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Your Ref:
AD-0664-13

Our Ref:
HYPS071/01

Date:
11th October 2013

Ms A Dillon
Solicitor to the Inquiry
Arthur House
41 Arthur Street
Belfast
BT1 4GB

Dear Madam,

RE: INQUIRY INTO HYPONATRAEMIA RELATED DEATHS – CONOR MITCHELL

I refer to the above matter and to your letter of 9th October 2013.

Please find enclosed a copy of the report of Dr Brian Lynch which was obtained on behalf of the Southern Health and Social Care Trust by, its then legal representative, Carson McDowell Solicitors. I would ask that you note that whilst Dr Lynch was instructed on 29th April 2004 his report was not furnished until 5th September 2004.

Yours faithfully

Joanna Bolton
Solicitor Consultant

REPORT

5/9/04

TO:
Carson McDowell Solicitors
Murray House
Murray St
Belfast BT1 6DN

RE:
Conor Mitchell

Date of Birth: 12/10/87

Date of Death: 8/5/03

Your Reference: CB/CAHMN390/JAT

Dear sirs

Further to previous telephone consultation, I have prepared this report based on documents supplied, which include the following:

Medical Records, Craigavon Area Hospital

Medical Records, Royal Victoria Hospital re: Conor Mitchell

Various witness statements for the purpose of the inquest:
Independent
Dr Brian Herron, consultant neuropathologist
Dr E Sumner, Consultant paediatric anaesthetist
Family:
Joanna Mitchell
Jonathan Mitchell
Judy Mitchell
Trust employees:
Dr Suzie Budd
Dr Michael Smith
Dr Paul Rice
Dr Catherine Quinn
Dr Richard Brady
Dr C McAllister
S/N Ruth Bullas

S/N Barbara Margaret Anne Wilkinson
Dr Andrew Murdock
Mr Paul Kerr
Statements of RBHSC employees:
Dr Ian Rennie
Dr McKaigue
Dr Elaine Hicks

I will not reiterate all of these statements or records in detail but will abstract from them.

Medical Records, Craigavon Area Hospital

Conor, a 15 year old boy, was referred to the hospital by his general practitioner because of vomiting and poor oral intake. He had a previous history of cerebral palsy. There was a preceding history of lethargy and poor oral intake. He had been treated with penicillin for an upper respiratory infection, improved, and then started vomiting. Strong-smelling urine was noted. "Spasms" were increasing in frequency over the past 2 weeks.

His pre-existing epilepsy is described as "occasional absence seizures, treated with Epilim (sodium valproate). Dose of Epilim was 200 mg three times per day. In the nursing notes, it is recorded that he has had 3 seizure since Christmas, described as "petit mal".

On examination, he was noted to be drowsy, pale, with flexion contractures of both arms. He was afebrile. Pulse was 72 and regular. Cardiac and abdominal examination were normal.

Initial investigations revealed normal glucose, (7.6). sodium (138), Urea of 7.8 creatinine of 57, pH of 7.41., Haemoglobin was 13.6, white cell count 19.1, platelets 232, C reactive protein less than 5. His urinalysis was positive for protein, blood and ketone bodies.

The intial note is timed at 1 pm. At 6:30 pm, concern was voiced by the family re: a rash on his abdomen. The doctor who examined Conor did not find a rash. Because of the family's ongoing concerns, transfer to the Royal Victoria Hospital was arranged.

At 8:43 pm Conor was assessed because of a "tonic seizure" which resolved within seconds. A second seizure followed, again lasting seconds, after which no respiratory effort was made. Bag and mask ventilation was initiated. The pupils were dilated and

unresponsive to light. At this time the pulse was 130, blood pressure was 112/78. Intravenous phenytoin was administered. Acyclovir was added.

An urgent CT scan of the brain showed: "A large left-sided porencephalic cyst with a smaller right porencephalic cyst. Most of the supratentorial area is of CSF density and there is only minimal cortical tissue. There is abnormal high density material on the tentorium cerebelli and around the basal cisterns suggesting subarachnoid haemorrhage. The basal cisterns appear tight."

