

Dr Dewi R Evans MB, FRCP, FRCPCH, DObst.
Consultant paediatrician

Department of Child Health
Singleton Hospital
Sketty Lane
Swansea
SA2 8QA

e-mail: [redacted]

18.2.2001

direct [redacted]

My Ref: Leg2000 / Crawford / B.34
Your Ref: K2/E/2281
Enquiries to: Mrs H W Williams

Re: Lucy Crawford, d.o.b. 5.11.98

1. This report is prepared at the request of Murnaghan & Fee Solicitors, 37 Townhall Street, Enniskillen, Co Fermanagh, Northern Ireland.

2. I have received the following documents.

- i. Copy of medical notes from Erne Hospital;
- ii. Copy of medical notes and letters from the Belfast Royal Victoria Hospital;
- iii. A copy of the post mortem report and death certificate;
- iv. Copy of the GP notes and records;
- v. Copy of the hand written history prepared by Mr and Mrs Crawford, Lucy's parents.

3. My instruction is to look at the events leading to the death of Lucy Crawford. She was admitted to Erne Hospital. Her condition deteriorated. She was transferred to the Royal Victoria Hospital in Belfast. Sadly, the medical staff were unable to treat her condition successfully and she died.

4. The next few paragraphs summarise Lucy's medical history. I have numbered the pages, 1-100. There is some duplication. The numbers in brackets relate to my numbering system.

Summary from the notes of Mr and Mrs Crawford

5. The main points from the parents' history, according to time and date, are as follows.

10.4.2000		Lucy cared by baby minder. All well.
11.4.2000	8:45am	Went to baby minder.
	11.15am	Lucy vomited.
	12:00noon	Collected Lucy and went home.

13:00hr Made appointment with Dr Graham (presumably the GP)
 14:40hr Lucy seen by Dr Graham. *"nothing to worry about"*. Returned home.
 12.4.2000 07:45am Lucy *"...seemed reasonable"*. Mother went to work. Father stayed with Lucy.
 13:00hr Return from work. Lucy was in the sitting room. Given Calpol.
 18:00hr Temperature 100°F. Given spoonful of Calpol.
 18:450hr Took Lucy to Dr Kirby. Dr Kirby advised that Lucy should be admitted to hospital.
 19:15hr Went to hospital.

6. Lucy was admitted to the Children's Ward, Erne Hospital at 7:20pm on 12.4.2000 (3). Dr Malik saw her. Lucy was weighed and a urine bag attached. Mother told of the probability of gastroenteritis. Lucy had no history of diarrhoea. There was no family contact of gastroenteritis. Temperature 39.2°C. Dr Malik tried to locate a vein for an intravenous infusion. He tried *"...at least eleven times."*

7. At 8:50pm, Dr O'Donoghue saw Lucy. He placed more cream on her hand (this would be analgesic cream, to reduce the pain of intravenous access). He obtained IV access at 10:30pm. At 11:30pm, Mrs Crawford felt that there was no response from Lucy to the arrival of her sister. Her eyes were staring. Lucy vomited at 12:15am (Thursday, 13.4.2000). At 2:15am, Lucy had a bowel action that was *"...runny, green and foul smelling..."*. At 03:00am, Mrs Crawford notes that Lucy *"...moaned a little, she started to breathe loudly for 3-4 breaths, her body twitched, eyes were flickering, her body rigid, her hands I noticed were clenching backwards and tight fisted"*. Lucy did not respond to her mother. Nurses gave Lucy oxygen. Dr Malik, Dr O'Donoghue and Dr Otherson came to see Lucy. (I think that Dr Otherson is an anaesthetist). Lucy was moved to the intensive care ward at 03:35am. At about 5:30am, parents were told by a nurse that Lucy had been stabilised and she was transferred to the Royal Victoria Hospital in Belfast. The ambulance left with Dr O'Donoghue at 6:45am on Thursday 13.4.00.

