

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 (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, 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 + a 4th - 129. The probability is that the 121 sample was correct. The 121 samples were taken at 11.30 p.m. ^(time of result) + 3 a.m. The drop could have been exponential. It could

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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 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 & shown low sodium her sodium levels would have been monitored thereafter. At 8 p.m. I was not informed of Clive'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 or we were providing on-call. At 2 p.m. she 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 notes are very significant. I think it is surprising

TAKEN before me this

28th day of April 2006

Coroner for the District of Spoke

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091-008-037

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

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

David Webb

STATEMENT OF WITNESS

C5

STATEMENT OF: DAVID WEBB, CONSULTANT PAEDIATRIC NEUROLOGIST
Name Rank

AGE OF WITNESS (if over 21 enter "over 21"): OVER 21

NOT SIGNED IN POLICE OFFICER'S PRESENCE

I declare that this statement consisting of _____ pages, each signed by me is true to the best of my knowledge and belief and I make it knowing that, if it is tendered in evidence at a preliminary enquiry or at the trial of any person, I shall be liable to prosecution if I have wilfully stated in it anything which I know to be false or do not believe to be true.

Dated this _____ day of _____

SIGNATURE OF MEMBER by whom statement was recorded or received

SIGNATURE OF WITNESS



Re: Claire Roberts (deceased) DOB: 10/01/87


I am a registered medical practitioner and my specialist field is Paediatric Neurology. I am a Fellow of the Royal College of Paediatrics and Child Health in London and a Fellow of the Royal College of Physicians of Ireland. My medical training was in Ireland, England and Canada. I have a doctorate in medicine by thesis and have published over 25 peer reviewed papers. I have been a Consultant Paediatric Neurologist in Our Lady's Hospital for Sick Children and the National Children's Hospital in Tallaght since 1997. Prior to this appointment I was a Consultant Paediatric Neurologist at the Royal Belfast Hospital for Sick Children in Belfast for two years.

Contact with the deceased

I saw Claire Roberts in consultation during her inpatient stay at The Royal Belfast Hospital for Sick Children, at the request of Dr Sands who was Paediatric Registrar to the admitting Consultant Paediatrician, Dr Heather Steen. I saw and examined Claire twice on October 22nd 1996, the day prior to her death. I believe I discussed the consultation with a member from Dr Steen's team before seeing Claire and during my consultations I would have been accompanied by members of the ward nursing staff. On the first occasion I believe Claire's grandmother was present and on the second I believe I spoke to Claire's mother but this may have been by phone. For the purpose of this statement I have reviewed photocopies of the medical and nursing notes relating to Claire's admission.

Background History

Claire Roberts had a history of epileptic seizures that went back to the age of 6 months. At that time she had two episodes felt to have been seizures in the absence of fever. One of these lasted 2 minutes and the other about 10 minutes. Following discharge from hospital at that time she had a further 3 convulsions over a 3 day period with eye rolling, generalised jerking, harsh cry and snorting, each attack lasting about 3 minutes. She later had 6 seizures in one day, some of which were associated with cyanosis. On another occasion she required admission to hospital with a prolonged seizure and received Diazepam and Phenytoin intravenously (anti-convulsant medications used acutely to stop seizures).

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TO BE COMPLETED WHEN THE STATEMENT HAS BEEN WRITTEN

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STATEMENT CONTINUATION PAGE

STATEMENT OF: DAVID WEBB CONTINUATION PAGE NO: 1

In August 1987 aged 7 months she was started on Carbamazepine (an anti-convulsant). Investigations at that stage including cerebral ultrasound, electrolytes, calcium, magnesium, urine for amino acids and reducing substances, and electroencephalography (EEG) were all normal.

