

**MEDICAL REPORT NEVILLE & MAY CRAWFORD on behalf of
LUCY CRAWFORD (Deceased) -v- Sperrin Lakeland Health & Social Care Trust**

Strictly Private and Confidential

Date of birth: 05.11.1998
Date of death: 14.04.2000

Date of report: 7.03.2002

This report has been prepared at the request of the Directorate of Legal Services, Central Services Agency and is based on material made available including hospital notes relating to admission of the child to Erne Hospital in November 1999 and April 2000. The first admission was for bronchiolitis and is not directly relevant to the problems which occurred during the subsequent admission in April 2000. At that time Lucy was admitted to hospital with a history of fever and vomiting for 36 hours associated with drowsiness for 12 hours. There was no history of cough and her chest was clear on examination. The history and clinical findings had been thought by the GP to suggest urinary tract infection but it was felt on admission that this was more likely to be a viral illness. Initial blood tests were performed and Dr Malik attempted to commence IV fluids but was unable to do so and so called Dr O'Donohoe (the Consultant Paediatrician). IV fluids were then commenced. The nursing notes record observations at 19.30, 22.30 and 23.30 during which time the high temperature gradually came down to 37.4.

There is then a gap in the observation sheet with no apparent entry until an episode of sudden collapse which occurred around 3.00 am. It appears that mother called nursing staff as Lucy had passed diarrhoea and then become rigid. Dr Malik was called and felt that this could be a febrile convulsion so administered Diazepam. He discussed the case with Dr O'Donohoe who then came directly to hospital arriving at 3.20 am. At around this time Lucy's condition further deteriorated as she stopped breathing and required respiratory support. The on-call Anaesthetist was called at 3.40 am and Dr Auterson arrived shortly after 3.50 am and assisted with the resuscitation including intubation and transfer to the Intensive Care Unit prior to stabilisation and transfer to the Paediatric Intensive Care Unit in the Royal Belfast Hospital for Sick Children later on the same morning. The doctors involved seem clear that there was no episode of cardiac arrest or circulatory instability during this period but it was noted that the pupils became fixed and dilated and did not respond to ventilation or the administration of Mannitol.

Subsequently tests in Belfast revealed evidence of brain stem death and post mortem examination was performed. This showed bronchopneumonia and cerebral oedema with evidence of herniation of the brain. The Pathologist is unable to comment as to whether the bronchopneumonia had been present from admission to Erne Hospital or had occurred in association with the collapse and resuscitation. Further specimens have shown rotavirus infection suggesting that the initial admission was likely to be due to rotavirus gastroenteritis. Urine cultures showed no significant growth.

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Comment

This child's admission to Erne Hospital was very typical of gastroenteritis in this age group. This is often associated with high temperature and vomiting with or without diarrhoea and young children can become very unwell. The standard treatment is to administer fluids either orally or (if there is significant dehydration or vomiting) by the intravenous route. The solution used is one which is commonly used in Paediatric practice to provide maintenance fluids in these circumstances as it replaces small amounts of electrolytes but also gives Dextrose which is required by young children who are unable to take calories orally during the acute phase of the illness. Initial physical findings were suggestive of poor peripheral circulation with delayed capillary refill time >2 seconds. The GP noted that the mucosae were moist but there is little specific detail in the admission note regarding evidence of dehydration. However, the urea was 9.9 which is slightly elevated suggesting a mild degree of dehydration. Anything other than appropriate fluid replacement with careful monitoring and nursing observation. However, in this situation the intravenous fluids for replacement should contain a higher content of sodium (eg "normal saline" - 0.9% NaCl - sodium chloride)

In these circumstances it is always very difficult to understand an episode of sudden collapse. Sudden onset of convulsions is most commonly due to high temperature in young children and this was considered. However, the features were not typical and the temperature had in fact improved since admission. It is unclear as to what alternative diagnoses were considered at this time but the blood test for electrolytes was appropriately repeated immediately. This showed a significant fall in sodium from 137 to 127 and in potassium from 4.1 to 2.5, together with an increase in glucose from 4.5 to 10.9. These changes do raise the question as to the fluid management in the period from insertion of the IV line at 2300 to the collapse at around 3.00 am. Unfortunately there appears to have been confusion between the staff involved as to the fluid regime ordered by the Consultant. In addition it is difficult to interpret the records made by nurses on the fluid balance chart and no totals have been calculated for this period. It will be most important to determine from the staff involved exactly how much of each type of fluid was given at each stage throughout this time period, and following the change of fluids to normal saline through until the child arrived in the Paediatric Intensive Care Unit in Belfast.

Other aspects of this tragic case demonstrate a rapid and effective response by the medical staff concerned. In particular both the Consultant Paediatrician and Consultant Anaesthetist appear to have been available within a very short time period of being called and to have done their best in the difficult circumstances involved in caring for a child of this age in an adult intensive care setting for stabilisation and transfer in the absence of a Paediatric transfer service in Northern Ireland.

Over recent years concerns have begun to be expressed regarding the use of 0.18% saline in Dextrose as a standard solution for intravenous use in young children and a number of cases of

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symptomatic hyponatraemia have been identified, some resulting in death or cerebral damage. It has been suggested that a more appropriate solution would contain a higher level of sodium and this has recently been the subject of discussions involving the Department of Health, Social Services and Public Safety and production of guidelines. However, it must be emphasised that this is a very recent development and that many Paediatric Units are continuing to use the solution which was initially given in this case. Although the sodium level of 127 is not in itself usually associated with severe problems, it is likely to be the rate at which the sodium falls rather than the absolute level which can cause problems in this setting.

While no definite conclusions can be drawn regarding the cause of this child's deterioration and subsequent death, there is certainly a suggestion that this was associated with a rapid fall in sodium associated with intravenous fluid administration and causing hyponatraemia and cerebral oedema. In these circumstances successful defence of the case would depend on clear documentation regarding the fluid type and rate prescribed, together with clear records as to the exact volumes of each fluid which were in fact received by the child throughout the time period concerned. This is where I would anticipate great difficulty in achieving a successful defence as there appears to have been confusion between the staff involved with inadequate documentation and record keeping. In this respect, unless this can be clarified in a satisfactory manner, it is my opinion that management fell below the standard which would be accepted by a responsible body of medical opinion as reasonable practice at the relevant time.



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