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MEDICOLEGAL REPORT

ON

CONOR MITCHELL

Deceased

dob: 12th October 1987

Died: 12th May 2003

Prepared for: John L Leckey LL.M
HM Coroner
Coroner's Office
Courthouse
Old Town Hall Building
80 Victoria Street
Belfast BT1 3GL

By: Edward Sumner MA, BM, BCh, FRCA
Consultant Paediatric Anaesthetist
[redacted]

November 2003

My name is Edward Sumner and I am a consultant in Paediatric Anaesthesia with an interest in Intensive Care.
I was consultant at the Great Ormond Street Hospital for Children, London, from 1973 until June 2001. I am the author of several textbooks on the subject and am the Editor-in-Chief of the Journal, *Paediatric Anaesthesia.*
Currently, I am the immediate past President of the Association of Paediatric Anaesthetists of Great Britain and Ireland.

In the preparation of this report I have carefully perused all the medical and nursing notes presented to me by the Craigavon Area Hospital and Royal Belfast Hospital for Sick Children, Northern Ireland and the Statements of Joanna Mitchell, her mother and brother

I understand that my overriding duty is to the Court on matters which are within my expertise. I also believe that the facts I have stated in this report are true and that the opinions I have expressed are correct.

Conor was born on October 12th 1987. During the first year of life it became apparent that he was suffering from spastic tetraplegia.
A CT scan showed severe abnormalities with extensive infarctive destruction in the left middle cerebral artery territory and a smaller area in the right frontal lobe.
It was presumed that there had been an episode of intrauterine ischaemia.

Conor was very intelligent but physically very disabled, however he was not prone to the common intercurrent illnesses such as the common cold.
He suffered from mild epilepsy and was taking Epilin. The seizures were of the petit mal type, but Conor did not suffer from muscle spasms.

On May 8th he was admitted to Craigavon Area Hospital via the A and E department.
At this time Conor weighed approximately 22 kg.
There was a 10 day history, starting with an upper respiratory tract infection which was treated with penicillin.
There was also intermittent vomiting and he was very tired, though his mother was able to give him small quantities of water by mouth at frequent intervals which seem to have been absorbed.

On May 6th it was noted that the urine contained sediment and was strong smelling.

By the 8th May, Conor was lethargic and unwell and seemed to be in pain, so the GP thought it best that he be admitted to hospital for observation.
On admission at 1055 am he was found to be unwell, with reduced responsiveness and pale, but he was afebrile. He was somewhat dehydrated with a dry mouth.
There were no abnormal signs in the abdomen which was soft and not tender.

The blood count at 1100 showed a high White Cell Count (19.1) with 88% granulocytes. CRP was normal.
At that time serum sodium was 138, potassium and chloride somewhat low at 3.0 and 97 respectively and the urea was at the upper limit of normal, but creatinine was normal.
Urinalysis showed protein, blood and ketones.
Urine and blood cultures taken at that time were subsequently found to be normal.

It was presumed that Conor was suffering from a urinary tract infection and that he should be given IV fluids and IV antibiotics. He was put in a side room at first, but eventually was transferred to the Adult Medical Admissions Unit as he was thought to be too old for the Paediatric ward. It was planned to discuss with the paediatricians the rate of intravenous fluid infusion.

The fluid chart is page 31 and notes that 110 ml Hartmann's solution was started at 1120 and a further 110 ml given at 1145.
A further 110 ml is noted, but it is not clear whether this was given.
This chart also notes that 10ml/kg fluid would be given and that Conor weighed 22kg.

The reverse side (page 32) prescribes 220ml Hartman's over half an hour and that 110ml was given at 1120 and 110ml at 1145. Then the prescription for 1litre normal saline plus 20mmol KCl over 8 hours, then 1litre 5% dextrose plus 20mmol KCl over 8 hours, then 1litre of normal saline over hours are crossed out.
Two hundred mg ciprofloxacillin were given at 1200.

At 2pm the IV cannula came out of the vein and was not replaced until 4 10 pm.
At 3pm the nurses noted that Dr Totten was informed that Conor was suffering from spasms and also that Dr Totten had been informed about the non-functioning intravenous line at 2, 2 30 and 2 45 pm.
A Uribag was attached to collect and measure the volume of urine passed.

A further 250 ml of fluid (no note of which type of fluid) were given between 4 10 and 7 40 pm.
There is no note of the volume of urine passed, though the Uribag was emptied at some stage.

At 7 40 a further 250 ml of normal saline were put up to run for 6 hours.

The statement of Conor's mother suggests that the infusion was via a syringe pump and that 110ml were given every 15 minutes and that he received 440 ml in one hour.
She also says that Conor's face became swollen and puffy.