In September 1987 aged 8 months of age she was admitted to the Royal Belfast Hospital for Sick Children under Dr Hicks, (Consultant Paediatric Neurologist) for further evaluation and treatment. She was found to be developmentally delayed. She subsequently had episodes that were felt to be infantile spasms, although her EEG was not diagnostic of hypsarrhythmia (an EEG pattern associated with severe infantile epilepsy). Her medication was changed to Sodium Valproate (an anti-convulsant) and she was weaned off Carbamazepine. A CT brain scan at this stage was normal.

At follow-up she was noted to have attention difficulties, hyperactivity and moderate learning difficulties and later attended a special needs school. Her anti-convulsant therapy had been discontinued about 18 months prior to her admission to the Children's Hospital Belfast in October 1996.

Factual Chronology of Events -October 21st - 23rd 1996

Referral Details

Claire Roberts aged 9 was referred to the Royal Belfast Hospital for Sick Children (RBHSC) by her GP from the Castlereagh Medical Centre, Belfast on Monday, October 21st, 1996.

The GP referral letter read -

*'Nine year old girl with severe learning disability and past history of epilepsy. Fit free for three years. Weaned off Epilim (an anticonvulsant) eighteen' months ago. No speech since coming home. Very lethargic at school today. Vomited x 3. Speech slurred. On examination - pale. Pupils reacting, does not like light. No neck stiffness. Tone increased. Right side plantar reflex up-going, left side plantar reflex down-going. Ear nose and throat - nothing abnormal detected. Chest- clear.
Further fit? or underlying infection?'*

Claire was seen in the Accident and Emergency Department of RBHSC at 7.15pm on October 21st 1996. She was assessed by Nurse EA Jackson and then by a doctor whose name I cannot decipher.

The Accident & Emergency Nursing Assessment Note read-

History of being off form and lethargy. Seizure? Apyrexia (No fever). Pale and drowsy. Temperature 36.9, Respiratory rate 24, Heart rate 96. Seen by medical registrar admit to Allen Ward at 20.45 on October 21st 1996.

The Accident & Emergency Medical note read -

'Nine year old girl. Past history of learning difficulties. Epilepsy - no fits for 3 years. Off anti-epileptic medication. Vomiting today (non-bilious), since this evening. No

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STATEMENT OF: DAVID WEBB CONTINUATION PAGE NO: 2

diarrhoea, cough or, pyrexia. Speech very slurred, hardly speaking. On examination - drowsy, tired, no fever, no enlarged lymph nodes. Pupils equal and reactive to light. No neck stiffness. Ears normal. Heart sounds normal with no murmurs. Pharynx - unable to examine. Abdomen soft and non-tender, no masses and bowel sounds present. Lungs - air entry good, no added sounds. Plantar reflexes down-going right and left. No apparent limb weakness. Limb tone increased. Reflexes brisker on left than right. Plan - admit.

Primary diagnosis - encephalitis ?'

Inpatient Care - Allen Ward

If concerned about a child the accident and emergency nursing and medical staff would have contacted the Paediatric Medical team on call. The child would then be seen first by the Paediatric Medical Senior House Officer (SHO) on call that evening who appears to have been Dr O Hare. Following a clinical assessment Dr O Hare would have discussed Claire's case with the Paediatric Medical Registrar who would then decide whether Claire required admission to hospital. The nursing note in Accident and Emergency would indicate that Claire was seen by the Paediatric Medical Registrar on duty who made a decision to admit her to Allen Ward (a Paediatric Medical Ward). The ensuing notes are then In-Patient medical and nursing notes and begin with Dr O Hare's admission note at 20.00 on October 21st 1996.

October 21st - 8pm

The Paediatric SHO admission note read -

'Nine year old girl admitted by Accident & Emergency. Vomiting at 3pm and every hour since. Speech slurred and drowsy. Off form yesterday. Loose motions 3 days ago. Previous history of severe learning difficulties. Seizures from 6 months to one year of age controlled by Sodium Valproate (Epilim). Aged 4 had one further seizure. Anti-convulsant therapy gradually weaned. Development - can speak in sentences. Vision and hearing normal. Scribbling, feeds herself with supervision, cannot dress herself. Walks up and down steps, favours the left side of her body. Attending special school in Dundonald. Under care of Dr Gaston and recently tried Ritalin (a stimulant medication used to improve concentration). Side-effects dry mouth and agitation. Medication - nil and allergies - nil. Family history - two younger brothers.

