time being involved in emergency or planned surgical theatre lists. Background in training could either be through anaesthetic training, specialising in paediatric intensive care (most) or increasingly in the 2000s doctors trained in paediatrics who acquired expertise including additional airway skills and induction of anaesthesia and who worked largely full-time in PICU as a consultant paediatric intensivist and, such consultants did not carry out anaesthetic surgical duties. In the intensive care units support at junior doctor level was usually from either paediatric trainees rotating through for experience or trainees wanting to specialise in paediatric intensive care (a few) or from trainees in anaesthesia aiming for general paediatric anaesthesia or intending later to specialise in paediatric intensive care. It appears in Belfast that paediatric trainees were supporting the consultant anaesthetists on 14 April. The consultant anaesthetic sessional arrangements may be supported by an identified Director of the Intensive Care unit : one of the consultant paediatric intensivists. It remains be clarified whether Dr Crean was an identified Director which

[244] 062-034.

might explain his name appearing on the admission documentation and being logged as a responsible consultant in the papers of the audit mortality section in August 2000.

571) In many children's hospitals if a child was admitted under a consultant and then transferred to the PICU, the lead would often remain with that consultant rather than the intensivist, although the care would be shared. In contrast, a child transferred in from another hospital directly to PICU would usually be under the care of the intensivist, at least in the beginning. It may help to determine the practice in RBHSC at the time.

572) When Lucy was admitted to PICU at RBHSC this was as a transfer from another hospital. On admission to PICU, Lucy was seen by Dr McKaigue, who assessed her, planned supportive care and discussed with and handed Lucy over to Dr Crean and to Dr Chisakuta around 9 AM. [245] Her care was provided by Dr Crean from the morning of 13th of April until the evening when he went off duty[246]

573) On 14 April her care and supervision was by Dr Chisakuta who took over her care from the team overnight and conducted a ward round supported by a paediatric trainee. He was involved in the brainstem testing. Trainees in paediatrics and anaesthesia supported the consultant paediatric intensivists in RBHSC PICU. Dr Chisakuta describes Dr Hanrahan, consultant paediatric neurologist as "*looking after Lucy*" [247]

574) On the hospital administrative forms Lucy was admitted to PICU under the care of Dr Crean (061-001, 061-013). The nursing record shows consultants Dr Crean and Dr Hanrahan (061-025-083). On the laboratory request forms for 13 April and 14th of April the consultant listed is Dr McKaigue (061-033).

575) Dr Crean expressed the view that Dr Hanrahan was the consultant "*in charge of Lucy 's care* " [248]

576) Lucy was admitted "officially " under the care of Dr Crean, consultant in intensive care but managed jointly by him and Dr Hanrahan, paediatric neurologist. While she remained alive her lead in terms of her support would , in my opinion, remain with both Dr Crean and Dr Chisakuta in terms of stabilisation and withdrawal of therapy after brainstem tests. However her diagnostic care (and continuity throughout her care over the consultant rota changes in PICU) at that stage was from Dr Hanrahan.

[245] WS-302/1 Q6 (c) Q9

[246] . WS-291/1Q6.

[247] WS-283/1 Q1 (g); Q4 (b).

[248] WS-292/2 Q9(a).

577) After the brainstem tests were carried out Dr Chisakuta was informed by Dr Hanrahan that he intended to inform the Coroner of Lucy's death but Dr Chisakuta has no recollection of a discussion about this or any knowledge of the Coroner's office discussion but he was involved in the request for the hospital autopsy describing this as needed because the death was sudden and unexpected. Dr Chisakuta recalls he possibly agreed the pathogenesis set out by Dr Caroline Stewart but has no recollection of it. [249] Dr Hanrahan took the lead in managing processes, discussing with the Coroner's office, requesting the autopsy, issuing the certificate and interviewing parents, and communicating with Dr O'Donohoe at Erne hospital. He thus took on the role of responsible consultant. It was the responsibility of both Dr Crean and Dr Hanrahan, as clinicians involved in her care to attend the mortality audit review meeting (and arguably Dr Chisakuta although he did not know it took place [250]) together with Dr O'Hara the consultant pathologist who had carried out the post-mortem.. In my opinion, Dr Hanrahan was the lead consultant for diagnostic care, for post death management and review of causation of the brain oedema and, for producing a discharge summary.

578) Dr Crean however had reviewed the fluid management in Lucy and contacted Dr Donohoe in Erne about this. He was aware of the fluid regime and the hyponatraemia which was no longer present by the time Lucy arrived in RBHSC.

579) It would be expected that Dr Crean and Dr Hanrahan would discuss causation and come to some agreed pathogenesis even though the final diagnosis was not clear. During that discussion Dr Crean was in a position to know that a large volume of normal saline had been given at least (250 ml and possibly 500ml) had been given. Dr Crean cannot recall agreeing the working pathogenesis with Dr Stewart nor has any recollection of discussion about Lucy relating to the coroner's referral or death certificate. [251]There is no evidence that such discussions took place at the time of death nor at the time of death certification although Dr Stewart makes reference to an agreement with anaesthetists about the points she listed on the autopsy request form and lists Dr McKaigue as one of the anaesthetists working in PICU at the time, she does not state that Dr McKaigue was involved in the discussion. [252] Dr McKaigue went off duty after admitting Lucy and may not have had full knowledge of the fluid regime used in Erne. He had not given any consideration to the cause of Lucy's death and is unable to recall

[249] WS 283/1 Q 6(a);Q6(e); Q 13; Q14

[250] WS-283/1 Q 19(e)

[251] WS-292/1 Q9; Q12

[252] WS-282/1 Q22(a)

any discussion about her. [253] He attended the August 2000 mortality meeting but he may not have been in a position to assist discussion there [254]

580) Referral to Coroner

581) The case notes record the result of the conversation with Dr Curtis was that a Coroner's referral was not needed.[255]

582) On 14 April 2000 Dr Hanrahan did not have a clear understanding of why Lucy developed cerebral oedema. This is a rare complication of gastroenteritis which in turn is a rare cause of death. On clinical grounds he had not come to any other explanation for the cerebral oedema. Referral to the Coroner was appropriate in these circumstances especially as Lucy died only a short time after being admitted. Dr Hanrahan was directed by the Coroner's office to discuss referral to the Coroner with Dr Curtis, at the time assistant state pathologist in Belfast. Guidance in Northern Ireland at the time included the criterion for referral "*death occurred in other circumstances that may require investigation*" and the death was unexpected. (*Coroner's guidance 1995 Northern Ireland*) this was updated in 2008 [256]

583) When discussing with the Coroner's office, it is usual that a question is posed to the clinician by the Coroner or Coroner's officer "*are you prepared/feel able to sign the death certificate*". In many circumstances it is possible for the clinician to say" yes " when they have a clear understanding of the cause of death e.g. the illness had a significant mortality and the death was explained even if unexpected. Dr Hanrahan was uncertain of the cause of death and if asked that question should have said no. Neither Dr Curtis nor Dr Hanrahan can recall the conversation and neither made a record of it. Dr Hanrahan records that Dr Curtis agreed that a post-mortem was desirable but he did not agree the content of the death certificate with Dr Curtis.[257]

584) Dr Curtis states that at times he does advise that a death certificate can be issued. But "*I would also have had no reason to discuss the contents of such a death certificate. A death certificate is a matter for a clinician in conjunction with the Coroner's office".*

585) Dr Curtis points out that "*in general, it was (and remains) my practice to advise clinicians who may seek advice about a death certificate that if they have any worries or concerns they should speak to the Coroner. I would also suggest that if the death does*

[253] WS-302/1 Q9

[254] 319-023-003

[255] 061-018-067

[256] [INQ-0240-11].

[257] WS 289/1 Q11

not fall within the guidelines for referral to the Coroner, but they have any doubt about a cause of death that they should have a hospital post-mortem done-that seems to me to be quite reasonable and I note that it was in fact done in this case. I do not recall if I suggested it, or if it was said to me as something which was to be done." He is unable to recall whether the death certificate was discussed with Dr Hanrahan or what was to be included. Dr Curtis explains also that he would have had little or no need to communicate with the Coroner's office about the conversation he held with Dr Hanrahan.

586) Dr Curtis states "*I am able to say, however, having read the information contained within the autopsy request form, that had I had all of that information relayed to me I would have made two recommendations-the first would have been to refer the case to the Coroner and the second would have been it may be sensible to involve a chemical pathologist-or medical biochemist-to look at the results in the case. These recommendations would have arisen due to knowing about dehydration and hypernatraemia in the same patient. My understanding is that dehydration would normally lead to hypernatraemia so this would be an unusual situation which warranted investigation. I am therefore sure that I did not know these details at the time I have spoken to as I suspect I would have recommended the case be reported to the Coroner."*[258]

587) Dr Curtis states *"I suspect I reached the view that Lucy died of natural causes on being told that she had gastroenteritis which is a natural cause of death."*

588) Comment: gastroenteritis is an extremely rare cause of death in United Kingdom in 2000 and this should have alerted him to the unusual features of the case.

589) Mr Leckey,Belfast HM Coroner writing to Dr Sumner on 3 March 2003 requesting a report on Lucy provides a record of the discussion :

a. *"The death was reported to my office 14/4/2000. The office note gives a history of gastroenteritis, dehydration and brain swelling. On behalf of my office Dr Michael Curtis who is assistant state pathologist spoke to Dr Hanrahan, the consultant in charge at the Children's Hospital. Dr Curtis was satisfied that a post-mortem examination was unnecessary and the office note indicates that a death certificate was to be issued giving the cause of death as gastroenteritis. Apparently a post-mortem examination was then carried out by Dr O'Hara. This was not a Coroner's post mortem. In retrospect Dr O' Hara should have reported his findings to me and I would have made this a Coroner's case.*

590) Mr Leckey was not aware of that a post-mortem had been being carried out until he received Mr Millar's letter in 2003.[259]

[258] WS-275/1 Q12;Q7;Q8

[259] 013-058-343

591) It is not clear how the information about the content of the death certificate was provided to the Coroner's office. Dr Hanrahan states in respect of information given to the Coroner's office *"I have no memory of the details. It would appear that the information I gave comprised "gastroenteritis, dehydration, cerebral oedema" see 013-053a-290"* and explains that he did not discuss the completion of the death certificate with Mrs Dennison in the Coroner's office. In respect of the lack of knowledge of the Coroner's office about the post mortem Dr Hanrahan states re Dr Curtis "*the note records that he agreed that a post-mortem was desirable".* [260]

592) A hospital autopsy was requested because Dr Hanrahan remained uncertain of the cause of death. The autopsy request form listed the clinical problems considered by Dr Hanrahan. When available on 14 April 2000 (after the discussion with Dr Curtis) the preliminary autopsy report added no additional information and omits the hyponatraemia which the clinicians recorded as a problem. When issuing the certificate on 4 May 2000, Dr Hanrahan had the result of the preliminary autopsy report. At that point, in my opinion, Dr Hanrahan arguably was in no better position to issue a death certificate than at the time of the discussion with Dr Curtis. In my opinion another referral to the Coroner should have been made then and thus a death certificate not issued. Arguably the pathologist should have taken steps to do so also but it would be necessary to obtain the opinion of a pathologist on their responsibility in this matter for further clarification on this point especially when considering what was noted as said at Dr O'Hara's meeting with parents on 16 June 2000 meeting (which was not attended by Dr Hanrahan) – see below.