8. Mr Crawford has also provided a very careful summary of events. He confirms that he went home at 23:50hr on 12.4.2000. The final part of Mr Crawford's account (pages 10-

11), records that Mr Crawford was telephoned at home as Lucy had taken a slight turn [Mr Crawford noted the time to be 14:50 but I wonder whether the time is actually 02:50, i.e. 2:50am and not 2:50pm]. He records that he arrives at the ward at 15:20 [or, more probably 03:20. This would appear to be the correct time to tally with Mrs Crawford's account]. Mr Crawford accompanied Lucy's transfer by ambulance to the Royal Victoria, Belfast at 06:30hr. Her condition did not change and he was told that the medical staff proposed carrying out brain stem tests the following day, Friday. These are tests carried out when a patient is deemed incapable of independent survival, i.e "brain dead".

The GP notes

9. The only GP entry relating to the index event is dated 11.4.00 (12). It notes "*history of vomiting and increased temperature, no diarrhoea. On examination, temperature 37.8°C. Slightly dehydrated. Abdomen ✓. ? Viral gastritis, advised re fluids and Calpol*".
10. The out of hours service records the following information. A call received on 12.4.2000 at 18:38hr (19) records a message that Lucy had been "*unwell all weekend – vomited yesterday and today – spiking temperature – breathing quite fast – has slept all day.*" It records that Dr Kirby saw the child at 19:25hr and the final message confirms that Lucy was admitted to hospital, the time was recorded at 19:46hr. The examination findings were noted as
Respiratory rate 60pm, temperature 38°, pulse rate 120pm. No localising signs. Admit children's ward".

Erne Hospital; Medical Notes

11. Lucy's previous hospital admission was on 18.11.99. She presented with typical features of bronchiolitis. The RSV (respiratory syncytial virus) test was positive. She was allowed home on 19.11.99 (22-23).
12. The GP referral letter of 12.4.00 confirms that the reason for referral was "*pyrexia – not responding to Calpol*". Lucy was recorded as being "*drowsy and lethargic, floppy, not drinking*". Examination showed a temperature of 38°C. The mucosa was moist. Ears, throat,

heart, chest and abdomen were normal. The impression of the GP was suspected u.t.i (urinary tract infection).

13. The admission note (31) records that the history was of Lucy not feeding as well for 5 days. She had been running a fever since the day before and had vomited everything she eats or drinks. Over the past twelve hours she had been very sleepy.

14. The clinical examination (bottom of page 32) notes that Lucy has a raised temperature of 38°C. The respiratory rate is 40pm and the heart rate 140pm. Weight is 9.14kg. The capillary refill time is over 2 seconds. No abnormality is found on examination of chest, heart and abdomen. There is no comment regarding blood pressure or degree of dehydration. The plan is to admit and observe and to encourage feeding. The investigations show the following.

Hb 12.1, WBC 15.0, neutrophils, 13.7, platelets 397 (all normal)

Sodium 137, potassium 4.1, chloride 105, urea 9.9, glucose 4.5, creatinine 95.

The electrolytes are normal. The urea is increased indicating a degree of dehydration (normal is 2.5-6.5, page 69). The rest of the investigations are normal. Interestingly, the urine is recorded as showing protein ++ in addition to ketones ++. There are no leucocytes. This means that there is no urine infection.

Intravenous line is inserted 23:00hrs. There is no entry regarding the fluids given.

15. The next entry (33) is timed at 03:15hr on 13.4.00. Dr O'Donoghue is called to assess Lucy's condition.

16. An entry timed 2:58am on 13.4.00 (35) records the events of the time. The entry notes

"Called to see Lucy who had a fit according to nurse. Respiratory rate 36/min, HR (heart rate) 140/min. Advised rectal diazepam as she was still twitching her hands.

3.15. Called Dr O'Donoghue to assess the patient.

3.20. Dr O'Donoghue came to see the patient had developed respiratory arrest and was making few respiratory efforts. Ambu bagging done, cardiac and pulse oximeter attached.

Passed large foul smelling stool. NaCl .9% 500 ml given over 60 minutes.

Anaesthetist called put in endotracheal tube. Pupils fixed and non responding to light. Heart rate above 100 during the whole time. BP 90/65 on average. Did not developed (sic) cyanosis. O₂ sat (saturation) 85 to 100%.