On examination - temperature 37°C, heart rate 80/min,, chest clear, abdomen soft, non-tender and no masses. Central Nervous System - fundoscopy (examination of the back of the eye) normal. Optic discs not blurred. Pupils equal and reacting to light. Cranial nerves normal. Sitting up and staring vacantly. Ataxic (unsteady)? Power not assessed. Tone in upper limbs - cog-wheel rigidity on the right, increased on the left. In the lower limbs tone increased on both sides. Reflexes brisk bilaterally - right more than left. Plantar reflex responses down-going. Clonus present bilaterally. Not responding to parents' voice. Intermittently responding to deep pain.

Impression - viral illness.

Investigation - full blood count, Urea & Electrolytes, chest X-ray, viral titres, lumbar puncture. Management - intravenous fluids. Intravenous Diazepam (an anti-convulsant). Seizure activity? Reassess after fluids.'

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STATEMENT OF: DAVID WEBB CONTINUATION PAGE NO: 3**October 21st - 12 midnight**Further Paediatric SHO notes -

Dr O Hare appears to have reassessed Claire at 12 midnight on October 21st 1996 and wrote

'Slightly more responsive, no meningism, observe and reassess in the morning.

The next entry is signed by a different doctor whose name I cannot decipher. This person was also a Senior House Officer. This entry documents the result of a blood test and reads -

Sodium 132 (reduced), potassium 3.8, urea 4.5, glucose 6.6, creatinine 36, chloride 96, haemoglobin 10.4, PCV 31, white cell count 16.6 (increased), platelets 422,000.'

Overnight Claire would have been observed by the Allen Ward Nursing Staff. The notes relating to their observations are signed by Staff Nurse G McRandall.

October 21st - 10 pm - October 22nd - 7 amThe initial ward nursing notes read -

10 pm: - 9 year old girl with mental handicap and severe learning difficulties. Admitted via casualty with history of vomiting this afternoon, slurred speech, drowsiness pallor. Seizure?

On admission to ward, child pale and lethargic. Apyrexia (no fever). Observations within normal limits. Bloods taken. Intravenous fluids - normal saline commenced at 64 mls/hour. Two small bile-stained vomits following admission to ward. Seen by Registrar - to be reviewed following blood results and erection of intravenous fluids.

7am: - Slept well. Much more alert and brighter this morning. One further bile stained vomit. Intravenous fluids continued as listed. No oral fluids taken. Apyrexia. Observations satisfactory.

Claire was seen on ward rounds on October 22nd 1996 by Dr Sands, Paediatric Medical Registrar to Dr Steen. This note is not timed but on the basis that it is referred to as a "ward round" note, it is likely to relate to contact the morning after admission.

October 22nd - morningPaediatric Registrar (Dr Sands) Ward Round Medical Note read -

Admitted. Viral illness?

'Usually very active. Has not spoken to parents as per normal. Retching not vomiting. Vagueness/Vacant. No seizure activity observed. Attends Dr Gaston at the Ulster Hospital Dundonald. At 6 months old had seizures and was investigated for this, nothing found. Urea and electrolytes - sodium 132. Full blood count - white cells 16.4 (increased). Glucose 6.6. Apyrexia. On intravenous fluids. Pale colour, little response compared to normal. Pupils sluggish to light. Ears, throat - difficult to fully see. Difficult to see fundi. Bilateral long tract signs.

Impression - non fitting status. Plan - rectal Diazepam. Dr Webb to review.