593) The entries on the death certificate were illogical unless the dehydration listed at 1b was made because Dr Hanrahan considered the <u>treatment</u> of the dehydration was the likely cause of the cerebral oedema. Dehydration itself does not cause cerebral oedema. Dr Hanrahan now questions how dehydration could have caused the cerebral oedema *"cerebral oedema was not due to dehydration, but rather to excessive rehydration leading to hyponatraemia.".* It is not evident when Dr Hanrahan formed this conclusion. If Dr Hanrahan was concerned about the treatment given in the Erne hospital, he should have referred to the Coroner and thus not issued a certificate.[261]

594) Dr Hanrahan wrote to the parents on 16 May 2000 (061-010) inviting them for a meeting in which he states it might be better to wait until he had the results of the autopsy. In the event he saw them on 9 June (that is before the final autopsy report) (061-018-069). The hand written entry relating to the meeting with the parents records "*they have met Dr O'Donohoe who did not have the notes. I went over the events concerning Lucy's death and encouraged them to attend Dr O'Donohoe to clarify events in the Erne. I will see them again if required."* He recalls that parents were unhappy

[260] WS 289/1 Q12 (iii) ; Q12 (v)P11; Q11 (d) P10

[261] WS 289/1 Q19(f)(iii) P20

about Lucy's treatment in Erne but he did not document the concerns. This should have been done in meeting good standards of record keeping. He does not remember what he told them but encouraged them to clarify events in the Erne hospital with Dr O' O'Donohoe because "*the sentinel event had occurred in the Erne, when Lucy collapsed. She was brain dead on arrival in Belfast. The events that led to her death, therefore, took place locally and I believe that Dr O'Donohoe should have been involved in the explanation to Lucy's parents*." He did not attempt to clarify events at the Erne hospital himself. [262]

595) Dr Hanrahan explains about whether he had concerns about events at the Erne hospital at the time of interview with Lucy's parents on 9 June 2000 *"the fluid management did appear inappropriate, both in the amount of solution 18 administered prior to Lucy's collapse and the size of the bolus of normal saline that she subsequently received. The cerebral complications were however due to hyponatraemia secondary to solution 18, the degree of which I was unaware that the time*". And when he states "*cerebral oedema was not due to dehydration but rather to excessive rehydration leading to hyponatraemia*." If this reflects his opinion at the time and influenced his entry of dehydration in the certificate, then arguably the certificate should not have been issued because the death was linked to treatment given. However it is not evident whether this was in his mind at the time. [263]

596) On 14/6/2000 Dr Hanrahan entered a record that he had contacted Dr J O'Donohoe who *"would rather wait for the PM report*." (Presumably before he wished to see parents again). Dr Hanrahan cannot recall the detail of the conversation, and therefore it is not known whether the cause of death as certified was given to Dr O' O'Donohoe at Erne. [264] Dr O'Donohoe confirms that he had a telephone discussion with Dr Hanrahan on 14th of June 2000 but is unable to recall whether any matters relating to the cause of Lucy's death were discussed. [265]

597) Training in death certification and Quality assurance of certificates

598) Training of junior doctors and consultants in completion of death certificates in the past been limited and this is referred to by Dr Caroline Stewart [266]when she describes the learning coming from observing others and being guided by senior doctors. Dr O'Donoghue's experience was similar in respect of training[267]. This is

[262] WS 289/1 Q14 P15 (d)

[263] WS 289/1 Q 14 (h); Q19 (f) iii

[264] WS 289/1 Q14 P16

[265] WS-278/1 Q14 P9

[266] WS-282/1

[267] WS 284/1

probably still the case today. In general paediatric practice and in many specialties of paediatrics, certification of death is not a frequent practice. In other areas such as neonatal or paediatric intensive care or in oncology, certification sadly is more frequent and in these specialties it is common practice to delegate the certification under supervision to a junior doctor. Many general consultant paediatricians choose to complete certificates themselves because of the relatively limited experience. Once issued, at the time and now, quality control of the content of death certificates is very limited although plans are in hand nationally to establish a medical examiner to review every death certificate issued because of a number of studies identifying poor quality of certification.

599) Discharge communication with Erne Hospital and GP

600) A full discharge letter was not provided to the general practitioner and copied to the Erne Trust as they had referred Lucy and could include cause of death and that a certificate had been issues and thus informed Erne Trust that no Inquest was planned. . Dr Crean confirms that he would not have written a discharge letter as this would be undertaken by the consultant paediatrician in charge of the case.[268] It was the responsibility of Dr Hanrahan. In 2000 it was usual practice for a discharge communication to be written in the form of a letter following up the immediate discharge note to the GP. Practice varied and a significant minority of district or regional hospitals did not do this for common conditions such as asthma, or viral infections. For more complex conditions or when a child dies it is a practice which should be in place because it serves a purpose of providing the referring hospital and general practitioner with information about their patient (and for children enables the GP to discuss management with parents) and to carry out any follow-up therapy or management that may be required. It also offers an opportunity for the treating team to review management of a particular case when composing the letter, usually produced a few days after the discharge, when results of tests are available which might not have been at the time of discharge. The letter may be written by a consultant or registrar and should include some detail of the presentation, investigation, treatment and diagnosis. The failure to produce the discharge letter is a significant deficiency.

601) <u>Final autopsy report Dr O'Hara 13 June 2000</u>

602) "…the autopsy also revealed an extensive bronchopneumonia. This was well-developed and well-established and certainly gives the impression of having been present for some 24 hours at least. Unfortunately swabs taken from the lines were unsuccessful and did not grow and there is no doubt that this pneumonic lesion within the lungs has been important as the ultimate cause of death, the changes being widespread throughout the lungs. The pneumonia could possibly prior to the original disease presentation but equally could have been induced during the time of seizure and

[268] WS-292/1. Q13

collapse. The changes seen in the brain are consistent with an acute hypoxic insult and there is no evidence of any underlying ineffective congenital or structural abnormality of the brain tissue."

603) In the clinical history refers to the clinical diagnosis of "dehydration and hyponatraemia. Cerebral oedema-acute coning and brainstem death. (Clinical history provided by Dr Caroline Stewart, specialist registrar, paediatrics, RBHSC). External appearance and body measurements. The body is that of a 12 kg infant measuring 73 cm in length."

604) ……. "no evidence of meningitis is noted."

605) Cerebral oedema was found at the autopsy together with bronchopneumonia and Dr O'Hara concluded that bronchopneumonia was important in the cause of death. The clinicians had not raised this diagnosis. There were no signs of bronchopneumonia when Lucy was admitted at the Erne hospital -there was no cough, no tachypnoea, no crackles in the chest and no evidence of respiratory distress in the form of chest indrawing or any other sign of undue respiratory effort: all of which are clinical features of bronchopneumonia. Bronchopneumonia can be found in children who have been ventilated even for as short a period of 36 hours and may be more likely in children who have had a period of hypothermia as Lucy had experienced during her transfer. A contribution may also have occurred from the fluid overload which can trigger development of bronchopneumonia by producing pulmonary congestion. There was no documentation of a hypoxic episode in the clinical course to explain the finding of features described. There was a respiratory arrest at Erne from coning secondary to cerebral oedema but no hypoxia was recorded.

606) Neither the provisional anatomical summary of 17 April 2000 nor final anatomical summary of 13 June 2000 [269] mention hyponatraemia although this had been listed as the third important clinical problem in the autopsy request form and mentioned in the clinical presentation and diagnosis section. This is a significant omission because no clear explanation was given about how cerebral oedema might have become present in Lucy. Dr O'Hara was not given information about the excessive volumes used but this does not appear to have struck the clinicians before requesting the autopsy. The RBHSC guidelines document states that if an autopsy is requested by a paediatric neurologist it is generally carried out by the neuropathologists. [270]But this process was not followed in Lucy.

607) The 12 kg weight recorded at the autopsy is likely to be an error but Dr O'Hara should have paid attention to the weight because Lucy weighed 9.14 kg at Erne

[269] 061-009-016

[270] 319-067a-031

admission yet weighed 9.8 kg on admission RBHSC a gain of 660 g equivalent to 660 ml fluid implicating fluid overload.

608) It is not evident that Dr O'Hara had sight of the Erne notes but these should have been included in the RBHSC notes and thus be at hand during his autopsy. Dr O'Hara did not mention hyponatraemia and did not provide an explanation (or appear to consider) how gastroenteritis could cause cerebral oedema. The only way was by fluid overload with or without dilutional hyponatraemia and thus Lucy's therapy was implicated and should have been reported to the Coroner.

609) **The significance of Cerebral oedema identified by the clinicians and in the autopsy.**.

610) The case records include sufficient information for a review for example by a pathologist or in an audit meeting to identify that fluid excess had been given and Dr Crean had specifically sought information relating to the fluids used. It was known that Lucy had hyponatraemia at Erne Hospital although this was not found at Belfast. The pathologist (like the clinicians) should have been aware of its relevance when making the diagnosis of cerebral oedema. No fluid overload at Belfast occurred. Lucy's body weight at autopsy was increased consistent with fluid overload. The cerebral oedema and pulmonary congestion reported by the pathologist suggests the presence of excessive fluid in the body. In a child who had presented with a degree of dehydration, as in Lucy, the only way that excessive fluid could be introduced over the 7 ½ hours in hospital before the seizure and collapse would be through an intravenous infusion. Hyponatraemia can occur in any dehydrating illness and be present at presentation. But Lucy had a normal blood sodium on admission and the low sodium developed after then following IV fluid treatment. The development of hyponatraemia in Lucy should have been recognised by the pathologist as a further marker of the use of high-volume low solute fluid inappropriate for her clinical state and consideration given in the autopsy report to this and /or SIADH.

611) It is not evident that the pathologist or clinicians reflected on the rarity of the death nor gave due consideration to the mechanism by which cerebral oedema might have occurred. Some consideration arguably could have been given to rotavirus encephalopathy which is not mentioned in any of the discussions but is a recognised although uncommon condition. (*M. Iturriza-Gomara et al , Rotavirus Gastroenteritis and Central Nervous System (CNS) Infection: Characterization of the VP7 and VP4 Genes of Rotavirus Strains Isolated from Paired Fecal and Cerebrospinal Fluid Samples from a Child with CNS Disease. Journal Of Clinical Microbiology. 2002; 40 :. 4797–4799.*) There is no record of CSF being sent for viruses. Dr Hanrahan investigated the other causes of acute encephalopathy but with negative results. The results found on imaging with CT and with EEG confirmed the presence of cerebral oedema and brain death.

612) Meeting of Dr O'Hara pathologist with parents 16/6/2000

613) On 16/6/2000 Dr.O'Hara met Stanley Millar and Mr and Mrs Crawford at the Royal to explain the outcome of the post mortem that he had performed. . The notes of the meeting made by Mr Millar record the following issues as having been discussed:[271]

"The PM was not under the Coroner's Act

The cause of death is less frequent than in years past and would not be common

Lucy probably died in Erne …

Dehydration was an important factor

Children can 'crash' very quickly due to dehydration and delay in getting in fluids could be crucial …

Dr. O'Hara conducted the PM at the request of Dr. Hanrahan …"

614) Comment: When Dr O'Hara commented that there was a delay in getting fluids, he was identifying a failure in standard of care which should arguably have been reported to the Coroner. There was no ' crash' in terms of circulatory collapse associated with dehydration at Erne. I advise an opinion on the responsibilities of a pathologist in this situation should be obtained from a pathologist.

615) COMMENTS ON RBHSC APPRECIATION OF THE SIGNIFICANCE OF THE HYPONATRAEMIA

616) The following considers reasons for the need to appreciate the role of the fluid regime and hyponatraemia in Lucy's illness after death. It is also relevant to any consideration by an External wider review in 2000 if one had been set up and information which might have influenced any conclusions which a review might have made.

