WRC 67

I, **DOCTOR DONNCHA HANRAHAN** of Royal Belfast Hospital for Sick Children, Grosvenor Road, Belfast have made a Statement of my own free will. I understand that I do not have to say anything, but if I do not mention something which I later rely on in Court, it may harm my defence.

I am a Consultant Paediatric Neurologist. I qualified in 1985 and was appointed to my present position in 1998. I was awarded the MD Degree from University College Dublin. I am a Member of the Royal College of Physicians of Ireland and am a Fellow of the Royal College of Paediatrics and Child Health.

I would refer to the section of the Notes of Royal Belfast Hospital for Sick Children concerning Lucy Crawford and the extracts from same. I have a desire to place on the record that I had no conversation with Dr. Jarlath O'Donohoe or anyone else from the Erne Hospital about this patient before or during my management of her.

On 13th April 2000 at approximately 10.30 I first encountered the patient, Lucy Crawford. I would have been in possession of a letter penned by Dr. O'Donohoe from the Erne Hospital which was written to Dr. Seamus McKeague who was the Intensivist/Anaethestist Consultant on call in Intensive Care at that time. The letter pointed out that the child presented at approximately 1930 hours the previous day with a history of fever, vomiting and drowsiness. Capillary refill was more than two seconds which would have been suggestive of dehydration. She was given an intravenous line at approximately 2300 hours. There were various investigations carried out including haemoglobin, white cell count, platelets. On admission sodium (137), potassium, chloride, CO2, Urea, Glucose and Creatinine were all normal. At 3.00 am the patient's mother noticed her rigid and she was given Diazapam rectally but there was thereafter quite a lot of diarrhoea. She responded to bagging and was intubated and had a pulse but her pupils were fixed and dilated from 3.30 when Dr. O'Donohoe said he first looked at them. Dr. Peter Crean had previously examined the Patient and his typed note was later

put into the Chart. The Paediatric Intensive Care Unit SHO Dr Louise McLoughlin who was on call noted that the patient arrived at 8.00 am on 13th April 2000. An Anaesthetist

from The Erne Hospital rang at 9.00 am to report sodium level at 127, Potassium at 2.7 and renal function normal. From the Notes it is clear that I took a detailed history from the patient's parents. My Note states that on 11th April 2000 the Patient was vomiting everything, not eating, went to the Baby Minder, was brought home at 12.00 and at 2.30 pm went to the General Practitioner, was checked out and felt to be OK and was playing away. She slept quite well on the Tuesday night. On 12th April 2000 her father stayed at home, the child was listless, lethargic but drinking. When mum came home the child was still lethargic and was given Calpol and kept down water and she went to sleep at 6.30 pm. She was pyrexic, meaning that she had a high temperature, and they contacted the Contactors Bureau and were advised to go to the Erne Hospital and they went there about 19.30 hours. Tried IV placement for three hours – patient trying to drink – got IV at about 10.00 pm. At 23.20 hours her eyes were glassy and she went back to sleep. At 3.00 am she was restless with abnormal breathing, her arms, legs and fists were tonic – pupils were not reacting and she was unconscious. She was intubated at 4.00 am.

When I examined her, she was cold and pale and unresponsive and there was no sign of brain stem function. It was proposed that she should have a CT Scan and an EEG. My differential diagnosis did not include dilutional hyponatraemia, but did include infection, eg Herpes, haemorrhagic shock encephalopathy, metabolic disease (eg urea cycle defect). I suspected she might have cerebral oedema but was unsure of the cause. I recommended a clotting screen, ammonia and herpes PCR if a lumbar puncture was carried out.

When I reverted at 17.45 I would have had the result of the CT Scan and the EEG and I concluded that the case was hopeless and that the child should have brain stem tests as she was still on a ventilator. I subsequently carried these out in the company of Dr. Chisakuta and found no evidence of brain function.