As there was no development of respiration, he was intubated and transferred to the intensive care unit at 23:00 hrs. On arrival in the ICU he was unresponsive with a Glasgow coma scale (GCS) of 3. Pupils were fixed and dilated. There was no seizure activity.

A neurosurgical opinion was sought based on the CT scans. The neurosurgeon reviewed the CT scans and felt that no surgical intervention was appropriate.

The neurological examination remained largely unchanged. On subsequent biochemistry, sodium level rose to 149 urea 5.9, creatinine 65, Calcium 2.58, pH of 7.45 with pCO2 of 23.8.

At 10:10 am on 9/5/05, a note documents that the patient is brainstem dead, with no corneal reflexes, no cough, no breathing, . He has reflexes to supra-orbital stimulation.

Serum sodium rose to 150. Urine output was not recorded due to a difficult catheterisation. A urinary catheter produced 80 mls urine at 16:15.

He was transferred to the Royal Belfast Hospital for Sick Children at 17:10.

Nursing notes at 5:30 record: "patient noted to have spasms". No description of these spasms is entered.

Further nursing notes at 6:30 pm record the family's ongoing concerns re: Conors spasms and intermittent rash.

Review of nursing notes from the intensive care unit indicates that potassium dropped to 1.9, which was treated with intravenous potassium. An elevated blood sugar was treated with an insulin infusion.

A note on 8/5/03, time not recorded, records: ? had a seizure?, but does not describe the event.

Royal Victoria Hospital Medical Records

These records include documentation of Conor's previous attendances at the hospital which document his severe spastic quadriplegic cerebral palsy and associated visual impairment. A letter from Dr Elaine Hicks dated 5/5/88 when Conor was aged 7 mths, records that his birth and perinatal history were unremarkable. At this time he had clear evidence of cerebral palsy and visual impairment, and his CT scan showed an extensive porencephalic cyst in the left hemisphere, with a similar, but smaller, cyst in the right frontal region. Dr Hicks felt that it was likely that he had a cerebrovascular accident antenatally.

Following transfer to the Royal Victoria Hospital, Belfast on 10/5/03, a neuroradiology note recorded "extensive diffuse oedema of most of the brain including midbrain and brainstem, loss of grey/white differentiation suggestive of infarction, cause not apparent but ischaemic changes would give this appearance.

The notes are out of sequence in the copies available to me.

Dr Elaine Hicks, consultant neurologist, wrote a note on 10/5/03 (begins on page 122) in which she reviewed the history, noting that he had been well until 10 days ago, then became unwell with poor appetite, malaise, pink throat, had been seen by his GP and symptomatically "sort of improved" until 1 week ago, the throat got worse, vomiting seen by GP again who treated him for an infected throat and ears, treated with penicillin. He improved for a day or too, then got worse, vomiting, and was changed to amoxil. He was not back to baseline, the eating improved, and vomited on and off. He became worse and was treated in Craigavon hospital for dehydration. (The remainder of this note appears to

continue on page 137). Per the grandmother's history she records that Conor had at least 12 major stiffening attacks that "sound like tonic seizures", and that his face became increasingly puffy and he became more lethargic prior to deterioration.

She notes Conor's previous medical history, and that he had been on alternative therapies including hyperbaric oxygen and diaphragmatic exercises.

Based on her examination, she recorded an impression that Conor had severe cerebral and brainstem dysfunction- possibly irreversible damage.

Numerous subsequent notes document his severely abnormal neurological examination. , with absent cranial nerve findings and occasional reflex movements in his lower limbs and abdomen.

He became dependent on inotropic support, with infusions of adrenaline to maintain his blood pressure. He developed diabetes insipidus, another indication of severe diffuse brain injury.

Numerous conversations with the family are documented, in which the patient's mother described her concerns that he had seizures which were untreated, and that he was treated with an excessive amount of fluids in Craigavon hospital.

