CORONERS ACT (Northern Ireland), 1959

Deposition of Witness taken on the day of (month), (year), at inquest touching the death of **CONOR EDWARD JOHN MITCHELL**, before me MR J LECKEY LL.M Coroner for the District of GREATER BELFAST as follows to wit: -

The Deposition of DOCTOR JANICE BOTHWELL

of c/o Bostock House, Royal Group of Hospitals, Grosvenor Road, Belfast *(Address)*
who being sworn upon her oath, saith

I am a registered Paediatric Consultant. I initially qualified from Queens University, Belfast, in July 1992 and completed my higher specialist training in paediatrics in January 2003. I was appointed to my Consultant's post in April 2003. My qualifications are MB BCh BAO MRCP (UK) MRCPCH. Conor Mitchell was admitted to PICU in the Royal Belfast Hospital for Sick Children, under my care on the evening of Friday 9th May 2003 at 9 25 pm. On arrival to PICU Conor was fully ventilated and dependent on IV infusion of adrenaline to maintain his blood pressure. He had not received any muscle relaxants or sedatives. On clinical examination his pupils were fixed and dilated. No discernable cranial nerve function was observed. Peripheral reflexes were present with some ankle clonus. He had fixed flexion deformities at the right elbow and hips. Problems present included hypernatracmia, diabetes insipidus and brainstem dysfunction. Full ventilatory support, inotropic support and ongoing fluid management continued. Conor was given IV antibiotic/antiviral medication to cover bacterial/viral infections and DDAVP to help in the management of the diabetes insipidus. A copy of the brain CT scan from Craigavon was obtained and a further CT scan of brain taken in RBHSC and reported by Doctor I Rennie, Neuroradiology after comparsion with a scan taken in Craigavon. Doctor Hicks, Consultant Neurologist, was consulted and the opinion was that it was very unlikely that Conor would recover from this illness as the clinical signs were of severe and likely irreversible brain damage. Conor's brainstem dysfunction remained unchanged from Friday 9th May 2003 to Monday the

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12th May 2003. His physical condition deteriorated in that he was unable to maintain his temperature, blood pressure, respiration and fluid balance without increasing medical support. After long discussions with Conor's family, the decision to withdraw treatment was made. Treatment was withdrawn at 3 15 pm on Monday 12th May 2003. Life was pronounced extinct at 3 45 pm on Monday 12th May 2003. In view of the lack of diagnosis as to why Conor had developed brainstem dysfunction secondary to a probable hypoxic ischaemic event and family concerns over his care in another hospital, the Coroner's Office was contacted by myself. The family's concerns about the care in Craigavon related to fluid management and unrecognized seizures. The hypernatraemia level was 164 which in my opinion was very high. Normal range is 135-145. I felt the level was due to his brainstem function and not the fluids actually given. In my view the fluid management at Craigavon was appropriate. The brainstem function changed - it became abnormal - during his time in Craigavon. Glasgow coma scale observation would be critical and observations should be taken every 15/30 minutes. In my view at some time during Conor's time in Craigavon he should have been assessed by a paediatrician. That would not have required Conor to be admitted to a paediatric ward. Before anything could be done it would be necessary to know why Conor's brainstem had started to function abnormally. Regardless, if it had been recognized at Craigavon that that was happening, that in itself

TAKEN before me this 7th day of June (month), 2004 (year).

[signature] Coroner for the District of Greater Belfast

CORONERS ACT (Northern Ireland), 1959

Deposition of Witness taken on the day of 20 , at inquest touching the death of , before me Coroner for the District of as follows to wit:—

The Deposition of DR. JANICE BOTHWELL

of (Address)

who being sworn upon her oath, saith

would have required the fluid management prescribed to be reassessed. What happened would not be a common problem.
A sodium level of 164 is a retrievable situation. In formulating the cause of death I would put hypoxia at 1(b) tonic seizures at 1(c) and cerebral palsy at 1(d).
Mr. McKillop: From his arrival at RBHSC Conor's prognosis was hopeless. He was showing signs of acute brainstem dysfunction. The hypernatraemia was a reflection of his brain damage – the brainstem dysfunction. The management of hypernatraemia depends on the reason for it. A decreased level of consciousness or seizure would point to the need for regular Glasgow Coma Scale observations. This is a clinical guide. The GCS on admission would provide a base line for assessing subsequent observations. You would take the family's views into account in establishing the base line. A CT brain scan may be carried out if there was deemed to be a significant problem. Before a CT scan you would have to ensure that

P.T.O.

the patient was stable. Paediatricians are more used to children and a paediatric assessment at Craigavon would have been helpful.

Dr. Millar: I first saw Conor at about [illegible] p.m. on 9th May. I was relying on the Craigavon notes and the information from the family. When Conor was admitted to Craigavon he was drowsy ~~but rousable~~ & on admission to RBHSC he was moribund. You cannot ignore a history given by a parent. I was heavily influenced by what I was told by the family. If I had been involved with a child over a period another admission might be to the paediatric ward also. A tonic seizure would be reflected in oxygen levels. Clinically I do not rule out a viral background. The CT scan of Dr Rennie suggested a hypoxic event. It is a very difficult case to account for the cerebral oedema.

[signature]

TAKEN before me this 7th day of June 2004

[signature] Coroner for the District of Greater Belfast