Catheterised (urinary)

4.45 Shifted Adult ICU, to be shifted to ICU Paed in RBHSC by Dr O'Donohue."

17. The entry timed 05:15hr, in another handwriting, notes that mother had noticed Lucy being rigid for 5 minutes at 03:00hr. There was also a history of diarrhoea. Oxygen saturation was 85-100%. Capillary refill time was less than 2 seconds, pulse was easily felt. Pupils were dilated and unresponsive. Repeat electrolyte investigations showed the following.

Sodium 127, potassium 2.8, urea 4.9, creatinine 28. A dextrostix level (a bedside method of checking glucose value) was 12.

Lucy was transferred to the ICU at 05:00hr, claforan, an antibiotic, was given, 1g IV immediately. She was also given mannitol 5g IV over 30 minutes.

18. A chest x-ray was recorded as showing nil abnormal. Abdominal x-ray was noted to query air in the colon and small intestine.

19. An entry dated 14.4.2000 comments on the query from the Royal Victoria Hospital regarding Lucy's fluid regime. The hand written entry is not particularly clear. It notes as if Lucy received 0.18% sodium chloride/dextrose 4% at 30ml per hour. [This is contrary to the fluid regime on the fluid chart]. She also apparently received a bolus of 100ml but the concentration of this fluid is not clear.

I am not quite certain of the total fluid volume that Lucy received in Erme Hospital. I am less certain that this was the volume she actually received. I suspect that she received more than this. From the fluid chart (42) I estimate that it is as follows: -

Oral	150 ml
0.18% NaCl	400 ml (100ml/hr from 11 pm until 3.00 am)
0.9% NaCl	500 ml (in 60 min)
<u>Total</u>	<u>1,050 ml</u>

20. An entry dated 18.4.00 (36) comments on 'Rota gastro-enteritis' Rotavirus is a common cause of gastro-enteritis in children.

Erne Hospital; Nursing Notes

21. The nursing progress note (39) confirms admission on **12.4.2000**. Temperature was 39.2° at 8:40(pm). The entry timed at **20:30hr** notes that IV fluid "...of No 18 solution commenced at **22:30hr** at 100ml per hour, to encourage urinary output. Urine specimen was obtained at **21:00hr**. IV fluids remained at 100ml per hour".

A large pale green stool was recorded at **02:30hr**. The nursing entry records Lucy's collapse at **02:55hr**.

22. The nursing observation sheet (46) contains information regarding Lucy's condition between **19:30hr** on 12.4.2000 and **04:00hr** the following morning. Her temperature is raised throughout, ranging from 38.7° down to 37.4° (normal 36.9°). The initial entry, **19:30hr**, notes that Lucy is "floppy, slightly flushed". The BM (a bedside glucose estimation) is 3.6. This is at the lower end of the normal range. At **20:30hr** she receives paracetamol 120mg pr. At **23:30hr** she is noted to be asleep. Respiratory rate is a normal 26.

23. The first entry of heart rate is at **03:15hr** where the value is 163. I think that the figure '144/113' indicates blood pressure. If so, it is a very abnormal raised value. There are no other recordings of blood pressure. The BM glucose value is now 13.6. The reference to the figure 98% is presumably the oxygen concentration.

24. The intravenous fluid chart records "SOL 18" IV (44). The daily fluid balance chart (42) records that Lucy receives 150ml of fluids orally at **09:00pm** and **10:00pm** (juice and dioralyte). From **11:00pm** she receives intravenous fluids. The hourly entry is recorded as 100/100, 100/200, 100/200, 100/200 and finally 500. I am not quite clear whether this means that Lucy receives 100ml per hour of 0.18 NaCl.

Admission to Adult ICU

25. The assessment entry notes that Lucy is unconscious. The Glasgow coma scale (GCS) is 3. This is the lowest possible score and indicates no spontaneous reaction. The normal GCS is 15. Lucy is on ventilatory support at this time.

26. The evaluation at 4:35am records that she is on full ventilatory support. The oxygen saturation is 92-99%. BP 92/49 (normal). Heart rate 112. The pupils are fixed and dilated. Mannitol 20%, 25ml over 30 minutes, is prescribed as well as claforan, which is an antibiotic.