I refer to my entry in the Notes where I stated that if the patient succumbed during the night a Post Mortem would be desirable and that the Coroner would have to be informed. I felt a Post Mortem was desirable as I was not confident as to the cause of death. My uncertainty did not extend to believing that the patient had died an unnatural death but simply that a child presenting with Gastroenteritis should not then have brain oedema without the matter being further investigated. From the Clinical Notes I see that Dr. Caroline Stewart noted that I discussed the matter with Dr. Curtis in the Coroner's Office but I do not recall this conversation. In retrospect an earlier Inquest might have been helpful as stated by Mr Leckey, but the findings of the Post Mortem Report were not considered suspicious by Dr O'Hara who carried out the Post Mortem but who did not choose to refer back to the Coroner's Office. The Clinical History Section of the Post Mortem Report would have emanated from an Autopsy Request Form completed by Dr. Caroline Stewart, my Specialist Registrar at the time. Under Clinical Diagnosis it states: Dehydration and Hyponatraemia Cerebral Oedema – acute coning and brain stem death. I would point out that Dr. Stewart was my Registrar and placed Hyponatraemia within the Clinical History Section. I believe that Dr. Stewart placed hyponatraemia as a clinical feature when filling in the Autopsy Referral Form but this is not the same as implicating it in the chain of events leading to Lucy's death. I delegated the writing of the Autopsy Form to her. Whilst I was aware that the Deceased child was Hyponatraemic for a period of time, the significance of this was not apparent to me as the sodium level in the Notes of NA127, having dropped from NA137 did not appear to me to be a marked and significant drop in sodium. One often in Clinical Practice sees a sodium level at 127. At the time I did not believe that this drop in sodium level was sufficient to have caused brain oedema and coning. On reflection and given that there has been some debate over Lucy Crawford's death since the Inquest and the calling of a Public Inquiry I believe that the sodium levels were considerably lower than 127 when the patient coned which in retrospect I believe occurred around 3.00 am on the 13th April at Erne Hospital. It appears that the patient was given 500 millilitres of normal saline .9% per hour after this event and it was some time after the drip was changed to normal

saline when the sodium was re-tested. The resultant sodium levels (showing at 127) was not the alarm bell that it would have been if it had been taken at 3.00 am when the patient coned. I would stress that this was not something that I was (aware of) at the time of my management of the patient and is something that has come to my attention subsequently when at a recent Study Day at Royal Victoria Hospital I had a brief conversation with Dr. Jarlath O'Donohoe.

I am aware and was at the time aware of the term Hyponatraemia. Cerebral Oedema can result from Hyponatraemia leading to raised intra-cranial pressure and coning. The Cerebral Oedema occurs when blood becomes too dilute i.e. there is too much water relative to sodium and when this occurs fluid will run from the blood to the brain and the brain will swell up. Essentially, the system of the patient will have no time to acclimatise. At the time that I was considering the patient, the drop in sodium from 137 to 127 was not in my view a marked drop. I would stress that I was unsure what had caused the death of the patient and hence my differential diagnosis.

I have no recollection of my conversation with the Coroner's Office. From the Notes it does appear that I discussed the matter with Dr. Curtis for his advice. The appropriate section of the Notes was written by Dr. Caroline Stewart. I am not aware if I mentioned at this point Hyponatraemia along with dehydration but I may not have as it was not something to the forefront of my mind at this time. I was however sufficiently concerned that the cause of death would be properly examined and I assume that I did at least say to Dr. Curtis's office that the patient died of Gastroenteritis, Dehydration and Brain Oedema. The Note states that a Coroner's Post Mortem was not required but a Hospital Post Mortem would be useful to establish the cause of death and rule out another diagnosis. The parents' consent was obtained for the Post Mortem. It may have been felt that a Paediatric Post Mortem would be more helpful than that of a Forensic Pathologist. The Pathologist would have had the power to request an Inquest if felt to be necessary by referring back to the Coroner.

The note of 4th May 2000 is written by Dara O'Donoghue and in relation to the filling out and compiling of the Death Certificate. The Death Certificate was not written until the Post Mortem Report was obtained. He was the Intensive Care fellow. I do not recall the conversation that I had with Dr. O'Donoghue and I am therefore relying on the Notes in this regard. It would appear from the Notes that Dr. O'Donoghue spoke to Dr. Stewart. It would further appear that the Post Mortem Result was on the front of the Chart. It would seem that there was a conversation between Dr. O'Donoghue and myself in relation to his liaising with me in relation to what he should put on the Death Certificate as the cause of death. It was not uncommon for the more Junior Doctors to write Death Certificates. On looking at the Post Mortem Report I note that it is dated 13th June 2000 and on the first page there is the final Anatomical Summary and the Commentary. On looking at the Hospital Notes and Records Dr. O'Donoghue's note is dated 4th May 2000 and thereafter the Funeral Undertaker was provided with a copy of the Death Certificate. I assume that Dr. O'Donoghue would have been in possession therefore of the provisional Anatomical Summary only. This is dated 17th April 2000. I imagine that Dr. O'Donoghue would have discussed with me the content of same where it states history of 24 to 36 hours of vomiting/diarrohoea illness with dehydration and drowsiness. History of seizure with pupils fixed and dilated following intubation. Relatively little congestion with some distention of large and small intestine with gas and patchy pulmonary congestion, pulmonary oedema. Swollen brain with generalised oedema. Heart given for transplantation purposes. I would have been of the opinion from that that the pulmonary oedema co-existed but was not caused by the brain oedema and I therefore assume that gastroenteritis, dehydration and brain oedema were put on the Death Certificate due to this provisional Anatomical Summary and after Consultation with me. I have considered the final Anatomical Summary which is different in that it states extensive bilateral broncho-pneumonia. I again believe that this co-existed with brain oedema but was not part of the primary chain of events leading to death and this had not been mentioned at the time that the Death Certificate was being compiled. The Pathologist did not mention Hyponatraemia despite the fact that that was placed in the Clinical History by Dr. Caroline Stewart, my Specialist Registrar at the time. It was in my opinion appropriate to

