ROYALB	ELFAST HOSPITAL FOR SICK CHILDREN PAEDIATRIC NEUROLOGY
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17th June 2003.

Mr. A.P. Walby,
Associate Medical Director,
Litigation Management Office,
1st Floor,
East Wing,
R.V.H.

Dear Peter,

Re - Lucy Crawford (dec'd)

Please find enclosed my report on my dealing with Lucy. I am sorry for the delay in preparing it. I was somewhat surprised that the Coroner's Office did not feel their involvement was necessary but, at least, Lucy did have a post-mortem which can be made available to the Inquest.

Yours sincerely,

DR. DONNCHA HANRAHAN

CONSULTANT PAEDIATRIC NEUROLOGIST

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Lucy Crawford dec'd

I, Dr. Donncha Hanrahan, am a registered medical practitioner and am a Consultant in Paediatric Neurology in the Royal Belfast Hospital for Sick Children. I qualified in 1985 and was appointed to my present post in 1998. I achieved the Diploma in Child Health in 1988 and became a member of the Royal College of Physicians of Ireland in 1990. I was awarded the M.D. Degree from University College, Dublin in 1997 and am a Fellow of the Royal College of Paediatrics and Child Health. The following is a statement outlining my role in the care of Lucy Crawford, who was transferred from the Erne Hospital, Enniskillen to the Paediatric Intensive Care in the Royal Belfast Hospital for Sick Children on 13th April 2000 and who died the following day (14th April).

The principal purpose of this deposition is to describe my role once I became involved, although I will make reference to her previous medical records and the commentary already provided by Dr. Sumner.

Lucy Crawford arrived in Paediatric Intensive Care at approximately 8.30 a.m. on 13th April. I first reviewed her approximately two hours later at 10.30. At my initial assessment, I did not have access to her Enniskillen notes, an entry in her notes relating to the Ward round of that morning by Dr. Crean, stated that he was awaiting faxes of her notes and that she was to be reviewed by a Neurologist that morning. It was shortly after that that I saw her and it was subsequent to my reviewing her that Dr. Crean's typed-up entry was inserted into her notes. My impression, when I first encountered her, was that there was no sign of brainstem function and I believe, therefore, that she was brain-dead, with an irretrievable situation when I first examined her.

The story which I got at that stage was that, on 11th April, i.e. two days before transfer, Lucy was vomiting. She was somewhat better that evening and the following day, was drinking some boiled water but vomited this. She was listless and lethargic. At 2 p.m. she was given Paracetamol for pyrexia but her level of consciousness was somewhat decreased. Her parents rang the Contractors and she was seen by an out-of-hours doctor, who referred her to the Erne Hospital, with her arriving there at approximately 19.30. According to Dr. O'Donohoe's letter at that stage, IV placement was achieved three hours later. Her initial sodium was 137 and her potassium was 4.1. She was given solution 18 (0.18% NaCL and 4% dextrose) and Dr. Sumner has commented on the fluid regimen. According to her fluid balance chart, she was given 100 mls an hour of this once IV placement was achieved.

At 11 p.m., she was noted to be staring somewhat with her eyes quite glazed. At approximately 3 a.m., she was restless with some abnormal breathing and then she went rigid. At that stage, she had some offensive diarrhoea and it was then noted that her pupils were fixed and dilated. She was intubated by an anaesthetist and approximately at that stage, her sodium was found to have dropped to 127. She was then given what appears to have been a large bolus dose (500 mls) of normal saline (0.9%).

Lucy had been previously well apart from some bronchiolitis. She had been born by Caesarean section but was well. Her vaccinations were up to date and she was taking no

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regular medications. She had one older sister aged sixteen and one older brother aged thirteen.

When I examined her at 10.30a.m., she was cold (31 degrees) and was pale. She was completely unresponsive. Her pupils were fixed and unresponsive. I could elicit no corneal, gag or doll's eye reflex. Her fundi appeared normal with no haemorrhages or papilloedema. Her reflexes were present but diminished.

My overall impression, assuming that her paralysis had worn off from intubation and that she had been given no sedation, was that she was showing no signs now of brain-stem function and that she was brain-dead. I recommended a C.T. scan and an E.E.G.

Not having had access to her fluid chart at that stage, my differential diagnosis included infection, possibly from herpes, haemorrhagic shock encephalopathy, metabolic disease, including urea cycle defect and cerebral oedema from other cause. Various investigations were all normal and subsequent events transpired to indicate that cerebral oedema, probably related to hyponatraemia, in turn related to a gastroenteritis, was the cause of death.

Her E.E.G. was isoelectric, showing no discernible cerebral function. Her C.T. scan showed obliteration of the basal cisterns, suggesting that she had coned, which means that due to raised intracranial pressure, her brain was forced down through the foramen magnum, causing pressure on her brain stem and death.

I reviewed Lucy again that evening, at 17.45, and I felt that her prognosis was hopeless. I discussed her with her parents, who were agreeable to her not being actively resuscitated in the event of acute deterioration. I mentioned at that stage that if she succumbed that a post mortem would be desirable and that the Coroner would have to be informed.

In the company of Dr. Chisakuta I performed two sets of brain-stem testing, neither of which showed any sign of life. After discussion with the transplant team, Lucy was electively extubated at 1300 on 14th April 2000 and was declared dead at 13.15.

Earlier that morning, Dr. Stewart (who was then my Registrar) made a note that I contacted Dr. Curtis on behalf of the Coroners and discussed the case. The Coroner's office advised us that a Coroner's post mortem was not required but that a hospital post-mortem would be useful to establish the cause of death and rule out other diagnoses. Her parents subsequently consented to post-mortem.

Subsequently, Lucy was shown to have been suffering from rotavirus. Her post-mortem showed cerebral oedema and bronchopneumonia. The brain was swollen and showed some signs of early necrosis. The bronchopneumonia was established and was felt to have been present for twenty-four hours at least and could have happened, therefore, at Lucy's acute worsening at approximately 3 a.m. on 13th April.

Having discussed with the Coroner's office, I subsequently interviewed her parents on 9th June and I encouraged them to re-attend Dr. O'Donohoe to clarify events in the Erne Hospital.

This ends my deposition on Lucy Crawford.

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DR. DONNCHA HANRAHAN

CONSULTANT PAEDIATRIC NEUROLOGIST

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