During the afternoon there appeared, intermittently a rash over the abdomen.
After 5pm the nurses noted that he was having spasms.

At some stage he may have bitten his tongue as dark fluid was oozing from the side of his mouth. A suggestion of transfer to the Royal Belfast Hospital for Children was made.
Conor's mother was of the opinion that the spasms were in fact seizures and of a type that he did not usually have and that 10-12 occurred between 1 and 8 pm.

The tendency was for them to become more violent and prolonged.

He was being given supplemental oxygen via a nasal cannula.

There is no note to say whether oxygen saturation was being monitored by pulse oximetry.

An urgent portable chest Xray was requested at 7 15 and at 7 20 the family called the nurses because they thought Conor had stopped breathing after a seizure.

He was seen by the registrar, Dr ~~Murphy~~ MURDOCK who found him to be breathing satisfactorily with stable observations. The chest Xray was normal and an ecg taken at the time, though of poor quality, showed no obvious abnormality.

At 7 40 he was given cyclizine for nausea.

The paediatric registrar was present when Conor had 2 further seizures after which there was no respiratory effort and he was turning blue.

At first he was ventilated with bag and mask, after which he was intubated by the anaesthetist after a dose of 25mg suxamethonium. The oxygen saturation during mask ventilation was 98%. He was described as "bubbly" and the tracheal tube was suctioned.
A nasogastric passed after tracheal intubation yielded "coffee grounds"

The pupils were dilated and unresponsive to light. At this stage he was give 300mg phenytoin intravenously and acyclovir, though it was thought to be unlikely to be a viral encephalitis.

Arterial blood gases at 21 25 show over ventilation (PCO_2 2.2) with 100% oxygen. Sodium was 134 and potassium 3.4; glucose was normal.
An urgent CT scan was undertaken. The old abnormalities were noted, but in addition there was a suggestion of subarachnoid blood and the basal cisterns appeared tight. The images were linked to Belfast.

Conor was then transfered to the ICU and on admission had a Glasgow Coma Score of 3, with fixed and dilated pupils.
Full ventilation and cardiovascular monitoring were undertaken. A left subclavian line was inserted, but this proved to be arterial.
Serum potassium was 2.5. The blood pressure was low so an infusion of adrenaline was commenced. The blood sugar rose to 13 which was treated with intravenous insulin (Actrapid).
Potassium supplements were also given IV and he was cooled to 34 ^{0}C.

The Department of Neurosurgery was contacted, but they did not think that neurosurgical intervention was indicated.

The following day (9th May) at 1005, the sodium was 149 and potassium 3.8. The plan was to test for brain stem responses as it looked as if Conor was brain dead as he was without corneal reflexes, had no cough and no spontaneous respiratory effort.

At 1500, the sodium had risen to 154 and intravenous sterile water was given at 50 ml per hour, but during the afternoon, the Glasgow Coma score seemed to improve as he seemed to move his foot on command.
Conor was catheterised at1615 and 70 ml residual urine drained freely. The ICU chart suggests that he had been incontinent of urine at least 4 times since 8 am and no estimate of the urine output was made.
There is no note of urinary electrolytes or osmolality having been measured.

The ICU nursing staff thought it more appropriate that Conor be moved to a PICU as his body habitus was that of an 8 or 9 year old.

Arrangements were made to transfer Conor to the Paediatric ICU in Belfast and he was admitted there at 2125.

At this stage the serum sodium was more than 160 and he developed diabetes insipidus for which he was given DDAVP (Desmopressin).
A further CT scan was performed on 10th May which showed no significant intracranial haemorrhage, but there was extensive diffuse oedema of the majority of the brain including the midbrain and brainstem. There was loss of grey/white differentiation suggestive of infarction. The cause was not apparent, but the radiologist thought ischaemic changes would give this appearance.

The pathological processes involved were not completely clear. It was suggested that a viral illness had provoked cerebral irritation and seizures which had caused a subarachnoid haemorrhage and the brainstem signs.

A full history and examination was undertaken by the neurologists at 1750 on 10th May.
They comment that Conor seemed to have had multiple tonic seizures and then a respiratory arrest which seemed to be due to acute brainstem compression, secondary to cerebral ?swelling ?hypoxic/ischaemic episode.

After a period of observation and adjustment, with Conor's neurological condition remaining unchanged and with loss of cardiovascular, respiratory and temperature control, supportive treatment was discontinued on 12th May and he died at 1545.

The Autopsy Request Form written by Dr Bothwell puts the clinical diagnosis as "Brainstem dysfunction with cerebral oedema related to:
1) viral illness
2) over-rehydration / inappro fluid management
3) status epilepticus causing hypoxia"

Postmortem findings were of cerebral oedema and coning.
All viral studies were normal

I would like to make the following comments:

Conor died of the acute effects of cerebral swelling which caused coning and brainstem death.