draw on the Anatomical Summary for the purposes of the compilation of the Death Certificate.

I would advise that in the Notes and Records to which I have had access there is a provisional Anatomical Summary and it is noted that where the copy you have disclosed shows a total number of pages 9 – the page I have access to states total number of pages 1. The content of the provisional Anatomical Summary is the same as Page 9 of that in the Post Mortem Report with the exception that the page numbers are denoted to be 1 as opposed to 9 and the provisional Anatomical Summary is denoted from points 1 to 5 as opposed to from points 7 to 11 and it is signed as well as dated. I believe that Dr. O'Donoghue would have been in possession of only this at the time that we discussed what should be placed on the Death Certificate.

On 16th May 2000 I wrote to Lucy's parents advising that I would be happy to meet with them.

On 9th June 2000 I had a discussion with her parents. It was stated that they had met Dr. O'Donohoe from Erne Hospital who did not have the Notes at the time. I went over the events leading to Lucy's death with the parents and encouraged them to re-attend Dr. O'Donoghue and have him explain events that occurred in Erne. I said that I would meet them again if required. On 14th June 2000 I contacted Dr. O'Donohoe who said he would see them again but that he would rather wait for the Post Mortem Report.

SUMMARY

In summary, Lucy Crawford died on 14.4.00, having been admitted to the Erne Hospital with gastroenteritis. She developed cerebral oedema which is felt to have been due to hyponatraemia. I voluntarily contacted the Coroner's Office because I felt that the death (in the context of a usually trivial illness), was unusual. In retrospect, the death may have been due to unnatural means which I was not aware of at the time. Unnatural death

would have arisen from inappropriate fluid administration, causing hyponatraemia. I state that I was not in possession of the facts concerning the severity of her hyponatraemia – I believe that when she coned, her sodium was considerably less than the figure of 127 that I was given. If I had been in possession of the full facts, the knowledge of the profound hyponatraemia, which in my view was necessary to result in cerebral oedema and death, would have led me to then suspect that her death might have been due to unnatural means. I did not consider that the mild hyponatraemia, which Lucy reportedly had, was enough to explain her presentation. I would further emphasise that Dr. O'Hara, who was a Paediatric Pathologist, did not attribute her cerebral oedema to hyponatraemia either, despite being given, by a member of my team and therefore under my control, all of the information about Lucy's hyponatraemia that I was privy to at the time.

Solution 18, at the time of Lucy's death, was a very widely used intravenous fluid. I would not have been aware of a contra-indication to its use in a child with normal sodium, which Lucy had when the infusion commenced. It is only in the recent past that controversy has grown concerning its use.

My input to the Death Certificate reflected the post mortem findings. No mention was made of hyponatraemia in the summary of Dr. O'Hara's Post Mortem.

Although I was in no way responsible, I greatly regret the tragic death of Lucy Crawford. I am particularly upset that her family may not have the same high opinion of me that they once had and which they wished to be publicly expressed at the Inquest. Her family raised money for the Royal Children's Hospital and on its presentation to us, I told them that I would see them at any time. I was told that an individual in Omagh (presumably Mr Miller) was helping them and I believe that I told them that I would help them in any way I could.

Very hard lessons have been learned following the deaths of Lucy and others. The hazard of hyponatraemia has become much more publicised, both in Northern Ireland and in the broader medical literature. Furthermore, it is proposed that a Medical Panel be set up to examine deaths and it is my sincere wish that a similar death to Lucy's not occur again.