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STATEMENT OF: DAVID WEBB CONTINUATION PAGE NO: 4**October 22nd - early morning (8 am - 2pm)**Paediatric Nursing notes read -

*Slept for periods during the early morning - bright when awake. No vocalisation but arms active. Late morning Claire became lethargic and vacant. Parents concerned as Claire is usually very active. Seen by Dr Sands - **status epilepticus (non-convulsant)***

Rectal diazepam 5mg given per rectum and commenced on neurological observations hourly. Pupils equal and reacting to light. Blood pressure 120/76 mm Hg, pulse rate 88, respiratory rate 28, temperature 37oC

The next medical note entry is dated October 22nd 1996 at 4pm and is written by myself. It would appear from my note that Claire's parents were not present and I obtained history from her grandmother. Although the note is timed 4pm it would seem more likely from the ensuing medical and nursing notes that this entry was actually written at around 2pm.

October 22nd - 2pmPaediatric Neurology Consultant (Dr Webb) 1st note read -

'Nine year old girl with known learning difficulties. Parents not available. Grandmother provided the history. Vomiting and listless yesterday evening followed by prolonged period of poor responsiveness. On no anticonvulsant medication. Note - appears to have improved following rectal Diazepam 5mg at 12:30.

O/E afebrile, no meningism, pale. Rousable - eye opening to voice, not vocalising, withdrawing from painful stimulus. Reduced movements on the right side. Anti-gravity movement present in all four limbs. Mildly increased tone in both arms. Reflexes symmetrically brisk. Clonus sustained at both ankles and toes up-going. Sits up, eyes open and looks vacantly. Not obeying commands. Pupils equal and reactive to light 5mm. Optic discs pale but no papilloedema. Facial, palatal and tongue movements appear normal.

Impression - I don't have a clear picture of the prodrome and yesterday's episodes. Her motor findings today are probably long-standing but this needs to be checked with the clinical notes. The picture is of acute encephalopathy, most probably post-ictal in nature. I note the normal biochemistry profile.

Management - suggest starting iv Phenytoin 18mg/kg stat followed by 2.5mg/kg 12 hourly. She will need levels 6 hours after the loading dose. Hourly neurological observations. CT tomorrow if she does not wake up.

The next medical note written October 22nd at 2.30 pm documents the calculations made to prescribe intravenous phenytoin (an anti-convulsant) initially as a bolus dose and then a 12 hourly dose. It would appear from the notes that I reviewed Claire during the afternoon and because of concerns about ongoing seizure activity recommended the use of midazolam (an anti-convulsant).

The next note reads "*seen by Dr Webb, still in status*" and then goes on to document the calculations undertaken to prescribe midazolam as a bolus and then as a low dose infusion. Following this therapy I reviewed and examined Claire again and this contact is documented in the note timed 5pm.

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STATEMENT OF: DAVID WEBB CONTINUATION PAGE NO: 5**October 22nd - 5pm**Paediatric Neurology Consultant (Dr Webb) 2nd note read -

'Claire has had a loading dose of Phenytoin and a bolus of Midazolam. She continues to be largely unresponsive. She responds by flexing her left arm to deep supra-orbital pain and does have facial grimace but no vocalisation. She has intermittent mouthing and chewing movements. Background (history obtained from mother) - she had contact with her cousin on Saturday who had a gastro-intestinal upset. Claire had loose bowel motions on Sunday and was vomiting on Monday. She had some focal seizures on Monday with right sided stiffening. Management plan - cover with Cefotaxime (anti-biotic) and Acyclovir (anti-viral therapy) for 48 hours. I did not think that meningoencephalitis was likely (in the absence of fever and meningism). I suggested checking viral cultures for the possibility of enteroviral infection.. I recommended stool, urine, bloods and throat swab cultures. I suggested adding intravenous Sodium Valproate 20mg/kg iv bolus followed by an infusion of 10mg/kg iv over 12 hours.'