617) Lucy was in coma on admission to RBHSC. When admitted at Erne hospital she was not in coma and her blood sodium was normal at 137mmol/l. When she developed a respiratory arrest blood sodium was recorded in the Erne hospital as 127mmol/l after large volumes of hypotonic and isotonic saline had been given over 4-4 ½ hours. The (probable) first laboratory blood sample in RBHSC was 145 mm/L at around 0900 hours. (Sequence of RBHSC biochemistry results on 13 April shows the first lab number – R003235567- reporting 145mmol/l 061-033-108). Between Erne hospital blood test and admission to RBHSC, a small volume 30ml/h normal saline had been infused in addition to the rapidly infused large 250 ml (and possibly up to 500ml) volume of normal saline.

[271] 015-006-031

618) Low blood sodium is a common finding in coma of any cause in paediatric practice. The level was marginally out of range only to an extent common in any acute childhood illness and which usually is tolerated.

619) A blood sodium level at 127mmol/l was not usually regarded as *causative* of cerebral oedema in the year 2000 (**see Notes [1] to [3] below)** although many intensivists and some paediatric neurologists were aware that a rapid fall could make worse an acute encephalopathy whatever its cause. This was not necessarily widely known in paediatric practice. In general paediatric practice there was awareness of risks from rapidity of fall in blood sodium when managing (the not rare) hypernatraemic dehydration in gastroenteritis when a very high blood sodium is corrected too swiftly leading to encephalopathy from rapid changes in the osmolality in the blood and brain. Cerebral oedema is also a complication of diabetic ketoacidosis which can also result from rapid changes in blood osmolality and acidosis such that fluid management with careful slow reduction in blood glucose avoiding too rapid a change in blood sodium aims to prevent this happening. Linkage of blood sodium to this risk is explained in *Changes in serum sodium levels during treatment of hyperglycaemia Oudesluys-Murphy et al. Arch Dis Child 2003;88:647-8*.

620) Because the second blood sodium sample in Erne hospital followed a large infused volume of normal saline (at least 250 mls and possibly up to 500ml) it is a possibility that the blood sodium level had been much lower reaching the range recognised at that time to be causative of brain oedema.

621) In the absence of any other satisfactory explanation for Lucy's death a review by RBHSC of the fluid management in Erne hospital was justified. RBHSC had copy Erne case records. Careful scrutiny of these records shows that 100 mL/hour of low solute fluid number 18 solution had been given over 4-4 ½ hours followed by an excessively high volume of normal saline given over one hour. Thus it was evident from review of Erne fluid management that Lucy had been overloaded with fluid. This in itself can arguably contribute to or cause cerebral oedema even without hyponatraemia. (It can also cause pulmonary oedema which was reported in the preliminary autopsy report on Lucy). But this view of mechanism in Lucy reported in the 2001 and 2002 reviews in Erne and for the Inquest has been challenged in oral hearings in January 2013 by Prof Kirkham and I comment on this in **Note [5]** below.

622) In my opinion it is understandable that a detailed fluid review of Erne therapy was not carried out in the intensive care unit during Lucy's life. The opportunity to do so was after Lucy's death. This arguably should have been done when considering referral to the Coroner and at death certification. Dr Crean and Dr Hanrahan should have reviewed the fluid regime used in Erne as a minimum after Lucy's death. Arguably the greater responsibility lay with Dr Hanrahan to do this because he was involved in diagnosis of causation and care and management after Lucy had died. Dr Crean had supported Lucy while she was in PICU when it is possible that a detailed pre-admission fluid review is not likely to have significantly altered his regime during her stay on PICU. I have

provided information on respective consultant responsibilities in paragraph 569-580. Although several clinicians reviewing the Erne case records identified difficulty in interpreting the fluid record it was evident that volumes of low solute fluid used were too high as was the volume of normal saline and both volumes were given too rapidly for her condition.

623) A further opportunity to review Lucy's fluid regime would have been on consideration of the first autopsy report and issue of the death certificate and another on review of the final autopsy report of 13 June by Dr Hanrahan but, although he had requested the autopsy, he did not consider this report. And yet another when preparing for the August RBHSC mortality review which neither he nor Dr Crean nor the pathologist Dr O'Hara attended. Preparation for an audit meeting often entails review of published literature which might have included the Arieff paper but that this was not done. It is thus in my view a significant failing on the part of Dr Hanrahan that he did not seek further an explanation of Lucy's death- especially when gastroenteritis was complicated by cerebral oedema - a very rare event. I consider that his lack of appreciation of the significance of both level and rate of change of the blood sodium was in part understandable in the context of knowledge at the time (see note [4] below) but if he had conducted a fluid management review and referred to texts and published literature it would have been clear to him that the fluid types and volumes used had probably been contributory or causative.

624) **NOTES**

625) **Note [1]** In the APLS Handbook relevant at the time (second edition) (See Page 38 in Annex C) the following is stated about management of hypernatraemic dehydration:

626) "*In patients with a low or normal sodium lost fluid can be replaced over 24 hours. In hypernatraemic patients it must be replaced over at least 48 hours and sometimes longer depending on the severity-the higher sodium the slower the re-hydration must be. If the sodium and water are corrected too rapidly in the extracellular space, water will pour into cells, and if this happens in the brain, cerebral oedema and even death may occur."*

627) **Note [2]** The major UK Paediatric Textbook of Paediatrics Forfar and Arneil (5th edition relevant at the time) provides guidance on fluid management of gastroenteritis in several chapters : in the fluid section, the section on disorders of the alimentary tract and liver and, in the infections section. In respect of hypernatraemic dehydration Text page 453 in annex C page 44 the following text appears

628) *"If a child is severely shocked intravenous fluid in a volume of 15-20 mL per kilogram should be given rapidly over 10-15 minutes. In less severe cases it is more usual to give 40-80 mL/kilogram over four hours as 0.45% sodium chloride/2.5% dextrose although some of this fluid may be given as plasma protein solution.*

629) Once the child has been resuscitated, rehydration should be planned over the next 24 hours for 5% dehydration and over 48 hours for 10% dehydration. ……..

630) *In children with hypernatraemic dehydration rehydration should continue over 48 hours to prevent sudden fluid shifts which might precipitate convulsions. The rehydration is similar to that described above for normonatraemic infants except that after an initial period of half strength (0.45%) saline this can be reduced to one fifth strength (0.18%). Where hyponatraemia is present the total body sodium may paradoxically not be depleted and therefore 0.18% normal saline can be used for both rehydration and maintenance. However when the sodium falls below 115 millimoles/litre extra sodium may be required."*

631) Forfar & Arneil (p412) also includes the following on water intoxication. (see page 43 Annex C)

632) "The finding of a normal plasma sodium concentration excludes the presence of water intoxication."

633) <u>Note [4] Relevant Emerging Literature</u>

634) It has been recognised for some time that a common cause of low blood sodium found in hospitalised patients is inappropriate use of intravenous fluid. It is also known that small drops in blood sodium can be particularly significant in children who have acute disorders of the brain. Less prominence had been given up to 2000 generally to rapidity of fall of blood sodium as a risk factor for development of acute encephalopathy otherwise except in hypernatraemic dehydration and to some extent in diabetic ketoacidosis although in the latter it is changes in osmolality generally which was seen as a risk factor including sodium. In paediatric neurology practice the significance and avoidance of even small changes in blood sodium levels is considerable when managing coma.

635) Wide awareness of hyponatraemia causing an acute encephalopathy during routine use of intravenous fluid in hospital has only been from the early 2000s. Since then there has been greater recognition that routine use of hypotonic fluids in patients who have impaired ability to excrete free water such as the post operative state, volume depletion and pulmonary and central nervous system diseases can cause cerebral oedema.

636) The Arieff et al 1992 paper was used by RBHSC in publicity related to Adam Strain's death, *Hyponatraemia and death or permanent brain damage in healthy children. BMJ 304: 1218-1222* and drew early attention to this issue. The review by *Halberthal et al Lesson of the week: acute hyponatraemia in children admitted to hospital : retrospective analysis of factors contributing to its development and resolution.BMJ 2001;322:780-782* points out that excessive fluid administration is a risk factor for the development of hyponatraemia. Many of the neurological complications

from hospital acquired hyponatraemia resulted from fluid administration well in excess of that recommended by Holliday and Segar in 1957 which is the basis for the routinely advised regimes up to early-mid 2000s. Cases with gastroenteritis were reported from 2001 Halberthal paper which appeared after Lucy's death. In the Arieff BMJ 1992 paper 16 cases were reported 13 of whom had surgery and 3 had infections 2 with tonsillitis and 1 with pneumonia. The Arieff paper concluded that

637) *" generally healthy children with symptomatic hyponatraemia (101-123mm/l) can abruptly develop respiratory arrest and either die or develop brain damage. "…….*

638) *"The hyponatraemia in these children seems to have been caused by extensive extra renal loss of electrolyte containing fluids and intravenous replacement with hypotonic fluids in the presence of antidiuretic hormone activity."*

639) Other than in hypernatraemic dehydration and diabetic ketoacidosis, the risk of rapidity of fall in blood sodium is not highlighted in any readily available textbooks although discussed in some research papers including older papers referred to by *Duke T, Molyneux EM. Intravenous fluids for seriously ill children: time to reconsider. Lancet 2003;362:1320-23* and also i*n Halberthal et al BMJ 2001.* Arieff even states

640) *" neither the actual concentration of serum sodium nor the rapidity of development of hyponatraemia seemed to predict the ultimate outcome…"*

641) Halberthal et al report that the volume of maintenance fluid given in 16/23 cases of rapidly developing hyponatraemia was 50% greater than recommended values And that infusion of hypotonic fluids increase the risk of acute hyponatraemia and brain swelling. The following content is relevant to understanding likely mechanisms in Lucy: (my emphasis in bold)

642) *"Serious symptoms may become evident when hyponatraemia approaches 120 mmol/litre but there are cases where symptoms become evident with a higher plasma sodium concentration, whereas others tolerate this electrolyte disturbance without developing seizures. Apart from underlying conditions that might make a patient more susceptible to seizures,* ***a possible important factor could be the extracellular fluid volume of the brain. If this volume was expanded by a large infusion of iso-tonic saline, a higher intracranial pressure might be present at a given degree of hyponatraemia.*** *Moreover because there is a relatively larger proportion of brain cell volume extracellular fluid volume in young patients, they are more vulnerable to an increase in brain cell volume."* [NB 0.9 % normal saline is isotonic]

643) A paper published 6 years after Lucy's death: *Neville et al. Isotonic is better than hypotonic saline for intravenous rehydration of children with gastroenteritis: a prospective randomised study Arch Dis Child 2006; 91:226-232* addressed the most appropriate use of intravenous fluid for treatment of gastroenteritis noting that there was

no consensus on the most appropriate electrolyte composition of intravenous fluids with recommendations ranging from 0.45 % to 0.9 %. Hyponatraemia was common at presentation (36%) in the 102 children in their study. They highlighted that osmotic inappropriate ADH activity is common and was persistent in children with gastroenteritis (*Neville at al High antidiuretic hormone levels in children with gastroenteritis. Pediatrics 2005;116:1401-7)* and that in children with gastroenteritis the use of hypotonic fluid exacerbates the tendency to develop hyponatraemia whereas the use of isotonic saline is protective. (0.45% saline is hypotonic).

644) This view is supported in *Powell KR, Sugarman LI, Eskenazi AE, et al. Normalization of plasma arginine vasopressin concentrations when children with meningitis are given maintenance plus replacement fluid therapy. J Pediatr 1990;117:515–22*. Which suggests that administration of sodium results in a more rapid return to normal of antidiuretic hormone concentrations than does use of low sodium-containing fluid. (in a study in which Arginine vasopressin was measured).

645) **<u>NOTE [5] Commenting on</u> evidence in Inquiry hearings on January 14th and 15th 2013.**

646) I note that differences have emerged between opinions of Professors Rating and Kirkham about mechanisms by which cerebral oedema may develop after infusion of hypotonic fluids leading to excess free water load and in relation to the evidence relating to rapid fall in sodium being a factor.

