CORONERS ACT (NORTHERN IRELAND) 1959

Deposition of Witness taken on Monday the 25th day of April 2006, at inquest touching the death of CLAIRE ROBERTS, before me Mr J L Leckey, Senior Coroner for Northern Ireland as follows to wit:-

The Deposition of Dr David Webb

Who is being sworn upon his oath, saith

I am a Consultant Paediatric Neurologist and at the request of Mr J L Leckey, Senior Coroner for Northern Ireland, I prepared a statement in relation to the circumstances surrounding the death of Claire Roberts.

I now produce my report marked Exhibit C5.

Paediatric SHO admission note very important. On evening of admission GCS 9. Oct 22nd – 12.30 p.m. Diazepam resulted in an improvement suggesting she had had a seizure. P9 – CFR sample is abnormal – white cell count indicative of infection. Ratio of red to white cells – meningitis. When Claire died I would have formulated the cause of death as 1(a) Cerebral oedema (b) Meningo encephalitis and SIADH and at II Status Epilepticus. In my view the SIADH arose from her infection. I believe the blood test giving the low sodium result should have been repeated. Probably it was taken by the SHO on duty.

TAKEN before me this 28th day of April 2006

J L Leckey Senior Coroner for Northern Ireland

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 DAVID WEBB

of *(Address)*

who being sworn upon his oath, saith

How a blood sample is taken varies, Dr may depend on how difficult it is to obtain a sample.

Mr. McCrea: The excess sodium in the ~~hypernatraemia~~ occurred in the context of meningitis. Fluids are necessary for hyponatraemia to have an effect. Claire did need intravenous fluids. The trigger for the SIADH was the meningitis. I feel the view that Claire's presentation was a demonstration of her underlying epilepsy provoked by a viral infection HSV and with which I would usually associate SIADH. She was a candidate for electrolyte imbalance. The blood ~~test~~ taken on admission showed low sodium. The fluctuating GCS would indicate that at that time hyponatraemia was not relevant. Hypotonic fluid could have led to hyponatraemia in Claire's situation. You cannot assume that the drop from 132 to 121 was linear. A 3rd sample showed 121 + a 4th - 129. The probability is that the 121 sample was correct. The 121 samples (time of result) were taken at 11.30 p.m. + 3 a.m. The drop could have been exponential. It could

have fallen gradually and then dipped. There would have been a falling level of consciousness. Eventually a patient could go into a coma. There is no fluctuation. There is a change in the GCS after 8 p.m which could be attributed to a falling sodium level. Initially I treated her for her non-convulsive status & then warning it is. Her condition on admission could not be accounted for by her sodium level. There had to be something else. A blood sample is likely to have shown a drop in sodium leading to the medical team treating her in a different manner. The fluid regime would have been changed. When the hyponatraemia was identified it was appropriately managed. If a blood test had been taken on the morning of the 22nd & shown low sodium her sodium levels would have been monitored thereafter. At 8 p.m. I was not informed of Claire's deterioration. I later assumed it was a manifestation of her epilepsy. I would have expected to have been informed. When I was informed she was brain dead. No member of my team was informed. I would have been available if we were providing on-call. At 8 p.m. there commenced a sustained deterioration & I would have been expected to have been alerted to that. If I had I probably would have ordered a blood test. Her earlier blood test should have been repeated.

Mr. Lavery: The urinary output rates are very significant. I think it is surprising

TAKEN before me this 28th day of April 2006

Coroner for the District of Belfast

CR - PSNI

096-010-067

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. DAVID WEBB

of *(Address)*

who being sworn upon h ~~is~~ oath, saith

she had urinary output, at all
regarding the rapidity with which sodium dropped. Her seizure activity was a consequence of her infection. The fact that she was immobile does not have a major impact on her fluid regulation in my opinion.

DWebb

Cross-Examination of Dr David Webb

I now produce my report marked Exhibit C5.
Paediatric SHO admission note very important. On evening of admission GCS9. October 22nd – 12.30 pm. Diazepam resulted in an improvement suggesting she had had a seizure. P9 CFR sample is abnormal – white cell count indicative of infection. Ratio of red to white cells – meningitis. When Claire died I would have formulated the cause of death as 1 (a) Cerebral Oedema, (b) Meningoencephalitis and SIADH and at II Status Epilepticus. In my view the SIADH arose from her infection. I believe the blood test giving the low sodium result should have been repeated. Probably it was taken by the SHO on duty. How a blood sample is taken varies. It may depend on how difficult it is to obtain a sample.

Mr McCrea: The excess secretion of the ADH hormone occurred in the context of meningitis. Fluids are necessary for hyponatraemia to have an effect, Claire did need intravenous fluids. The trigger for the SIADH was the meningitis. I took the view that Claire's presentation was a deterioration in her underlying epilepsy provoked by a viral infection not one with which I would usually associate SIADH. She was a candidate for electrolyte imbalance. The blood test taken on admission showed low sodium. The fluctuating GCS would indicate that at that time hyponatraemia was not relevant. Hypotonic fluid could have led to hyponatraemia in Claire's situation. You cannot assume that the drop from 132 to 121 was linear. A 3rd sample showed 121 and a 4th - 129. The probability is that the 121 sample was correct, the 121 samples were taken at 11.30pm (time of result) and 3 am. The drop could have been exponential. It could have fallen gradually and then dipped. There would have been a falling level of consciousness. Eventually a patient could go into a coma. There is no fluctuation. There is a change in the GCS after 8 pm, which could be attributed to a falling sodium level. Initially I treated her for her non – convulsive status and then meningitis. Her condition on admission could not be accounted for by her sodium level. There had to be something else. A blood sample is likely to have shown a drop in sodium leading to the medical team treating her in a different manner. The fluid regime would have been changed. When the hyponatraemia was identified it was appropriately managed. If a blood test had been taken on the morning of the 22nd and shown low sodium her sodium levels would have been monitored thereafter. At 8pm I was not informed of Claire's deterioration. I later assumed it was a manifestation of her epilepsy. I would have expected to have been informed. When I was informed she was brain dead no member of my team were informed. I would have been available as we were providing on-call. At ??? pm there commenced a sustained deterioration. I would have expected to be alerted to that. If I had I probably would have ordered a blood test. Her earlier blood test should have been repeated.

Mr Lavery: The urinary output notes are very significant. I think it is surprising she had urinary output at all regarding the rapidity with which sodium dropped. Her seizure activity was a consequence of her infection. The fact that she was immobile does not have a major impact on her fluid requirement in my opinion.

D W M Webb