Ultimately it was agreed with the family that withdrawal of medical support was appropriate. At 15:45 on 12/5/03, treatment was withdrawn and he was pronounced dead.

I have read the witness and expert reports supplied as above and will not reiterate each in detail.

The most important of these expert reports is that of Dr Brian Herron, consultant neuropathologist.
He notes that the main findings were:

1. Bilateral porencephalic cysts which were old lesions related to his diagnosis of cerebral palsy.
2. Brain swelling which was to such a degree that vital areas that control breathing and heart-rate were damaged leading to death.

He quotes extensively from the independent report of Dr Sumner, consultant anaesthetist. He notes the issue of the reported seizures, and feels it is beyond my specialty to comment on the nature of these seizures, their cause and their outcome, feeling this would be better addressed by a Consultant Paediatric Neurologist.

He is confident that the ultimate cause of death was cerebral oedema (brain swelling).

In the accompanying autopsy report, it is noted that the brain showed extensive oedema, with focal subarachnoid haemorrhage but no subdural or extradural haemorrhage. In his summary, he indicates that the acute changes to the brain were coning of cerebellar tonsils, cerebral oedema, cerebral perfusion failure, global tissure necrosis with an early inflammatory response. There was haemorrhage into the brain which may be secondary to reperfusion following a respiratory or cardiac arrest due to leakage of damaged vessels.

In the report of *Dr Sumner, consultant anaesthetist*, he reviews the fluid management. He records that the initial fluid bolus was large, but not excessive, and that Conor later became puffy and swollen. He notes that the hypernatremia (high serum sodium) occurred after the rerpiratory arrest, and thus could not have been a cause of this arrest.

He makes significant comments relevant to Conor's neurological status, which are important in the context of my report and I will quote them in entirety:

"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 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".

Statement of Joanna Mitchell, mother of Conor

In this statement, Ms Mitchell details Conor's previous neurological condition and various therapies tried. He had mild epilepsy, treated with Epilim., 600 mg 5 times per week, 400 mg 2 times per week. She reports that his previous seizures were "absences" with staring to one side. He was extremely intelligent, making progress with home teaching and facilitated communication. He did not have formal speech but his ability to communicate nonverbally was excellent. She outlines the details of his illness before coming to hospital. Over the three days before he attended hospital, he had increasing lethargy and frequent vomiting. He also had episodes of arching of his back as if in discomfort.

While in the A/E department in Craigavon hospital, Conor had an "untypical seizure" witnessed by a doctor and his grandmother. He had further "seizures". A nurse was called, who referred to these events as "spasms" rather than seizures.

On page 8 of her statement, the mother describes the "seizures" as follows: "Conor went completely stiff, corkscrewed his body sideways and pulled his fists tight up under the chin. Each seizure lasted two to three minutes. He was also making dreadful strangulated choking sounds and a brown liquid was coming from his mouth. A red and angry blotchy rash then appeared on his head, face, legs, tummy and arms during the next and following seizures, disappearing in between them. During the seizures, Conor's nose, eyelids and lips swelled up and became very red...In total, Conor suffered from some ten to twelve violent and unwholly untypical seizures between his admission around 1 pm and approximately 8 pm."

At around 8 pm he suffered his final seizure, turned blue and stopped breathing.

In the statement of Dr Smith, consultant paediatrician, he relates: "when I was called to see him he had recently had a sudden onset of tonic contraction with extensor posturing. This was followed by pallor, apnoea and hypotonia. When he was initially assessed it appeared that he was having a seizure but then failed to breathe adequately and his oxygen saturation and heart rate dropped. He was given bag and mask ventilation.....It then became apparent that he was having an intracerebral event."

In the statement of Ruth Bullas, staff nurse at Craigavon Hospital, she records: "His mother told me that she had noted him spasm on several occasions. I also remember seeing him spasm. It was not a prominent or violent spasm but more of a twitch. At first when I was working with him, I did not observe him spasm but as I worked with him more, I did see him spasm and asked for him to be seen by the SHO."