647) On P52 14 Jan the Chairman summarises Prof Kirkham's view that she believes there is no evidence in the literature that infusing a high volume of free water or developing a low sodium over two or three hours, either separately or together, overwhelm the brain.

648) I note that in Prof Kirkham's view cases that developed cerebral problem had another risk factor. She considers that children who become hyponatraemic can seize and can develop cerebral oedema but "*to develop the cerebral oedema and die of it, all of the cases that I reviewed appear to have had a second factor which had not been fully investigated*". She considers one factor- hypoxia might impair brain compensatory mechanisms and that if there was an impaired excretion of water by the kidneys, cerebral oedema could lead to cerebral herniation (P75 Jan 15).

649) Prof Kirkham (P54 14 Jan) considers that dilutional hyponatraemia could have *contributed* to cerebral oedema. But she considers that even if a low blood sodium results from a speedy administration of excess free water she has not seen evidence that there could be a progression towards raised intracranial pressure with brain shift and brain herniation.

650) **My observations on this debate relevant to Lucy Crawford**.

651) First I wish to state that I may not have understood Prof Kirkham's position correctly and am only basing any comment on this exchange of views in the hearing as they bear on Lucy. These are significant qualifications because I am neither an academic nor a clinical scientist nor am I familiar with the research papers in this area other than those relevant to clinical practice in the late 90s and early 2000s and with publications relating to hyponatraemia associated with use of hypotonic intravenous fluid in children in clinical studies in the 2000s to which I refer.

652) I note that there was discussion about whether an additional factor may be a limited ability of the kidneys to excrete a water overload. There was no reference made in this discussion to the role of excessive secretion of ADH as another factor to be considered. (Although it may well be present in expert reports). This is relevant to Lucy because of reports of a significant proportion of children with gastroenteritis who have SIADH. And that use of higher concentrations of IV sodium in use can suppress this. ADH acts on the kidney and reduces excretion of water.

653) From a clinician's viewpoint there is considerable variation in children in the way that they respond to diseases and trauma : some recovering, and some dying given a similar degree of illness or injury. This observation, empirical in nature, suggests that there is inherent variation in the way children's physiological systems respond including the theoretical potential for some children to have compensatory mechanisms which are overwhelmed when most would compensate when challenged e.g. with a high volume of infusion of hypotonic fluid. The following points relate.

654) Use of hypotonic intravenous fluid has been common particularly up to the early 2000s and, in the light of the paper which I quote below *Armon et al, 2008* it is likely that a significant proportion of the many children receiving intravenous fluids in hospitals in United Kingdom in any year will receive hypotonic fluid and some in excess of what is required. Many of these children will have gastroenteritis and a proportion hyponatraemia. Yet cerebral oedema complicating intravenous hypotonic fluid is rare as are deaths from gastroenteritis. This suggests that those who developed a severe fatal complication may form a subset of children in whom individual compensatory response may be impaired.

655) Lucy was given a significantly high volume of intravenous hypotonic fluid well outside the range of practice advice at the time over a period of 4 ½ hours. This was then followed by a very high volume of normal saline at least 250 mL and possibly 500 mL. *Halberthal et al* suggest such an infusion of isotonic fluid could trigger serious and even fatal cerebral oedema.

656) The empirical clinical observations which have been reported in the literature link hypotonic fluid infusion with deaths in children.

657) In conclusion in the circumstances relating to Lucy, it is, in my opinion, difficult at this stage of knowledge to refute the suggestion that the therapy she received at least contributed to even if it did not cause the cerebral oedema from which she died.

658) **CHRONOLOGY OF AWARENESS OF HYPONATRAEMIA RISK IN 0.18% Saline (No18solution**). **AFTER 2000**

659) It was only from the early 2000's that the risk of developing cerebral oedema with hypotonic fluid administration used in routinely use volumes has been widely recognised as a probable contributor to cerebral oedema in some children. Up to that time, number 18 solution was in routine use in children's units for intravenous maintenance therapy : for example, in a child who was unable to drink or receiving intravenous antibiotics. It was also in routine use for prevention of dehydration, for example in a child who is vomiting. In treatment of dehydration, where maintenance and replacement volumes were required, number 18 solution was also used in many units for mild dehydration if the initial blood sodium was normal although guidance favoured use of 0.45% or 0.9% saline especially where continuing or excessive electrolyte loss occurred say through diarrhoea.. In more than mild dehydration, guidance advised use of 0.45 or 0.9% saline and this was used in most, if not all, units. In severe dehydration nearly all units would use 0.45% or 0.9% saline.

660) In my hospital Paediatric Department in 2000 practice was as above. It was only from late 2001 following investigation of an incident of a child who had been vomiting after a mild head injury who developed hyponatraemia and coma from cerebral oedema while on maintenance volumes of number 18 solution, that we proposed a change in our practice in the paediatric unit and withheld No 18 solution from use other than on instruction from a consultant. I issued guidance in January 2002. All surgical and anaesthetic staff were circulated with the new policy which was adopted gradually over the ensuing 4-6 months after gaining agreement with surgical and anaesthetic colleagues to change prescription of number 18 solution across surgical and anaesthetic specialities and 0.18 % saline could be withdrawn from the children's wards, restricted to consultant use only. The routine IV maintenance or postoperative fluid selected for use was to be 0.45 % or 0.9 % sodium chloride with a range of choices for additional concentration of potassium. In Annex C Pages 57-58 I provide the analysis we conducted to check on the effect of introduction of the guidance.

661) In RBHSC : The *Paediatric Medical Guidelines RBHSC July 1999 were current in 2000 and* advised *" "With normal serum sodium treat shock if present. Then use 0.18% saline + 4% dextrose as the intravenous fluid. Fluid may be replaced over 24 hours."* (see Appendix C; P 55/56 for the copy of the page.) [272]

[272] 319-067a-091

662) Intravenous fluid usage in 2000s

663) Armon K et al. Hyponatraemia and hypokalaemia during intravenous fluid administration.Arch Dis Child 2008;93:285-287

664) This paper reported a snapshot of one day in 2004 including 10 district general hospitals and 7 university teaching hospitals with some children in paediatric intensive care units. Comparison was made with the guidance on maintenance fluids in the APL S manual 4th edition recommended also in the British National Formulary for Children.

665) 99 children in the sample were receiving IV fluids. 55 % under general paediatric care, 25% under surgical and 17% under haematology/oncology teams.

666) Hypotonic fluids were received by 78% (The hypotonic fluid group included those receiving 0.45% saline)

667) 16% received more than 20% of calculated volume requirement and only ¼ were documented as dehydrated.

668) 21/86 were hyponatraemic but with levels no less than 127 mmol per litre of whom 71% received hypotonic fluids and 6 received more than 120% of calculated requirement.

669) The table 1 shows that 10/99 received 0.18% saline used in 10/ 77 receiving hypotonic fluids.

670) Drysdale S et al. The impact of the National Patient Safety Agency intravenous fluid alert on iatrogenic hyponatraemia in children. Arch of Dis Child 2010;95:A46;

671) These authors report a study to assess the impact of the NPSA alert on maintenance fluids and the development of hyponatraemia. Practice and outcome children receiving intravenous fluids in June 2007 before the guideline implementation were compared with a sample from June 2008 after guideline implementation.

672) Before implementation of the guideline of the 44 children receiving maintenance intravenous fluid, 6/44 received either 0.18% saline or 5% dextrose and one became hyponatraemic.

673) After implementation 56 children received maintenance IV fluids and one received 5% dextrose and became hyponatraemic.

674) Comment: This study was from Kings College Hospital London- a university teaching hospital - showing that even in 2007, 0.18% saline was still being used for IV maintenance therapy with 14% of those being treated received fluid of <= 0.18% saline. .And that change in policy there only happened after the NPSA alert.

675) It is possible to conclude from these 2 illustrative reports that change in policy from traditional practice was slow even given the attention in Northern Ireland. The Armon study paper indicates that a significant proportion of children on intravenous therapy received hypotonic fluid and also in volumes greater than they required. If this was representative across United Kingdom, given the scale of intravenous therapy used in hospital (increasing over the last decade) then it is possible to deduce that the complication of cerebral oedema is rare even when children are exposed to greater volumes of hypotonic fluid than they need.

676) LATER EVENTS

677) RBHSC audit and mortality meeting 10th August 2000

678) Lucy's death was discussed in the mortality section of the monthly RBHSC audit meeting 10/8/2000 chaired by Dr Robert Taylor consultant paediatric anaesthetist in RBHSC.[273] From December 1996, and at the time of Lucy's death, Dr Taylor was coordinator of the audit programme in the Children's Hospital.

679) As audit coordinator he explains he was responsible for ensuring that clinicians were given the opportunity to present clinical audit projects that they had completed at monthly audit meetings.[274]

680) Aim and purpose of the mortality meetings RBHSC

681) Dr Taylor reports ". *the goal of these meetings was to discuss every child's death for learning purposes among the clinicians present*. He points out that it was not his role to investigate deaths in the Trust which would be a Trust matter. [275]

682) Dr Taylor describes audit as "*comparing your outcome to a guideline or a publication or some sort of standard at one can find*." [276]

683) Process

684) Dr Taylor has explained the process in his witness statements and oral hearings of the Inquiry on Claire Roberts

685) Every death was discussed in these meetings. The meetings were organised by the PICU/Audit secretary who would assemble the papers, identify the responsible lead

[273] 061-038-123

[274] C.Roberts hearing 11 Dec 2012

[275] WS-280/1

[276] C.Roberts hearing 11 Dec 2012 P112 Para16

consultants and a pathologist (if a post-mortem had been done) and schedule their attendance when they would answer any questions raised by the clinicians present.[277] Dr Taylor explains the process for the mortality review.

686) *"Each case presentation could have a time limit and the consultant supervising the case should have the opportunity to express problem areas in the management of the case and in a non-hostile environment and those presenting cases should indicate to Dr Taylor how long they will require."*

687) Up to 3 cases would be discussed in the hour. The audit would be multi-professional. [278]

688) The lead consultant would be identified amongst those involved. The presentation would usually follow the pattern of *"chronological discourse of the patient's past medical history, reasons for presentation for the final illness, as it were, then the issues around the death and the cause of death, any x-rays, CT scans, blood results and investigations and autopsy reports or coroner's inquest reports would all be presented in order*." And "…. *The review is really following the investigation, it is an element of postgraduate learning that is done to a mixed audience…."* [279]

689) He would expect whoever presented the clinical records to have worked their way through the clinical notes before the meeting in order to inform the presentation. It would be usual for letters and materials from the transferring hospital to be present at the audit meeting together with any RBHSC post mortem report and notes. It was not usual practice for the complete medical records from other hospitals to be transferred with the patient to RBHSC but summary records, and x-rays and investigations would be included.

690) Copies of the clinical records were not handed out or circulated amongst other clinicians attending the audit meeting or reviewed by another clinician but presented visually e.g. on an acetate by the consultant in charge of the case or a junior doctor who would make a summary from the case notes. It was the expectation that for each death all relevant materials would be presented. "*the mortality meeting was not minuted so that clinicians could speak openly."* And any questions raised by others present would be addressed. [280]

691) Dr Taylor considered that in the mortality meetings "..*(the deaths) weren't audited. Clinical audit is, as far as I described, you pick a national standard, whatever*

[277] WS-157-2

[278] C.Roberts hearing 11 Dec 2012 P119; P134; P135

[279] C.Roberts hearing 11 Dec 2012 P121; P116; P118

[280] WS-157-2, Q25(b)

that may be,…….. and you compare practice to the practices in publication." And "*The mortality was a review of the death, it wasn't an investigation into the death. It is a review of the finality of the final statements reports*".