Professional fluid balance involves accurate assessments of the degree of dehydration and the volumes of maintenance fluids required, based on the body weight, plus accurate measurement of urine output and any abnormal losses such as vomiting (in Conor's case) and diarrhoea (which Conor did not have) Regular measurement of serum electrolytes is advisable.

It is not possible to say exactly how dehydrated Conor was. His mouth was said to be dry, but there are other reasons for a dry mouth than dehydration, for example, mouth breathing. There is no mention of skin turgor or capillary refill. On balance, I think he may have been 2-5% dehydrated which is a deficit of 600-1000ml. The initial electrolytes do show a low potassium and the possibility of early pre-renal failure with a high, normal blood urea.

Maintenance intravenous fluids would be in the region of 5ml/kg/hour, which for Conor would mean 110ml/hour as he weighed 22kg.

There is no real need for someone with mild dehydration to be rehydrated quickly. It is common practice to replace the deficit over 12 or even 24 hours.
In Conor's case, if over 12 hours, this would be approximately 60ml/hour, plus the normal hourly maintenance so the total for the first 12 hours would be 170ml/hour, after which he could go back to the normal maintenance of 110/hour if there were no other losses from vomiting etc.

The original prescription for fluids, crossed out was appropriate.

Hartman's solution or normal saline plus a potassium supplement are appropriate solutions for short term maintenance therapy.

A urine collecting bag had been put on, but there is no record of how much urine had been passed.

It is not clear how much intravenous fluid was actually given.
If 440ml had been given over the first hour, this amounts to 20ml per kg and though this is a large fluid bolus, it is not excessive for a mildly dehydrated child with normal cardiovascular and renal systems. There followed several hours with no fluids. However, there is evidence that Conor's face became puffy which does imply some acute fluid overload.

In my opinion and on the balance of probabilities, Conor was having major seizures during the afternoon. The evidence is that he did not suffer from spasms as the abnormal movements had been diagnosed and also it is likely that he had bitten his tongue which does occur with grand mal epileptic seizures.

Conor may not have properly absorbed his epilepsy medication because of the vomiting and his being generally off colour for a period of time may possibly have triggered his renewed epileptic activity.

I am not a neurologist, but my understanding is that prolonged, untreated seizure activity is very damaging neuronally and that electrical seizure activity is itself damaging and that secondary neuronal damage can also occur from the relative cerebral hypoxia and increased cerebral metabolic demands which often accompany seizures.

There is little evidence for generalised hypoxia having a major role. Conor was having supplemental oxygen and though there is no note of oxygen saturations until the respiratory arrest, when from his mother's evidence he went blue and the oxygen saturation fell to 85%, it is unlikely that significant cyanosis would have gone unrecognised during the afternoon.

It is my opinion that the coning which eventually led to Conor's death, occurred at the time of his respiratory arrest. This is the time when his pupils became fixed and dilated and he lost respiratory effort. On admission to ICU the Glasgow Coma Score was 3 and though later he seemed to improve to a score of 6, the movements thought possibly to have been on command, may actually have been spinal reflex movements of the legs.

The marked hypernatraemia which occurred after this event is hard to explain. Hypernatraemia occurs after a large sodium load with relative loss of water. Conor did have a fluid and sodium load, but this was in a balanced solution, an excessive volume of which, in the normal way would cause tissue and pulmonary oedema, with a normal serum sodium. The chest Xray in the evening was said to be normal. The electrolyte changes occurred from the day after the coning.

There is no evidence that a viral illness was the cause of the encephalopathy.

To conclude and summarise:

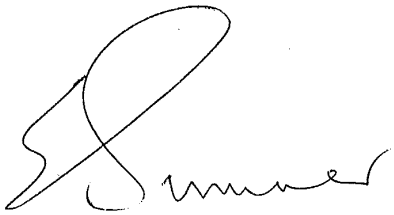
I think it is regrettable that Conor was not nursed in a paediatric environment as he was small for his age, weighing only 22 kg.

It is impossible for me to be dogmatic about the cause of the acute brain swelling that occurred on 8th May 2003.

The total volume of intravenous fluids given was not excessive and the type of fluid was appropriate, but was the initial rate of administration too great for Conor? There was no pulmonary oedema, but his face did become puffy.

We may never know exactly what sparked off the seizure activity and whether this prolonged, untreated fitting caused the brain swelling, leading to coning.

The major hypernatraemia occurred after brainstem death and, in my view probably had no part in the causation of the initial brain swelling.



24th November 2003