In the statement of Dr Andrew Murdock, paediatric registrar:
"At approximately 8:45 pm while being assessed by the paediatric registrar Conor suffered a generalised seizure lasting seconds. A second seizure closely followed again lasting seconds after which no respiratory effort was made by Conor. I was not present during the first seizure but present during the second seizure.".

In the statement of Mr Kerr, A/E consultant he records:
"I witnessed several jerks in the arm, which lasted for a brief time only. I told Conor's mother that I did not feel this was due to the cannula and might be atypical seizure activity. She said she did not think this was like a seizure. I said I thought this might be possible even thought he had not had previous symptoms like this. The jerking was brief and did not recur therefore I felt there was no need for treatment at this time.".

OPINION:
From the information I have received, a number of important questions arise:

1. What was the cause of Conor's illness and death?
2. Did his fluid management in any way affect his outcome?

3. What was the nature of the "spasms" as described above?
4. Would different medical management in Craigavon Area Hospital have improved his outcome?

Firstly, we do not definitively know the nature of Conor's illness. It is clear that he had a gradual prodrome of vomiting, was found to have some findings indicative of an upper respiratory infection, and gradually deteriorated subsequently, with clinical and radiological evidence of brain swelling, confirmed by autopsy.

It is clear also that he was mildly dehydrated on arrival in hospital, and it was appropriate to administer intravenous fluids. I am not an expert on intravenous fluid management, and this has been addressed in some detail by Dr Sumner. As a neurologist, I know of only three mechanisms where the wrong fluid management can adversely affect the brain:

a) Administration of excessive amounts of dextrose without sodium, causing a drop in the serum sodium.
b) Inappropriately rapid correction of a low or high serum sodium.
c) Over-rapid correction of serum glucose in diabetic ketoacidosis

Based on the information we have, it is clear that neither of the above situations apply in this case. The serum sodium was normal during the early stage of his management. It became high after his catastrophic deterioration, presumably due to diabetes insipidus, a result of progressive brain injury. It is certainly possible that vigorous early administration of fluids could have resulted in some puffy facial swelling. However this is not likely to have affected his brain in any way, particularly as he had normal heart and kidney function.

Based on descriptions supplied by Conor's mother, Dr Hicks felt that it was likely that Conor was suffering from tonic seizures after arrival in Craigavon. Dr Sumner has reiterated this in his report and has further stated that these could have been a cause of severe brain injury, but allows that he is not a neurologist and is not expert in this matter.

Conor's previous epilepsy was reasonably well controlled. The episodes which his mother describes as "absences" were more likely brief complex partial seizures, associated with

gaze deviation to one side. Such a type of epilepsy would be common in a child with a previous history of prenatal brain injury and cerebral palsy. Conor's parents are clear that the events which he had that day were unlike any seizures he had previously experienced, and this was a cause of their concern.

Tonic seizures are most frequently seen in the context of a severe generalised epilepsy in children. They are associated with some specific epileptic syndromes. A typical tonic seizure is characterised by generalised stiffening of the body, including the trunk, upper and lower limbs. Typically these seizures will last 20 to 30 seconds or more and will not usually cause any catastrophic brain injury. In this case, Conor's mother describes episodes lasting 2-3 minutes. However parents will often have difficulty being precise about duration of episodes, particularly in the context of an acute emotionally disturbing situation. The medical and nursing notes are describing much briefer events.

In my experience of managing children with epilepsy, I have never seen tonic seizures arise de novo and in such a catastrophic manner in a child who previously had a very different form of epilepsy. Based on the descriptions available to me, I feel it is unlikely that these events were epileptic. It is more likely that these events were a form of decerebrate posturing, and were a manifestation of his progressive brain swelling causing pressure on his brainstem, which ultimately led to cessation of breathing following one of these events and death.

Dr Sumner has stated that these "seizures" could have been a cause of brain injury, leading to swelling and death. There has been extensive literature on the issue of brain damage due to epileptic seizures. There is little dispute that prolonged or repetitive epileptic seizures may cause brain injury.