692) Dr Taylor described his experience in Canada where mortality presentations were open to discussions of clinical disagreement and heated exchange of views. Dr Taylor's experience was that he would expect an exchange of views. He describes the lack of minuting of such discussions as practice at the time adopted to encourage free expression.

693) Dr Taylor states *"the mortality section of audit is not an audit of the clinical records, it is not an investigation of the death: it is a review following the completion of any investigation that has been undertaken and the finality is presented to the consultants the purpose of learning from that death*." Further, it is *"not an examination of the death; it's a review of the cause of the death in the Children's Hospital so that the doctors may learn that the case has been concluded and this is the final outcome of the cause of death. That helps to educate the doctors present that a child with diabetes or hyponatraemia has died within the hospital*."

694) Dr Taylor explained that the audit is a report to the meeting that a child has died and this is the cause of death. [281]

695) Outcomes / Output of Audit Meetings

696) Minutes and attendance lists were typed up by the PICU/Audit secretary who sent them to the Trust Clinical Audit Department.

697) Dr Taylor explained that he produced a few audit minutes, "*where I did write down lessons that had been learnt*".

698) He cannot recall any procedure for disseminating results learned from the death of a child to clinicians other than among those who attended a specific meeting. [282]

699) Dr Taylor gave examples of things that change because of the mortality meetings. After several cases of meningococcal disease, guidelines were issued because items were identified following mortality reviews and triggered this action.

700) He explains that an adverse event investigation would go through the clinical records in a more detailed way. And explains "*audit is not really a benefit in terms of mortality review. Audit is a system, as I have is explained, of looking at the macro-*

[281] C.Roberts hearing 11 Dec 2012 P126, P127&P128

[282] WS 280/1 Q 3 (r) P5; WS-280/2 Q2 P2

looking at the larger numbers of patients coming through the service and comparing that to national standards." Then he referred to use of CEMACH and PICANET. [283]

701) Lucy Crawford Mortality review August 2000

702) Documentation has been provided to the Inquiry relating to the meeting including the attendance sheet, minutes and an audit list which is redacted. The audit department has no documentation touching upon the mortality section of the audit meeting. Thus it is not evident what conclusions were drawn.[284] (Dr Hanrahan reported minutes were not taken in audit meetings. [285]) Dr Taylor has no memory of the issues discussed and cannot recall if any conclusions were reached. The death certificate would usually be given to the funeral director and is not retained in the patient records. He was not aware if there were any communications or discussions with Erne hospital staff nor aware of any changes which occurred in the audit process following her inquest as he was no longer chair or paediatric audit coordinator after January 2003. (Comment : Death certificate details were recorded in Lucy's case notes by Dr D O'Donoghue and the full Erne notes were available in RBHSC [286]).

703) The attendance list notes 34 attendees including consultants, registrars and SHOs both in medical and surgical specialties. Dr Taylor, Dr McKaigue, and Dr Hicks are listed. There is no indication that Dr Hanrahan, Dr Caroline Stewart, Dr Crean or Dr O' Hara were present.

704) It is not evident who presented the case of Lucy. Dr Crean, Dr Hanrahan and Dr O'Hara the relevant consultants, were not recorded as present. Dr McKaigue, who had accepted the transfer and saw Lucy on admission to PICU, was present.

705) The minutes record that five cases were presented in the mortality section and discussed. And also that the audit process was discussed. On a separate sheet, Lucy Crawford is listed linked with Consultants - ICU Dr Crean, PM Dr O'Hara. [287]

706) In response to further enquiry on 11th of March 2013, DLS confirms there are no Trust records to confirm what steps were taken to contact the responsible consultant and pathologist to attend the audit meeting, nor that the Trust is able to confirm that Dr

[283] C.Roberts hearing 11 Dec 2012 P129; P 138 & P 144

[284] 319-023-001 DLS,

[285] WS 289/1 Q 26 (d) (iv) P23

[286] 061-018-068

[287] 319-023-003; 319-024b-001; 319-023-004; 319-023-005,

Crean and Dr O'Hara attended. No other records were made of the meeting than those provided.

707) Dr Hanrahan confirms arrangements as described and thought Lucy's death was discussed at a mortality meeting. He believes that Dr O'Hara was likely to be present at the meeting. He cannot recall whether he provided any information to the meeting. [288]

708) RBHSC IT Support for Audit

709) Dr Taylor reports a PIC computer database was developed in the 1980s and used for clinical audit in the PIC Department. It was not supported by Trust IT Department. Data was entered by the doctors and secretary. Cases of hyponatraemia admitted to PICU between 1991-2001, were used as the basis of his presentation prepared in 2001 but did not include data from 1995 -1996 and states that the presentation was not taken forward in the hyponatraemia working group. The computer search was done by the PIC secretary. [289]The death identified was Raychel Ferguson. He believes Lucy's name was not identified on the PICU computer database as being due to hyponatraemia. Dr Taylor reported that the PAS " *was not very useful for the purposes of clinical audit as it did not contain sufficient or sufficiently accurate information".* [290] In the oral hearing related to Claire Roberts Dr Taylor states "*The PAS is basically administrative….. It does not contain important data such as.. Clinical codes; it's purely an administrative system. So that's not really of much use to me although you can get sometimes rough statistics about the number of admissions in the month or over a year…"* Dr Taylor referred to a clinical coding system CHKS which entered clinical codes. [291]

710) Dr Taylor does not appear to have been aware of the full functionality of the hospital coding and PAS for identifying patients with specific diagnoses. In the hearing Dr Taylor was given the clinical code used for Claire Roberts (302-153-003) on the hospital PAS system which included the terms "hypo-osmolality" and " hyponatraemia." And he explains that he did not understand that the PAS system could be useful for that.

711) The Trust informed the Inquiry that the PAS Coding was by coding clerks without reference to clinical staff. The clinical coding related to Lucy Crawford recorded that she died, a post-mortem was held, that the primary diagnosis was cerebral oedema, and,

[288] WS-289/1 Q24;&Q25

[289] WS-280/1 Q5

[290] WS-157-2, Q4 (d)

[291] C.Roberts hearing 11 Dec 2012 P151

subsidiary diagnosis was intestinal infection unspecified.[292] Hyponatraemia was not coded.

712) **PICU coding :** A RBHSC PICU Coding form document on Lucy was signed by Dr McKaigue 13/4/00 and listed the following: Seizure, respiratory arrest, cerebral oedema, brain stem coning, intubated, ventilated, central line, arterial line, CT scan, hyponatraemia, hypokalaemia, diabetes insipidis. The covering letter 319-019-001 providing this from DLS points out that the form does not record the coding of the cause of death but is used for recording admission details together with treatment in PICU.[293]

713) Comment: It would be helpful to know whether the codes which appeared on the PICU coding form were entered into the PICU audit database and, if they were, why it was not possible for Dr Taylor to identify Lucy as one of the children with hyponatraemia when enquiring of the system in 2001.

714) **Formal investigation of Lucy's death in RBHSC**.

715) Dr Taylor has explained that any formal investigation of deaths was the responsibility of the Trust and not the audit meeting. Formal investigation of deaths by the Trust would only follow if a death was regarded as unexplained or seen to be as a result of treatment. I provided a review of the chronology of adverse events reporting in children and their investigation in Annex D to my Claire Roberts report and attach this as a separate Annex to this report.

716) My comment : It appears that Lucy was not regarded in the latter category and thus no notification of a concerning death was made to the Trust. It may be that it would only be reported if thought to be related to care in RBHSC rather than the referring hospital. Dr Steen in Claire Roberts case has outlined what would happen in those circumstances *"The deaths of all children were reported to the audit coordinator and the charts once available were given to the audit coordinator secretary. The coordinator then scheduled in a date for the case to be discussed at the mortality meeting at a time that ensured all relevant specialties could attend and any outstanding results e.g. post-mortem results were available. There were no records kept on the discussion but any learning points would have been disseminated to the relevant professionals within RBHSC* " …….. *"also that now a note is made in the chart that the case has been presented and any issues around care are recorded in minutes."* And *"And that if learning is of importance to teams outside RBHSC this would be forwarded to the Medical Director's office for action."* [294]

[292] Correspondence 319-011B-001 ; 319-011B-002

[293] 319-019-002

[294] WS-143-1. (Claire Roberts papers) P 113

717) Dr Carson Royal Hospitals Trust Medical Director from April 1993 to July 2002 introduced Clinical Governance in the Royal Hospitals Trust in April 1999 [295]. He also states that it was his expectation that if the Coroner was notified about a death , Dr Murnaghan or Mr Walby should be informed by the responsible consultant. There is no record that Dr Hanrahan took this step.[296]

718) **Comment on RBHSC audit processes** : By 2000 clinical audit was well established in the NHS. Clinical audit offers an opportunity to review the process of care and the outcome. (These terms are explained in Audit Annex F). It is not evident whether RBHSC audit meetings considered the treatment only in their own unit or included in discussion the care provided at the Erne hospital. When examining causes of death it is important to record if any issues relating to care were raised which should be logged and any necessary action taken. The purpose of this would be to identify a need for a change in practice immediately or, to log similar incidents as a proportion of similar cases. The latter is a form of monitoring the effect of change in practice with a view to either improving implementation of previous guidelines or changing guidance or protocols for care. This forms part of the "audit cycle" which I explain in Annex F. It is not evident how this process was managed in RBHSC and in the Clinical Directorate which had a responsibility to supervise audit processes. Audit can take place either with single cases being presented or, a number of similar cases could be aggregated and analysed by a clinical audit process. This usually requires the obtaining of notes with the similar diagnosis from the records Department using the PAS system to search for similar coded cases. Then the auditor –a trainee in paediatrics or a consultant or both, will design a specific pro forma and use this to extract and aggregate data from the case records before analysis, presentation and reflection. The purpose was to compare what was done with what was intended to be done (matching against guidance in the hospital such as in protocols). It may be helpful to seek audit reports from the Trust as examples used by trainees in assembling a portfolio or by consultants when completing their annual continuing professional development (CPD) portfolios. In 2000 CPD should have been in place.

719) It seems that the review of deaths was to identify any learning points. It does not appear that such learning points were routinely logged for subsequent aggregation and analysis.

720) It is not clear from this evidence where items raised (for example in the meningococcal cases) were recorded so that it would be possible to identify and quantify trends by logging such events and such issues.

721) It is not evident whether an annual (or other frequency) audit report was made to the clinical directorate nor whether any reports were sent to the clinical director or

[295] WS 306/1 Q 1 (d)

[296] WS 306/1 Q 1 (e)

whether the minutes were shared with him or her or with any oversight Audit committee in the Trust.

722) There was an expectation that a consultant presenting to the meetings would review the clinical records to identify potential learning points. Also that the pathologist would attend . During such a review fluid regimes used and other therapies and investigations would be considered including those in the illness in the referring hospital in order to get a complete picture.

723) The account given by Dr Taylor in attempting to distinguish an investigation of a death seen as an adverse event from the more regular mortality meetings is, in my opinion, somewhat difficult to follow logically. I would also point out that within the conventional structure of clinical audit : Structure-Process-Outcome (and audit cycle), outcome analysis forms part of the advised process. Death is an outcome in these terms.

724) In an Audit meeting any issues arising should be documented and there is a strong argument that any adverse issues identified in such a process should be reported back to the referring hospital but in practice this did not often happen in 2000.

725) Quantification of death from a particular set of events is also a requirement for the audit as would be identified shortcomings in the resources available for the management of a child or the way in which resources were applied and similarly, if there was no satisfactory answer provided for a cause of death.