The nursing notes for the afternoon and early evening of October 22nd document on going care and administration of anticonvulsants that had been prescribed. They also include a chart documenting "Attacks Observed". This outlines the time duration of events and Claire's state after the event.

October 22nd - 2pm - 8pmPaediatric Nursing notes read -

"Seen by Dr Webb - to have iv Phenytoin. Parents not in attendance. Continues on hourly CNS observation. GCS 6-7. Stat dose intravenous phenytoin at 2.45pm - to have this BD (twice per day).

Seen by Dr Webb - still in status epilepticus. Given stat iv Midazolam at 3.25pm. Continuous infusion running at 2mls/hr Midazolam to be increased by 0.1ml up to 3mls/hr. Given stat dose of Sodium Valproate at 5.15pm. Very unresponsive, only to pain. Remains pale. Occasional episodes of teeth clenching. Commenced on intravenous Cefotaxime (anti-biotic) and Acyclovir. Parents in attendance. Fluids at 64 mls/hour.

Paediatric Nursing - "Record of Attacks Observed"

*3.25pm Strong seizure - duration 5 minutes - sleepy afterwards
4.30pm Teeth tightened slightly - few seconds - asleep
7.15pm Teeth clenched and groaned - 1 minute - asleep
9pm Episode of screaming and drawing up of arms. Pulse rate increased to 165/minute, Pupils large but reacting to light. Dr informed - 30 seconds - asleep*

The next medical entry documents further blood test results and an interpretation of these results. They are signed I think by Dr Stewart (Paediatric Medical Senior House Officer). The results were apparently discussed with the Paediatric Registrar but this person is not identified.

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STATEMENT OF: DAVID WEBB CONTINUATION PAGE NO: 6**October 22nd - 11.30pm**Paediatric Medical Senior House Officer note read -

'Sodium 121, potassium 3.3, urine 2.9, creatinine 33, Phenytoin level 23.4mg/l (10-20). Hyponatraemic ? fluid overload with low sodium fluids ? SLADH.

Impression - increased need for sodium content fluids.

Discussed with Registrar - decreased fluids to two thirds of previous value - 41mls per hour. Send urine for osmolality.'

The nursing notes for the period 9.30pm - 2.30am document further drug administrations. At 11pm there is reference to the blood results with a comment that due to these "Solution 18 with 20 mmols of potassium chloride was erected as ordered by the Registrar" and that the patient was to have "fluid restriction of 41mls/hour".

The next medical note documents a sudden deterioration in Claire's condition in the early hours of the morning on October 23rd, 1996. The note is unsigned but is probably written by the Paediatric Registrar on call that evening.

October 23rd - 3 amDoctor's Note -

'Called to see. Had been stable when suddenly she had a respiratory arrest and developed fixed dilated pupils. When I saw her she was Cheyne-Stoking (very abnormal breathing pattern suggesting brainstem dysfunction) and requiring oxygen via facial mask. Saturation with bagging in high 90's. Good volume pulse. I attempted to intubate - not successful. Anaesthetic colleague came and intubated orally with 6.5 tube. Transferred to PICU.'

Nursing Note -

"2.30am - Slight tremor of right hand noted lasting few seconds. Breathing became laboured and grunting. Respiratory rate 20 per minute. Claire stopped breathing. Doctor contacted immediately. Oxygen and suction given. Registrar attempted to pass ET tube but unsuccessful. Anaesthetist called and ET tube inserted. Transferred to Intensive Care Unit at 3.25am"

In Patient Care - Paediatric Intensive Care Unit

The next medical notes are written by Dr Heather Steen, Consultant Paediatrician and myself, following arrival in the Intensive Care on the morning of October 23rd.