Most of the human and animal data supporting this hypothesis would apply to generalised tonic-clonic seizures which are prolonged, lasting for 30 to 60 minutes or more. Such prolonged seizures may cause injury to nerve cells but would not cause brain swelling as was seen in this case.

The other mechanism for brain injury due to seizures would be from a prolonged lack of oxygen and blood flow to the brain during a very prolonged seizure. There is nothing in the records to indicate that any significant period of hypoxia occurred.

Thus it is my opinion that this child did not have epileptic seizures, tonic or otherwise, and that his episodes of "spasm" were not epileptic, but were symptoms of the progressive brain swelling which eventually lead to his death. I do not know what the cause of his brain swelling was. I have seen similar cases before in which no cause was found. Often an assumption is made that an encephalitis, or virus infection of the brain, is the cause of unexplained brain swelling. In this case, no evidence of encephalitis was found by the neuropathologist. I do not think that this completely rules out this diagnosis. There are a number of other diagnostic considerations, including Reyes syndrome, metabolic disorders, toxic exposure, but none of these seem likely.

The question arises whether earlier recognition of the nature of these events would have lead to earlier and different intervention and prevented the severe consequences which occurred in this case. Based on the medical records, it is clear that the medical staff did not witness events which were as severe or prolonged as the mother describes. I do not think that different management would have been justified based on the events they witnessed. If they had earlier identified these events as due to brain swelling, intubation, hyperventilation and administration of medication appropriate for increased intracranial pressure would have been appropriate. It is possible that death could have been prevented by such measures, but likely that severe brain damage would have ensued.

Please contact me if you have further questions about this report.

Yours sincerely,

Bryan Lynch, FRCPI, FAAP, DCH

Consultant Paediatric Neurologist

DECLARATION

I am a Consultant Paediatric Neurologist at The Children's University Hospital, Temple St, The Central Remedial Clinic, Clontarf and Beaumont Hospital in Dublin, a position I have held since 1995. I qualified in Medicine at University College Dublin of the National University of Ireland in 1982. I completed paediatric, neurology and clinical neurophysiology training in Ireland and at Washington University, St Louis and The Children's Hospital Boston. Prior to taking up my current appointment I held the position of Assistant Professor of Paediatrics and Neurology at The Children's Hospital, Pittsburgh, since August 1992. I am a Fellow of the Royal College of Physicians of Ireland, a Fellow of the American Academy of Pediatrics and hold American Board Certification in Child Neurology and Clinical Neurophysiology.

I declare that:

1. I understand that my primary duty in furnishing written reports and giving evidence is to assist the court and that this takes priority over any duties which I may owe to the part or parties by whom I have been engaged or by whom I have been paid or am liable to be paid. I confirm that I have complied and will continue to comply with this duty.
2. I have endeavoured in my reports and in my opinions ot be accurate and to have covered all relevant issues concerning the matters stated, which I have been asked to address, and the opinions expressed represent my true and complete professional opinion.
3. I have endeavoured to include in my report those matters of which I have knowledge and of which I have been made aware which might adversely affect the validity of my opinion
4. I have indicated the sources of all information that I have used.
5. I have where possible formed an independent view on matters suggested to me by others including my instructing lawyers and their client; where I have relied upon information from others, including my instructing lawyers and their client, I have so disclosed in my report.
6. I will notify those instructing me immediately and confirm in writing if, for any reason, my existing report or opinion requires any correction or qualification.
7. I understand that:
    a. My report, subject to any corrections before swearing as to its correctness, will form the evidence which I will give under oath or affirmation
    b. I may be cross examined on my report by a cross-examiner assisted by an expert
    c. I am likely to be the subject of public adverse criticism by the judge if the court concludes that I have not taken reasonable care in trying to meet the standard set out above
8. I confirm that I have not entered into any arrangement whereby the amount of payment of my fees, charges or expenses is in any way dependent upon the outcome of this case.

Signed,

Bryan Lynch, FRCPI, FAAP, DCH
Consultant Paediatric Neurologist