726) The process used was for the consultants responsible to present a death. If this task had been delegated to a registrar or other colleague, questions may have been raised which could otherwise not have occurred to the consultant in charge of the case.

727) Dr Hanrahan and/or a member of his team should have been present when Lucy was discussed and also Dr O'Hara who might have identified points about care which he raised at the meeting with parents.

728) Thus it appears that the processes usually adopted in RBHSC were not used in the mortality meeting in which Lucy was discussed. The lead consultants Dr Crean and Dr Hanrahan were probably not present and it appears therefore doubtful that Lucy's case was presented in detail. Had it been then the logical flaws in the sequence on the death certificate could have come to light, the lack of satisfactory explanation of the cause of death identified, the role of the fluid overload in Erne hospital identified and a referral made to the coroner and to inform Erne Hospital of the issues raised. Further actions could have led to conclusions being drawn about the role of N.18 solution in generation of the hyponatraemia, and the impact highlighted, of the normal saline volume given on the blood sodium.

729) Later RBHSC Actions about Lucy

730) After the August 2000 mortality meeting it appears that RBHSC consideration of factors affecting Lucy's death ceased until the investigation surrounding her death in the course of the Coroner's inquest took place in 2003/2004.

731) On 23.10.2001 Dr. Robert Taylor wrote to Dr. Cheng Medicines Control Agency (MCA) replying to her letter of 17th October 2001 and providing summary details on Raychel's case [297]: *I am also conducting an audit of all infants and children admitted to the PICU with hyponatraemia. My initial results indicate at least 2 other deaths attributable to the use of 0.18NaCl/4%Glucose"* Lucy's death was not identified as hyponatraemia on the PICU database [298]. The MCA replied that a warning about No 18 solution was not necessary advising him that they were satisfied that there should be no amendments to product information re 0.18% NaCl and suggest that electrolyte imbalance is a risk with all IV solutions and careful monitoring is crucial.

[297] 007-033-060

[298] WS 280/1 page 6/7

RESPONSES TO SPECIFIC QUESTIONS POSED IN MY INITIAL BRIEF REGARDING THE DEATH CERTIFICATION *My response in italics*

732) Brief

733) Having regard to the Inquiry's revised terms of reference and the list of issues set out, the Inquiry has identified the following questions and issues concerning the role of clinicians at the RBHSC (particularly the paediatric neurologists and the paediatrician who completed the death certificate in consultation with the paediatric neurologists) which you are asked to address:-

734) Dr. Donncha Hanrahan (Consultant Paediatric Neurologist)

735) What information is a clinician expected to convey to the Coroner's Office when reporting a death in hospital?

*736) The information given by a clinician contacting the Coroner's office depends to a certain extent on the cause of death or the explanation or lack of explanation for it as perceived by the clinicians. In particular a death which was expected or anticipated (for example in a child with serious congenital malformation or who had a chronic disorder which was significantly life limiting such as severe tetraplegic cerebral palsy or a neuromuscular disease or a cancer) would probably not be subject to a discussion with the Coroner's office. A death which was unexpected but explained, such as a child who had a condition with a known high mortality and in whom the clinician felt management had not been at fault, would probably not be referred to the Coroner-an example here would be severe meningococcal disease. In other cases an unexpected death associated with a condition which usually has a low mortality-for example gastroenteritis should lead to a referral to the Coroner. Then a discussion would be held and enquiry made to the Coroner's office about whether or not this should be formally reported to the Coroner. If death is unexplained, especially if there was any consideration of incorrect or delayed therapy, a formal report should be made to the Coroner for further investigation. Dr Hanrahan agreed with Dr Curtis that a hospital pm should be done because " useful to establish cause of death "*because *" cause of death was not clear to me. Lucy had also died within a short time of admission to hospital"* [299]

737) What specific information ought Dr. Hanrahan to have conveyed to the Coroner's Office and Dr. Curtis with regard to the circumstances surrounding Lucy's death?

738) *Dr Hanrahan presumably was reporting that Lucy had died from cerebral oedema. Also that she had been admitted with gastroenteritis in the referring hospital at Erne and had initially been treated there and that there she had been found to develop hyponatraemia. Cerebral oedema is a most unusual complication of gastroenteritis and this should have been stated because the death was unexpected. Discussion should*

[299] 061-018-067; W/S-289/1

have considered whether or not a formal Coroner's enquiry should be undertaken and no doubt Dr Hanrahan was seeking advice upon this point. It appears the full clinical features and detail were not given during discussion with Dr Curtis. It is not evident whether Dr Hanrahan gave consideration to, or was asked for, or offered an explanation of how the cerebral oedema might have occurred: for example, was Dr Hanrahan considering some form of brain disease although there is no evidence in the clinical notes that this was under consideration. Dr Hanrahan is unable to recall the substance of the discussion with Dr Curtis.

739) *If issuing a certificate an entry of cerebral oedema and gastroenteritis were in my opinion appropriate. However, the entry made relating to dehydration is illogical unless Dr Hanrahan was implying that the <u>treatment</u> of the dehydration had resulted in the cerebral oedema. If so, then this would be the wrong treatment and dehydration should not be listed. A formal referral and investigation by the Coroner should follow that suggestion. Dr Hanrahan states that he did not discuss with or agree the entries made in the death certificate with Dr Curtis. Dr Hanrahan remained unclear of the cause of death during this discussion (because this was the reason for his request for the hospital autopsy which did not provide a satisfactory explanation). On receipt of the autopsy reports in my opinion Dr Hanrahan should have referred again to the Coroner.*

740) In particular, should the presence of hyponatraemia and the drop in serum sodium from 137 to 127 have been reported to the Coroner's Office and Dr. Curtis? Please provide reasons for your answer.

741) *Yes. The low sodium was not present on admission and developed and was thus not a form of hyponatraemic dehydration but was a signal of fluid overload and/or syndrome of inappropriate ADH both of which conditions can lead to cerebral oedema. In my opinion cerebral oedema is more likely to follow inappropriate ADH secretion if there is sufficient fluid present and does not usually occur in a dehydrated child. The only way sufficient fluid to cause it could have been given in Lucy would be by intravenous infusion.*

742) Is it expected that a clinician reporting a death to the Coroner's Office should comment on whether there is a need for a Coroner's post-mortem and an Inquest?

743) *When a clinician contacts the Coroner's office he or she is usually seeking advice and steer on whether or not they can proceed to issue a death certificate. But clinicians should express their opinion on the degree of uncertainty they have about how death had resulted from the illness. Dr Hanrahan was seeking advice but should have indicated to Dr Curtis the degree of uncertainty he had and discussed possible mechanisms of development of cerebral oedema such as encephalitis.*

744) If so, given the information that was available to Dr. Hanrahan at the time, what opinion should he have expressed regarding the need for a Coroner's post mortem and Inquest?

745) *See my answer to the previous point*

746) What information should Dr. Hanrahan have conveyed to Lucy's parents with regard to the circumstances surrounding her death?

747) Dr Hanrahan should have explained that cerebral oedema-brain swelling-had led to Lucy's death. He should have explained how cerebral oedema might have arisen including mention of the low sodium or brain infection . Otherwise how did he explain it?.

748) What information should Dr. Hanrahan have conveyed to Lucy's parents with regard to the requirements for a post mortem when obtaining consent?

749) *Consent was obtained for the autopsy by Dr Caroline Stewart, paediatric registrar and this followed discussion between consultants involved in Lucy's care but she is unable to recall what information was given to the parents. Dr Hanrahan should have explained that at times it is necessary for a Coroner's inquest and post-mortem to take place. He should have explained that he would discuss Lucy's death with the Coroner. If the Coroner judged that a post-mortem examination and inquest should not take place, then Dr Hanrahan should have explained-and possibly did-that post-mortem should be carried out in order to determine the cause of death for example to exclude brain pathology such as viral encephalitis. Otherwise Dr Hanrahan would not be able to explain the unexpected death. If the post-mortem did not find such a disease but simply cerebral oedema then in my opinion he should have considered reporting again to the Coroner and at least discuss this with the pathologist who also had a duty to do report to the Coroner when there was uncertain cause or when the death might be related to therapy..*

750) To the extent that there was any communication between Dr. Hanrahan and Dr.O'Hara in relation to Lucy, what information should Dr. Hanrahan have conveyed to Dr. O'Hara about the circumstances of her death?

751) *Dr Caroline Stewart working with Dr Hanrahan held a discussion with the pathologist Dr O'Hara. The clinical features should have been highlighted and especially the fact that this was an unexpected and at the point before the post-mortem, in their opinion an unexplained death. And, that they were looking for an explanation for it to come from the autopsy because the clinical, biochemical and imaging investigations had not revealed an explanation*.

752) What information should Dr. Hanrahan have conveyed to Dr. O'Donoghue about the cause of Lucy's death at the time when Dr. O'Donoghue was giving consideration to completing the death certificate?

753) *In my opinion Dr O'Donoghue was completing a death certificate on behalf of Dr Hanrahan. and he was merely acting as a kind of medical clerk or registrar. From his*

witness statement it is clear that he sought Dr Hanrahan's advice and followed his instructions – this is an appropriate action for an SHO grade junior doctor. [300]

754) <u>Dr. Caroline Stewart (Specialist Registrar in Paediatric Neurology)</u>

755) To what extent are treating clinicians expected to communicate with and exchange information with the pathologist who is tasked with the responsibility of conducting the post mortem?

756) Dr Stewart would be acting under the supervision of Dr Hanrahan and all her actions were on his behalf. It was thus reasonable for her to communicate with the pathologist and she did so in comprehensively in my opinion, when completing the autopsy request form on behalf of the consultant paediatric neurologist.

757) What communications and information exchange would you have expected to take place between the clinicians who treated Lucy and Dr. O'Hara before and during the post-mortem process?

758) *In the circumstances I would expect some telephone discussion because the cerebral oedema death was unexplained but Dr Stewart had a discussion with Dr O'Hara.*

759) What is the purpose and significance of the information which a clinician includes within the 'clinical diagnosis' section of the autopsy request form?

760) *The information provided in the clinical diagnosis section to the pathologist is to give the clinical perception of the illness as determined so far. This would be the working diagnosis determining treatment and steering the investigations which in Lucy provided no diagnostic answer other than cerebral oedema and hyponatraemia in Erne both of which at that stage were unexplained. It would be necessary to convey to the pathologist all the clinical and biochemical features which had been found including imaging, EEG, and the history. The history would help the pathologist to determine how the child came to acquire brain oedema and in part for information to the pathologist to assist in the way he performs the autopsy and the conclusions he draws from it. Of importance here is the fact that the low sodium was found in the referring hospital (but not in Belfast) and Dr O'Hara was made aware of this in the autopsy request form.*

761) Please comment on the adequacy and completeness of the information which Dr. Stewart conveyed to Dr. O'Hara in the 'clinical diagnosis' section of the autopsy request form.

762) In my view this was comprehensive although no detail of the fluid volumes used in Erne was included although the type of fluid used was noted.

[300] *W/S- 284/1*

763) Dr. Stewart has told the police that no significance attached to the reference to 'hyponatraemia' in the autopsy request form. Please comment on the significance that ought to have been attached to the presence of hyponatraemia when Lucy's death was being reported to Dr. O'Hara by Dr. Stewart?

764) *Dr Stewart makes it clear in her witness statement that when completing the form she aimed to provide as much factual information as possible rather than to provide a view about Lucy rather than drawing conclusions about the findings.* [301]

765) Should Dr. Stewart have implicated hyponatraemia as a possible factor in causing Lucy's death, or otherwise asked Dr.O'Hara to investigate the role of hyponatraemia in the cause of Lucy's death?

766) Dr Stewart was acting on behalf of Dr Hanrahan who should have considered the implications of the hyponatraemia and conveyed his view to Dr Stewart.