October 23rd - 4amPaediatric Consultant (Dr Steen) note read -

"Nine and a half year old girl with learning difficulties admitted 32 hours ago with decreased level of consciousness. Seen by Dr Webb - acute encephalopathy ? aetiology. Covered with Acyclovir and Cefotaxime. Loaded with Phenytoin and Valproate added. 11pm Sodium 121. Fluids restricted to 2/3rd maintenance. Observations otherwise stable. Registrar asked to see because of respiratory difficulties. Cheyne-Stoke breathing.

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STATEMENT OF: DAVID WEBB CONTINUATION PAGE NO: 7

Intubated and transferred to ICU. At present intubated and ventilated. Pupils fixed and dilated. Bilateral papilloedema, left more than right. No response to painful stimuli. Blood pressure 90/65, heart rate 100bpm. Plan - Mannitol stat. Dopamine infusion. Urgent CT brain.'

Paediatric Neurology Consultant (Dr Webb) note read -

'SIADH, hyponatraemia, hypo-osmolarity and cerebral oedema. Coning following prolonged epileptic seizure. Pupils fixed and dilated following Mannitol diuresis. No eye movements. For CT scan.'

Claire would have been taken over to the Royal Victoria Hospital to have a CT scan performed. This is an adult Hospital on the same site as the Royal Children's Hospital, Belfast. The next note documents the result of this scan.

October 23rd - 5.30 am

CT Brain scan result

There is severe diffuse hemispheric Swelling with complete effacement of the basal cisterns. No focal abnormality is identified.'

At this point it was clear that Claire had sustained severe brain injury and was not going to survive. The next medical note is written by myself and documents the first brain-stem death evaluation.

October 23rd - 6 am

Paediatric Neurology Consultant (Dr Webb) note read -

"Brain stem death criteria evaluation. Pupils 8-9mm unresponsive. Dolls eye movements. Corneal (responses) absent. No gag response. Iced calorics 40mls to both ears - no reponse. No response (motor or autonomic) to deep supra-orbital pain. Apnoea test in progress. CT brain shows cerebral herniation. Under no sedating or paralysing medication. Claire fulfils the criteria for brain stem death. This evaluation should be repeated in 4-6 hours'

The next series of medical notes were written by Dr McKaigue, Consultant Paediatric Anaesthetist and summarise the emergency care. Claire was attended to on the ward by the Senior Registrar and Anaesthetics who performed life saving therapy including the passage of a tube into her trachea (windpipe) to facilitate artificial ventilation. This set of notes describes this intervention and the ensuing medical care leading up to withdrawal of therapy.

October 23rd - 7.10 am

Paediatric Consultant Anaesthetis (Dr McKaigue) - note read

'9 year old girl admitted to the Paediatric Intensive Care Unit (PICU) from Allen Ward. Suffered a respiratory arrest and was initially bagged and intubated, performed by Dr Clarke (SpR in Anaesthetics) on the ward. At the time of intubation, vomitus was noted in

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STATEMENT OF: DAVID WEBB CONTINUATION PAGE NO: 8

the oropharynx (back of the mouth). Liquid material, no solid material. Following intubation (passage of a plastic tube into the windpipe) trachea was sucked out and a small amount of watery material was aspirated. The oral endotracheal tube (ET) was then changed to a nasal ET tube in the PICU.

Initially admitted to hospital with decreased level of consciousness with the clinical picture of acute encephalopathy. Status epilepticus subsequently developed requiring Phenytoin, Valproate and Midazolam. Serum sodium also noted to be low, presumably on the basis of SIADH. In PICU hyperventilated and given Mannitol 0.5g/kg. Pupils fixed and dilated. Blood pressure 95 systolic. Peripheral dopamine infusion commenced. Arterial line right dorsalis pedis and right internal jugular triple lumen line.

Then transferred for CT scan. Transfer uneventful. CT brain shows severe cerebral oedema. One set of brain stem tests performed by Dr Webb and Dr Steen. Serum sodium also checked at the time (133). Serum pH 7.13. Plan: maintain circulatory support as Claire was a potential organ donor. Laboratory sample at time of brain stem death showed a sodium of 129.'