27. The nursing entry recorded at the time of Lucy's collapse (pages 46-47) describe features entirely consistent with a tonic clonic convulsion. Lucy is given diazepam pr. There is actually an entry recording a blood pressure of 144/113 at 03.15 hr (40). The blood glucose is 13.4. IV fluids are changed to 0.9% saline and "...run freely into IV line". Urinary catheter is passed and a small amount of urine is found. The rest of the charts from Erne Hospital ICU confirm the following. Hourly blood pressures are normal at 90/50. Pulse rate ranges from 110-100. Oxygen concentration is 91-100%. Total infusion in ICU is 310 of fluid (presumably 0.9% NaCl) plus 25ml of mannitol, giving a total 335ml (53). The entry on page 54 notes that dopamine was also given.

Medical Entries of the Royal Victoria Hospital, Belfast for Sick Children (RBH)

28. The front sheet (73) confirms that the diagnoses at the RBH were cerebral oedema and viral gastro-enteritis. Also, that a CT scan of the brain showed cerebral oedema and coning. The contemporaneous record notes that Lucy was very cold, 31°C. I cannot find a record of her weight on admission to Belfast. Neurological examination confirmed that the pupils were *'fixed and unresponsive'*.

29. An entry of 13:4.00 states "*CT today shows obliteration of the cisterns suggesting that she has coned. Reasonable white matter / grey matter differentiation seen, but her EEG shows isoelectric pattern.*" The prognosis is considered *'hopeless'*. Two brain stem viability tests are *'negative'*. Treatment is discontinued and death is confirmed at 13.15 hr on 14.4.00.

30. The post mortem report (page 83 onwards) confirms that none of the investigations carried out to look for the cause of the gastro-enteritis isolated a specific organism. The only positive factor was the presence of an enterovirus PCR.

31. The general findings on post mortem showed that the body weighed 12kg and measured 73cm in length (85). There was evidence of congestion and oedema with a suggestion of consolidation, especially over the right lung. On examination of the central nervous system, the brain is recorded as showing "*...the features of generalised cerebral*

oedema with evidence of mild uncal herniation and some grooving in the tonsillar regions. Sectioning confirms the presence of diffuse oedematous change but there is no evidence of haemorrhage".

32. The significant microscopic findings are as follows. The lungs are shown to show evidence of pneumonia on both sides. No specific organisms are found. In the intestine, there are inflammatory cells at the upper limits of normal. The findings are remarkably normal. Microscopic examination of the brain shows features entirely consistent with "...widespread, generalised pericellular oedema with early neuronal changes." The findings are deemed to be entirely consistent with a history of acute cerebral hypoxia occurring shortly before death.

Apart from the post mortem report, I have not seen a formal discharge letter recording the events leading to Lucy's death.

Opinion

33. My opinion is based on my experience as a consultant paediatrician. The management of young children with a history of suspected gastroenteritis, with or without significant dehydration and with or without associated fever, is one of the most common indications for hospital admission to a children's ward.

34. This is a first draft. Much of the documentation from Erne Hospital is not very clear. I There may be additional information from the Royal Victoria Hospital, Belfast. I may amend my opinion in the light of additional information.

35. Lucy's care at Erne Hospital fell significantly below acceptable standards in several respects. It is probable that the combination of errors contributed significantly to her death. The following paragraphs summarise my opinion regarding the care that Lucy received. The observations are in chronological order.

36. I make no criticism of the general practice management. Her general practitioner carried out an examination. He did not feel that the little girl was seriously ill. Mild gastroenteritis is extremely common in young children and hospital admission is usually not required. When Lucy did not improve, her mother arranged a further general practice consultation. Dr Kirby arranged immediate hospital admission.

37. The initial clinical examination at Erne Hospital contained a number of omissions. The blood pressure was not recorded. There was no note of degree of dehydration. There was also a failure to take note of a capillary refill time (CRT) of over 2 seconds. The CRT should be less than 2 seconds, especially in a child whose temperature was increased. The respiratory rate was at the upper end of the normal range, at 40pm. The heart rate was also at the upper end of the normal rate, at 140pm (page 11, Advanced Paediatric Life Support (APLS), second edition).