767) If you have any concerns about the nature and quality of the information which was conveyed to Dr. O'Hara by Dr. Stewart at the time, please explain those concerns and identify the specific information which he should have obtained or which should have been provided to him.

768) No –see above

769) How should Dr. O'Hara have interpreted the information conveyed to him by Dr. Stewart in the 'clinical diagnosis' section of the autopsy request form?

> *Dr O'Hara has not provided a summary of the cause of death in his autopsy report. I believe this falls below the standard required. He has not mentioned the hyponatraemia and offers no satisfactory explanation for the cerebral oedema. He believes that the bronchopneumonia which he reported in the final autopsy report of June 2000 was a significant factor in leading to death. The clinicians had not raised this as a possibility for good reason. There was no clinical feature of bronchopneumonia on admission at the Erne hospital. Bronchopneumonia can result from pulmonary congestion and/or can be a complication of ventilation, aspiration during intubation, or increased by the episode of hypothermia during the transit. Thus it is difficult to see how Dr O'Hara placed such emphasis on this (it was not an issue in the autopsy request form) and, in my opinion, he did not provide sufficient explanation for the cerebral oedema in his summary.*

770) In particular how significant should Dr. O'Hara have regarded the reference to hyponatraemia in the 'clinical diagnosis' section of the autopsy request form?

[301] WS-282/1

771) *Hyponatraemia is a sign of inappropriate ADH secretion and/or volume overload. Both can lead to cerebral oedema and the pathologist should have interpreted this and reported the presence of hyponatraemia in his conclusion.*

772) What information should Dr. Stewart have conveyed to Dr. O'Donoghue about the cause of Lucy's death at the time when Dr. O'Donoghue was giving consideration to completing the death certificate?

773) I believe Dr Stewart did not need to do this. The certificate appears to have been completed following receipt of the preliminary autopsy anatomical summary and after discussion between Dr Hanrahan and Dr O'Donoghue

774) <u>Dr. Dara O'Donoghue (Paediatric Fellow)</u>

775) What steps is a clinician expected to take before completing a death certificate, and in particular explain:

776) What sources of information should he turn to in order to clarify the cause of death, in circumstances where a post mortem has been conducted?

777) *In response to this please see my comments above. I believe Dr O'Donoghue was simply acting on behalf of Dr Hanrahan.*

778) When (in relation to the completion of the post mortem report) is it appropriate to sign off on the death certificate?

779) *There is some pressure on clinicians to issue a death certificate so that parents can proceed to burial. However the information provided on the preliminary autopsy report should have been discussed with the parents. Pulmonary congestion was mentioned on the preliminary summary but not bronchopneumonia. Cerebral oedema was mentioned and was entered under category one a. Gastroenteritis was in my view correctly entered as a significant factor but the dehydration entry is illogical and there was an opportunity for Dr O'Donoghue to discuss this with Dr Hanrahan but as a junior doctor it was appropriate to follow the consultant's instruction.*

780) Please comment on the steps taken by Dr. O'Donoghue to complete the death certificate in Lucy's case, and explain whether in your view he,

781) Followed an adequate procedure when completing the death certificate

782) Obtained all relevant information before completing the certificate

783) Completed the certificate at the appropriate time

784) Completed the certificate accurately on the basis of the information that was available to him

785) see my foregoing comments

786) <u>RESPONSES (IN ITALICS) TO SPECIFIC QUESTIONS POSED BY THE INQUIRY.IN DECEMBER 2013</u>

787) Q1 HANRAHAN CLINICAL DIAGNOSIS Dr. Hanrahan has indicated that when he was treating Lucy on the 13 April 2000, he conducted a number of investigations which returned normal and that he did not re-evaluate his differential diagnosis in light of the outcome of his investigations (answer to question 9, WS-289/1). **In light of all that was known at that time, what steps, if any, should Dr. Hanrahan have taken in order to reach a definite diagnosis of the cause of Lucy's acute collapse ?**

788) In the course of clinical consultations it is usual to record a history of the condition. In the case of a transfer into a hospital the course of the events preceding the transfer should be reviewed. This is usually done by a review of the notes from the referring hospital and any questions which arise after that review can be clarified by telephone. This task may be delegated to a registrar. A summary can be assembled taking account of any referral letter which had accompanied the patient. This summary should have been entered into RBHSC records. The summary should include a review of the therapy given up to and during any collapse. A detailed review of the fluids would be difficult in the sense that it would be time consuming. However it is evident from a relatively quick scrutiny of the Erne hospital records that 100 mL per hour of number 18 solution was given between the setup of the infusion and the time of collapse. The overall volume given in Erne was arguably not strikingly excessive for her condition up to the arrest (see report on Erne) but when using such a volume at least 22-45 % should have been given as normal saline and the rest as 0.45% saline: in contrast – wrongly- all given was 0.18%saline. Thus the choice of fluid and hourly rate of its infusion was wrong. It is clear from the records of the blood sodium was normal on admission and fell during the infusion. It is also evident from the Erne records that a grossly excessive volume of normal saline had been given over one hour although its relationship to the timing of the second blood sample showing the level of 127 mmol/litre is not evident from the records. It was possible to conclude that the fluid regime was seriously wrong and if further questioning of the Erne review/ Dr O'Donohoe had been made by Dr Hanrahan it should have revealed that the blood sodium was probably affected by the IV normal saline and been significantly lower at the time of the arrest.

789) There is no evidence that any review of Lucy's fluid regime at Erne took place either during the clinical management of Lucy or subsequent to her death. Had it been done a more definitive diagnosis of the cause of her collapse would have become evident. In my opinion it was even more important that was done for Lucy before discussing with the Coroner because there was no clear explanation of Lucy's death other than cerebral oedema without evident cause.

790) **Q2 CONSENT TO POST MORTEM .** In her statement, Dr. Caroline Stewart indicates that the normal practice in 2000 was to obtain a verbal consent to conduct a hospital post mortem. She cannot recall what information was given to the parents about the purpose of the post mortem (WS-282/1, answer 6(I). Likewise, Dr. Hanrahan cannot

recall the information provided to the parents about the purpose of the post-mortem (WS-289/1, answer 12(c)(iii), although he interprets Dr. Stewart's note as suggesting that consent was taken in writing (WS-289/1, answer to question 12(c)(iv). **What information should have been given to the parents about the purpose of the post-mortem? At that time, should consent for a hospital post mortem have been taken in writing? Please review the answers given in the witness statements and comment on the adequacy of the consent process which was followed in respect of Lucy's post mortem.**

791) Records show Dr Caroline Stewart spoke to the pathologist Dr O'Hara and completed an autopsy request form. A handwritten entry by Dr Caroline Stewart (061-018-068) notes consent (written) by parents is accompanied by a tick. The autopsy request form notes "parents wish organs to be used for donation."[302]. A document completed in handwriting relates to consent for donation of heart valves. It is signed but it is not clear whether this is by a parent or by Dr Stewart. [303]

792) Dr Stewart cannot recall whether it was she who sought parents' consent for post-mortem or whether it was Dr Hanrahan although she made a record in the notes that parents gave consent. Dr Stewart cannot recall information given to parents or what questions were posed by them and does not know whether parents gave written consent. "It is likely that the normal practice consent for a hospital p.m. in 2000 was verbal not written".

793) *In the years around 2000 practice in the NHS varied between clinicians and even within hospitals in respect of the documentation of consent for a consent hospital post-mortem. In a child the parents as next of kin are asked for this consent. The documentation of the discussion and even the signing of a consent form varies between hospitals. Practice has become more standardised with a requirement for signed consent on a pre-designed form since the Human Tissues Act of 2004. The variation in practice was recognised in a review conducted by the Human Tissue Authority in respect of the examination and tissue retention matters which arose in the early 2000s. And a report which they issued in 2010 includes the comment "that training on obtaining consent for hospital PM examinations can be inconsistent. Moreover, processes for seeking consent are not standardised throughout the health service." In many situations, the clinician in charge of the care of the patient is involved in seeking consent. More recently, Trusts have appointed bereavement staff, who are becoming more involved in this process. (http://www.hta.gov.uk/legislationpoliciesandcodesofpractice/policyonconsentforpost-mortemexaminationandtissueretention.cfm)*

[302] *061-022 -074*

[303] *061-006-013.*

794) In RBHSC It is evident in the case of Claire Roberts in 1996 [? 090-047] that a consent form was signed by parents. Consequently it is evident that the RBHSC used written consent. By this standard written consent should have been obtained. In doing so the purpose of the post-mortem should have been explained-including the fact that it was to identify why cerebral oedema was present.

795) In RBHSC Paediatric Medical Guidelines Document issued In July 1999 Second Edition. (319-067a-031) (relevant Pages 25-26 are in Annex D) the following guidance is given: " a hospital autopsy is requested by the clinicians and requires written consent from the next of kin of the deceased. It is performed by the paediatric pathologist or by their junior staff in the RVH mortuary. For hospital autopsy the pathologist requires the written consent and the clinical summary on a completed request form. When the autopsy is complete, the pathologist will telephone the Ward with the result and a death certificate can be issued if this is has not already been done. A provisional summary is issued the next day and the final report is sent to the consultant clinician several weeks later." Of note, relevant to Lucy, is that the guidance is that if an autopsy is requested by a paediatric neurologist it is generally carried out by the neuropathologists. This was not done in Lucy. There is no written consent by parents on the autopsy request form (061-022-075) and the design does not have a section for this.

796) When obtaining consent parents should be advised of the clinical diagnosis made (cerebral oedema) and that the autopsy was being carried out to provide more information on why/how this had occurred and whether any other disease was present.

797) **Q3 DIAGNOSIS FOR DEATH CERTIFICATION** Dr. Stewart explains (WS-282/1, answer question 22(a) that it was Dr. Hanrahan, Dr. McKaigue, Dr. Crean and Dr. Chisakuta who agreed the following working pathogenesis: dehydration and hyponatraemia, cerebral oedema, acute coning and brain death. Dr. Stewart has explained (in answer to question 14) that within the clinical diagnosis section of. the autopsy form, she listed cerebral oedema, acute coning and brain steam death (after hyponatraemia) because they "were the sequel of events leading to her death." Dr. Chisakuta cannot remember the conversation leading to this view of the working pathogenesis, but he indicates that from his consideration of the notes, he would have been in agreement with it: WS-283/1, answer to question 14(a). Likewise, Dr. Crean cannot recall agreeing a working pathogenesis, but he does not disagree with anything recorded on the autopsy form: WS-292/1, answer to question 12. In addition Dr. Crean has explained that he recalls having concerns regarding Lucy's fluid management, and he was at that time aware that acutely developing hyponatraemia could cause neurological decompensation: WS-292/1 answer to question 8(b). You will recall that he told the Inquest into Lucy's death that Lucy's serum sodium registered a drop of 10 to 127 within a short period of time, and that "the rate of fall is the crucial factor" [Ref: 013-021-074]. Dr. Hanrahan has no recollection of discussing the autopsy request form with Dr. Stewart (answer to question 12(d)). **Consider whether, if this working pathogenesis had been formulated by relevant clinicians at the time of Lucy's**

death it should have been reported to the Coroner's Office? Please fully explain the answer that you provide.