Dr Mc Kaigue goes on to describe the planned medical care including circulatory support and fluid management. He also documents conversations between Dr. Steen myself and Claire's parents. There is then a note referring to the diagnosis of brain stem death.

October 23rd - 6.25pm

Paediatric Consultant (Dr Steen) - note read

"Diagnosis of brain death protocol completed. No spontaneous respiratory effort despite a CO₂ of 70mm/Hg. Discussed with parents – agreed that ventilation should be withdrawn and consent for limited PM given"

October 23rd - 6.45pm

Paediatric Consultant Anaesthetis (Dr McKaigue) - note read

'Ventilation discontinued at 18:45, death certificate issued.'

Autopsy Report

Claire had a limited autopsy of the brain only. The findings are reported by the Department of Neuropathology. I have outlined the summary findings below.

Neuropathology Report

"The features were those of cerebral oedema with neuronal migration defect and low grade sub-acute meningoencephalitis. No other discrete lesion has been identified to explain epileptic seizures. The reaction in the meninges and cortex is suggestive of a viral aetiology, though some viral studies were negative during life and on post mortem CSF. With the clinical history of diarrhoea and vomiting, this is a possibility though a metabolic cause cannot be entirely excluded. As this was a brain only autopsy, it is not possible to comment on other systemic pathology in the general organs. No other structural lesion in the brain, like corpus callosal or other malformations were identified.'

SIGNATURE OF STATEMENT MAI

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STATEMENT OF: DAVID WEBB CONTINUATION PAGE NO: 9**Review of Investigations**Blood results

	<u>October 21</u>	<u>22nd</u>	<u>23rd</u>
Sodium	132	122	139
Potassium	3.8	3.3	3.0
Chloride	96		103
Urea	4.5	2.9	3.4
Creatinine	36	33	34
Glucose	6.6		

Blood culture - no growth

Viral Studies - IgM for mumps, measles, herpes simplex, herpes zoster and CMV all negative

Serology for adenovirus, Q fever, PLGV, mycoplasma, pneumonia, influenza A and influenza B all negative.

Cerebrospinal Fluid Results (probably taken after death)

CSF appearance – blood stained, supernatant, straw coloured

Protein – 95g/L (raised)

Red cells – 300,000 (raised)

White cells 4000 (raised)

Ratio Red/white cells – 75:1 (raised, mostly lymphocytes – this ratio suggests meningitis)

CSF culture – no growth at 48 hours

Review of Fluid balance and administration

The fluids prescribed for Claire Roberts are documented on the "Intravenous Fluid and Prescription Chart", and were No 18 Solution, No 18 Solution with added potassium and Normal Saline for administration of drugs. The fluid charts run 8am to 7am the following morning documenting 24 hour input and output. She was initially prescribed 64 ml/hour (maintenance fluid volume for her weight) or 1,536 ml/day. She received:

October 21st - 22nd (22.30 - 7 am)-	volume = 536 ml	60ml/hour
October 22nd (8am - 7 pm)	- volume = 769 ml	64ml/hour
October 22nd - 23 (8pm - 2am)	- volume = 491 ml	70ml/hour

The volume was greater than 64 ml/hour over the last 7 hours as there was additional fluid (normal saline - 190ml) given with administration of her medication (phenytoin, midazolam and acyclovir). Instructions to reduce the fluid intake to 41mls per hour were given at 11.40pm on 22nd October. Fluid received after 12 midnight on 22nd October amounted to 33ml (17ml/hour). Claire's fluid output during the period of observation were 7 small vomits, one moderate vomit and 4 urine evacuations, one of which was large. The volumes were not recorded.

Review of Neurological Observations

The Glasgow Coma Scale is a widely used tool to assess a patient's level of consciousness and was devised initially in 1974 for use in adult patients. In a patient with reduced conscious level a painful stimulus is applied to assess their response. Three specific responses are examined - the patient's best visual response, verbal response and motor response giving a total score out of 15.