38. The clinical history of not feeding for 5 days and even more significantly, the history of '... vomiting everything', should alert any competent children's doctor to the probability, not possibility, that Lucy would be dehydrated. A presumed diagnosis of "viral illness" did not give sufficient gravity to the potential seriousness of Lucy's condition at that time.

39. The initial investigations carried out were appropriate. The most significant factor is the urea value of 9.9. This is increased and is consistent with a diagnosis of at least a moderate level of dehydration. I would estimate that Lucy's level of dehydration was 5 to 7.5%. For the purpose of all calculations noted in the following paragraphs, I shall use the dehydration figure of 7.5%.

40. Lucy weighed 9.14kg. The normal fluid requirement of a child of this weight is 100ml per kg per 24 hours. Lucy's fluid requirement is therefore 914ml. Assuming a dehydration level of 7.5%, she would have lost 7.5% of her body weight. This is approximately 750ml. This is on the assumption that the child's weight before she became ill was 10kg. 7.5% dehydration is equivalent to a weight loss of 750g (or 750ml of fluid). Lucy's total fluid replacement is therefore $914 + 750 = 1,664\text{ml}$ per 24 hours or about 70ml per hour. Estimating fluid loss is not an exact science. Lucy's fluid loss of course would not only include water. She would also have lost electrolytes in the form of sodium, chloride and potassium.

41. The standard management of Lucy on admission would be to insert an intravenous line and infuse a solution of 0.45% NaCl + 4% dextrose at a rate of 70ml per hour. If there was evidence of hypovolaemic shock, one would consider an initial bolus infusion of either 0.9% sodium chloride or human albumin solution (HAS) in a dose of 20ml per kg; in this case, 180ml. I would, probably, have given Lucy a bolus IV infusion of 180ml HAS (or possibly 90ml of HAS), followed by an hourly infusion of 0.45% NaCl with 4% dextrose with

added potassium chloride supplementation. I would base my decision regarding the giving of the bolus infusion on the little girl's clinical state of perfusion and dehydration, her blood pressure and the trend of her heart rate. By my estimation, the maximum volume of fluid that Lucy would have received up until her collapse at 03.00 hr was either: -

$180 \text{ ml} + 245 \text{ ml} (70 \text{ ml} \times 3.5 \text{ hours}) = 425 \text{ ml}$, assuming no i.v line was obtained until 23.00 hr,
or,

$180 \text{ ml} + 455 \text{ ml} (70 \text{ ml} \times 6.5 \text{ hours}) = 635 \text{ ml}$, assuming i.v line was obtained at 20.00 hr.

* I am allowing 0.5 hr for the infusion of the 180 ml HAS

42. The failure to calculate the fluid replacement and document the results in the notes is woefully substandard. The decision to infuse 100ml per hour of fluid was wrong. The decision to use 0.18% NaCl from the outset was also wrong. The decision to pour in 500 ml of 0.9% NaCl at the end was wrong.

43. Whilst I would not be critical of a doctor failing to gain intravenous access in a child of 18 months; it can be an extremely difficult technical procedure, there was an unnecessarily unacceptable delay in getting the assistance of a more experienced doctor. Lucy was admitted at 7:30pm. The IV access was not successful until 11:00pm. Custom and practice dictates that if a relatively junior doctor is unable to gain intravenous access after two to three attempts, he should call a more experienced colleague.

44. I am critical of the failure of the more experienced doctor Dr O'Donoghue to make any observation regarding Lucy's condition when he inserted the IV line. He should have taken the opportunity of assessing the little girl at this time, monitoring her heart rate, blood pressure, CRT, level of dehydration and also level of consciousness and awareness. There is no evidence that any of this was carried out. I am also critical of his failure to give accurate instruction regarding fluid replacement, both with regard to volume and type of fluid.

45. Lucy's collapse at 3:00am on 13.4.00 was caused by cerebral oedema. The brain swelling caused the tonic clonic convulsion and the significant fall in conscious level. I do not think that any alternative form of treatment would have saved this little girl once she was found with fixed dilated pupils and in need of ventilatory support. The account of the events that surrounded Lucy's accounts is extremely graphic. It is clear that the medical staff involved had little experience of dealing with the collapse of a young child. It is apparent that

the ward did not contain adequate resuscitation equipment or procedure, or if they did, the medical and nursing staff in charge of the ward that night did not know how to organise things properly. Whilst I am critical at this lack of expertise, I do not think that this contributed to Lucy's demise. I think that the irreversible damage had already occurred by the time she presented with fixed dilated pupils at her initial collapse.