798) Lucy's death fell into the category clearly defined in Northern Ireland for referral as set out in the 2008 guidance in Northern Ireland (given in Annex D) which is based on section 7 of the Coroner's Act (NI) 1959 and thus applied at the time of Lucy's death.

a. in any circumstances that require investigation;

b. the death, although apparently natural, was unexpected;

799) Lucy's death could have been reported to the Coroner listed in the report for the Inquiry from Bridget Dolan [Ref] :

a. "The death may be related to a medical procedure or treatment whether invasive or not"

b. "The death may be due to a lack of medical care"

c. "There are any other unusual or disturbing features to the case"

800) Lucy's death was unexpected and fulfilled at least one of the criteria set out: in that it was arguably "apparently natural" but was unexpected because it complicated a disease with a very low mortality and there were unusual features. The Coroner's guidance of 1995 for Northern Ireland (INQ-0240-11) states that a death which has occurred in other circumstances that may require investigation should be referred. The hospital autopsy which was performed provided no more clear explanation of how Lucy developed cerebral oedema and died. In my opinion Dr Hanrahan should have referred again to the Coroner on receipt of this report. There is also an argument that Dr O'Hara should have considered doing so.

801) **Q4 AUTOPSY REQUEST INFORMATION AND REFERRAL TO CORONER** Dr. Hanrahan had reached the view, at least by the time that he spoke to Lucy's parents (answer to question 14(h)) on the 9 June 2000, that the management of Lucy's fluids was "inappropriate" He did not document this concern answer to question 7(d)). Moreover, while he was aware that the parents "were unhappy about [Lucy's] treatment" he did not document their concerns either (answer to question 14(d). He thought that they should seek clarification of events in the Erne Hospital since the "sentinel event" took place there, but he does not indicate the areas in which clarification was required, nor does he recall attempting to clarify events himself (answer to question 14(g).Throughout his statement Dr. Hanrahan maintains that while he may have reached the view that the fluid balance was unusual, and that fluid management was inappropriate, he was also of the view that 127 represented only mild hyponatraemia (e.g. in his answer to question 8(c)), and not low enough to lead to cerebral oedema (at answer 17(b), but see also 14(h) and 29(a)). Dr. Hanrahan considers that he was not an expert in fluid management (Ref: 013-031-115), and that he was deprived of what he

refers to as "the most important link in the chain of events leading to her cerebral oedema" (answer to question 16(a)). By this, he appears to mean, the information that was supplied to him at a study day in late 2004 by which he discovered that a repeat U&E test was performed only after a quantity of normal saline had been run in (see question 17, and answers to this question, and answers to question 29**). Please review the evidence which Dr. Hanrahan has now provided. In particular, please comment on whether it was reasonable for him to take the view that a drop in sodium level from 137 to 127 within the period of time when she was treated in the Erne Hospital, could not have led to cerebral oedema? In this regard, please also comment on Dr. Hanrahan's view that he was unaware of the real extent of Lucy's hyponatraemia because he did not know that repeat electrolytes were taken after a quantity of normal saline had been run in.**

802) Dr Hanrahan was not aware at the time that a rapid fall in blood sodium can cause cerebral oedema regardless of the absolute level. This is a notable deficit of knowledge, in my opinion, for a paediatric neurologist who was involved in the care of patients on an intensive care unit e.g. for management of acute encephalopathy complicated by SIADH.

803) Many general paediatricians are aware of this risk in relation to hypernatraemia in diarrhoea illness and in diabetic ketoacidosis. His colleagues in the intensive care unit may have possessed this knowledge and the apparent deficit of communication between the two specialties is open to criticism here.

804) In respect of the second question raised, Dr Hanrahan lacked information about the relationship between the timing of the blood sample and the infusion of normal saline. On the other hand there is an argument that the Erne records should have been reviewed in much detail prior to the report to the Coroner and discussions held with Erne hospital, before the autopsy request and death certification and , before the mortality meeting held in August, especially given the rarity of the death and the lack of explanation for the cerebral oedema and probably before discussing with Dr Curtis and issuing the certificate.

805) **Q5 DEATH CERTIFICATION** Dr. O'Donoghue indicates that when it came to certifying the cause of death, he sought advice from the consultant in charge (Dr. Hanrahan) and was advised that the cerebral oedema was due to or in consequence of dehydration: WS-284/1, answer to question 16(b)(ii). He indicates that he no longer holds the view that Lucy suffered a cerebral oedema which was due to or in consequence of dehydration (answer to question 16(d) but rather that the oedema would have resulted from inappropriate fluid administration to treat the dehydration that resulted from gastroenteritis (answer to question 16(b)(iii). Dr. Hanrahan does not recall Dr. O'Donoghue seeking his input when the latter completed the MCCD, although he does not doubt that a discussion took place (answer to question 19(e) of 289/1). He also accepts that the cerebral oedema was not due to dehydration, but that instead, it was due to excessive rehydration leading to

hyponatraemia (answer to question 19(f)iii). **Please comment on the implications of the clarification now given in this witness statement. In particular, please comment on whether given what was known at the time it was reasonable to certify the death as 1(a) cerebral oedema (b) dehydration (c) gastroenteritis.** In his statement Dr. Dara O'Donoghue has explained the process by which the medical certificate of the cause of death was completed in Lucy's case. He obtained the advice of the consultant and then proceeded to complete it. He considers that he was an appropriate person to complete and sign the MCCD (WS-2B4/1, question 13), as does Dr. Hanrahan (WS-2B9/1, question 19(a)). **Please review Dr. O'Donoghue's evidence regarding the process leading to the completion of the death certificate, including the role played by him and others, and comment on whether the approach taken was appropriate in all respects by reference to any guidance on such matters**

806) *I have addressed these points above in my report in Para 553-568*

807) **Q6 AUDIT MEETING** In his statement, Dr. Robert Taylor explains his role within and the purpose of the mortality section of the Audit meeting (WS-2B0/1, answer 3). This is one of the areas in which we await further documentation from the Trust. You will note that other witnesses have commented on the Audit arrangements (e.g. Dr. Hanrahan, WS-2B9/1, in answer to question 26) but he says that he has no recollection of the particular discussions relating to Lucy's death. **You are asked to examine the answers which Dr. Taylor has given and to comment on the adequacy of the audit procedures which the RBHSC had in place at that time by measuring those procedures against the standards to be expected at the time.**

808) By the standards of the time one would have expected more structured documentation of the audit meetings both on the part of the audit committee and those clinicians who were completing their documentation in relation to continuing professional development or for trainees their portfolio. The clinical directorate should have a process for a review of the audit taking place over every year including analysis of aggregated causes of death and significant issues which were arising. In the absence of any documentation about this it is not possible to comment any further.

809) It was good practice in RBHSC to examine every death rather than only those were concerns about care had arisen. Of the total annual (20-30) deaths in RBHSC only a small subset are likely to be regarded as both unexpected and unexplained. From the RBHSC perspective, Lucy's death was expected because she had signs of brain death on admission. It was not satisfactorily or logically explained. The purpose of audit meetings and process should be to identify any learning points (as has been stated by Dr Taylor and Dr Hanrahan) which might lead to improvement in care of future patients and, to identify deficiencies in hospital resources, for example investigation and treatment. This is in order to monitor care against existing guidelines to ensure compliance. Audit also aims to identify substandard clinical care. Audit offers an opportunity for training and learning for consultants and for trainees.

810) In Lucy's care the clinical standards applied in Belfast were good. Documentation of consent autopsy is sketchy and there appears to be a lack of formal written consent which was not in keeping with the hospital's own standards at least in 1997.

811) During audit meetings opportunities exist for those attending to share their knowledge and experience and to interpret features present which might have not been evident to the treating clinicians. Also they are a forum in which to question the diagnoses made and to identify any unusual features or uncertainties about diagnosis. It is arguable that there should have been more documentation about the reasons for not referring to the Coroner and to order a hospital autopsy. The certified causes of death were open to challenge in their logical sequence.

812) Cerebral oedema as a diagnosis in Lucy was unusual. In order to provide context I have reviewed the diagnoses made in all discharges and deaths from all hospitals in England and Wales in one year (probably 2002) out of these 669,362 cases (which includes some 200,000 neonatal diagnoses), non-traumatic cerebral oedema was diagnosed in only 20 cases (Encephalopathy in 146 and encephalitis in 372). By any measure what happened to Lucy was unusual and deserved investigation. However the RBHSC may have confined its focus only to issues arising in relation to their own care rather than any referring hospital.

813) *All issues raised in an audit meeting should be logged in detailed minutes Such issues should then be aggregated and subject to an annual or other interval review. Major issues arising during such audits would require immediate attention through the clinical directorate or other hospital management structure. It would help to determine the hospital governance process which was in place in 2000 which reviewed its audit activity. Clinicians at the time were expected to record audits and conclusions/lessons learnt in the annual portfolio. The GMC Good Medical Practice (1998) which applied in 2000 requires doctors" take part in in regular and systematic medical and clinical audit, recording data honestly…"*

814) **Q7 INVESTIGATION OF DEATH WITHIN THE TRUST.** On the basis of the information currently before the Inquiry, Lucy's death was reported to the Coroner's Office and considered at the Audit meeting, but was not otherwise the subject of investigation or review by the RBHSC. **We would ask you to consider this information and to comment on whether Lucy's death was adequately considered by the RBHSC for the purposes of clinical governance, or whether other steps ought to have been taken given the standards of the time.**

815) I have provided some information in my commentary on the Erne review about the extent of investigation of deaths in children at the time. In this respect the processes within RBHSC were not out of line with most other hospitals. A more formal investigation of a death as an adverse or critical incident within a hospital requires that it is identified as such first. There is also an issue about whether or not identified mismanagement in

another hospital would be the subject of such an investigation. I have addressed that in part in my report.

816) Dr. Chisakuta has indicated that he was a Member of the Critical Incident Review Group at the RBHSC between March 2000 and August 2010. However, neither he nor any other witness has indicated that Lucy's death was reported as a critical incident. He explains that the group included representatives from medical, nursing and pharmacy departments. His responsibilities included follow-up of incidents involving medical personnel. And, every three months to present critical incidents and lessons learnt at the audit meetings. A form was completed in triplicate with a copy going to the nurse manager and the last retained in the area where the incident occurred. (Comment: Did a copy go to the Clinical director?) . He reports there were no criteria which determined whether an incident was referred to critical incident review group and that it was not a review group role to decide what constituted a critical incident. He reports that he and Dr Hanrahan decided that a hospital post-mortem should be arranged to get some answers on the loss of Lucy's brain stem function but that he has no recollection of seeing the post-mortem report. He agreed with Dr Hanrahan that the case be referred to the Coroner although he has no recollection of a discussion about the pathogenesis but had taken account of the presence of hyponatraemia, the rate of fall of the serum sodium within a short time and the way that fluids were managed.[304]

817) Dr Crean provides The Royal group of hospitals Trust policy on adverse incident reporting dated May 2000. P 47-P 52 covers the process and grading. It defines an incident as "any unexpected or unplanned incident that has a short or long-term detrimental effect on patients, staff or others, which results in material loss or damage, loss of opportunity or damage to reputation. This definition includes near miss reporting." Dr. Crean has said that while adverse incident reporting was introduced to the Trust in 2000, it was only rolled out over the following two years and was not "embedded in practice at the time of Lucy's death".[305] *I have provided as a separate document Annex D of my report on Claire Roberts which includes a review of the Chronology of adverse events investigation in children.(Paras 411-171)*

818) The clinical related adverse events to be reported are listed on P 54 but do not include unexpected or unexplained death but does list medication serious side-effects of use. Or errors in dispensing, prescribing and/or administration of medication.

819) *Comment: in many hospitals all deaths were reviewed as a critical incident, but in a significant proportion only those which were regarded as unexpected or unexplained, also in many hospitals drug or serious treatment errors (omission/commission) were regarded in the same way. In some hospitals other critical incidents such as all resuscitations or transfers for intensive care were reported/reviewed*.

[304] *WS-283/1, page 1.; WS-283/2*

[305] WS-292/2; *WS-292/1 Q15*

Dr Roderick MacFaul FRCP,FRCPCH,DCH

25 April 2013