46. I now return to the matter of the intravenous fluids. It is not entirely clear from my reading of the notes how much volume Lucy received. This in itself is indicative of substandard medical and nursing management. It is highly probable in my opinion that Lucy received far more fluid than she was actually written up for. Assuming that her weight was recorded accurately on admission, 9.14kg, and that her weight was recorded accurately at post mortem, 12kg, we have a difference in weight of 2.86kg. [I am surprised that the RBH medical notes have no record of her weight on admission to that hospital]. One can expect some weight loss from insensible fluid loss following death. I am unaware of any information that suggested a body will gain in weight following death. If the two weights are accurate, the difference can only be explained by the presence of additional fluid, a total of 2860ml. I am rather sceptical of the medical note written the following day noting that Lucy received 30 ml of fluid per hour. I think that this was an attempt to rewrite history. There is nothing in the contemporaneous record to indicate a volume of 30 ml per hour. There is no logic to that particular rate of infusion anyway.

47. Sick patients can develop a condition called inappropriate ADH secretion. ADH is anti-diuretic hormone. Some sick patients may retain fluid "inappropriately", causing dilution of electrolytes in the extra cellular space. There was a very significant change in Lucy's blood investigations between 7:30pm and 3:00am. Sodium had fallen to 127, potassium was very low at 2.8. Urea had returned to a normal value of 4.9 within 7 hours! I do not think that one can explain these findings on the basis of some conjectural inappropriate ADH secretion. It is far more probable that this was caused by the infusion of a too large volume of fluid, most of which was far too dilute. I would never advise the use of 0.18% NaCl in a condition like this and have not done so during my 21 years in consultant practice.

48. If intravenous fluids in the form of sodium and water is corrected too rapidly in the extra cellular space (the plasma), water will pour into the cells, causing swelling of these cells. If the cell swelling occurs in the brain, this leads to cerebral oedema. The brain is contained in the confined space of the skull and there is no room for the swollen brain to

expand. Cerebral oedema leads to raised intracranial pressure. This leads to a failure of cerebral blood flow. This can lead to a rapid fall in conscious level and consequent respiratory arrest and death. This is what occurred to Lucy at about 3:00am on 13.4.2000 (page 249, APLS).

49. If Lucy had been managed according to the basic standards of paediatric practice in a district general hospital, it is extremely unlikely in my opinion that she would have sustained cerebral oedema. She should have had more careful appraisal of her clinical state, to include an assessment of her degree of dehydration. She should have received a bolus of isotonic intravenous solution; 0.9% NaCl or HAS, in a total volume of 90 to a maximum of 180ml. She should have received 0.45% NaCl with added potassium subsequently, at a rate of no more than 70ml per hour. Careful monitoring of blood pressure, heart rate and urinary output would have allowed the medical and nursing staff the opportunity of adjusting her fluid control.

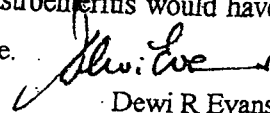
Conclusion

50. Lucy presented to hospital with a significant history of vomiting, sufficient to cause dehydration and to require intravenous fluid replacement therapy.

51. There was a failure by the admitting medical officers to assess Lucy's condition accurately. As a result of failing to carry out all the necessary assessments there was a failure to recognise the gravity of her condition. This led to a delay in the introduction of appropriate therapy, in the form of IV fluids. The level of care was significantly below acceptable standards for a district general hospital in the British Isles.

52. Both the volume of fluid that was prescribed and the concentration of fluid given were incorrect. It is also extremely likely that she actually received far more fluid than what was actually prescribed. I base this opinion on the very rapid fall in plasma, sodium and urea and the very striking difference in weight from the time of admission to the weight at post mortem.

53. Treating Lucy with the standard therapy for children with gastroenteritis would have prevented the cerebral oedema and prevented the neurological collapse.


Dewi R Evans
Consultant paediatrician