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STATEMENT OF: DAVID WEBB CONTINUATION PAGE NO: 10

- Best Eye opening
 - Spontaneously (4)
 - To speech (3)
 - To pain (2)
 - No eye opening (1)

- Best Verbal response
 - Orientated (5)
 - Confused, conversant (4)
 - Inappropriate words (3)
 - Vocalisation (2)
 - None (1)

- Best Motor response
 - Obeys commands (6)
 - Localises pain (5)
 - Flexion withdrawal (4)
 - Abnormal flexion (3)
 - Abnormal extension (2)
 - No response (1)

The Scale was modified for use in very young children with the omission of one of the motor scores (flexion withdrawal) - giving a total score out of 14. Claire's responses were recorded hourly from 1pm on October 22nd 1996.

Neurological Observations - Modified Glasgow Coma Scale

<u>Time</u>	<u>Score</u>	<u>Response</u>
13.00	9	opening eyes to speech, not speaking, obeying commands
14.00	8	opening eyes to speech, not speaking, localising pain*
15.00	7	opening eyes to pain, not speaking, localising pain
16.00	6	not opening eyes, not speaking, localising pain
17.00	6	not opening eyes, not speaking, localising pain
18.00	7	not opening eyes, making sounds, localising pain
19.00	7	not opening eyes, making sounds, localising pain
20.00	8	not opening eyes, making sounds, obeying commands
21.00	6	not opening eyes, making sounds, flexing to pain
22.00	6	not opening eyes, making sounds, flexing to pain
23.00		not recorded
24.00	6	not opening eyes, making sounds, flexing to pain
1.00	6	not opening eyes, making sounds, flexing to pain
2.00	6	not opening eyes, making sounds, flexing to pain

* Not recorded by nurses but based on my observations.

When using the original Glasgow Coma Scale a score of 8 or less would be considered by most to reflect the onset of coma. The modified score used in Claire's assessment gives a reduced score by virtue of its construction and to facilitate interpretation I have outlined below Claire's scores if the original Glasgow Coma Scale had been used.

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Neurological Observations - Glasgow Coma Scale

<u>Time</u>	<u>Score</u>	<u>Response</u>
13.00	10	opening eyes to speech, not speaking, obeying commands
14.00	9	opening eyes to speech, not speaking, localising pain*
15.00	8	opening eyes to pain, not speaking, localising pain
16.00	7	not opening eyes, not speaking, localising pain
17.00	7	not opening eyes, not speaking, localising pain
18.00	8	not opening eyes, making sounds, localising pain
19.00	8	not opening eyes, making sounds, localising pain
20.00	9	not opening eyes, making sounds, obeying commands
21.00	7	not opening eyes, making sounds, flexing to pain
22.00	7	not opening eyes, making sounds, flexing to pain
23.00		not recorded
24.00	7	not opening eyes, making sounds, flexing to pain
1.00	7	not opening eyes, making sounds, flexing to pain
2.00	7	not opening eyes, making sounds, flexing to pain

There are two periods of change observed here. The first is seen between 1 and 3pm and may have been related to the administration of anti-convulsant therapy and in particular midazolam or to the observed convulsive seizure at 3.25 pm. This is supported by the subsequent improvement in observations again at 8pm. After 8pm there is a definite and sustained change in the Coma Scale.

Throughout this period observations were being made of Claire's pupil responses and these remained reactive to bright light and equal in size between both eyes. She also had hourly recording of heart rate, respiratory rate, temperature and blood pressure. Claire's heart rate ranged from 90 to 110 to 100/min (normal) up until and including the measurement at 2am. Her respiratory rate was 20-25/min and normal. Her temperature rose mildly at 8pm to 38 °C and stayed mildly elevated from that time. Blood pressure was normal (120/60-70mm) throughout this period as were blood oxygen saturations levels 96-99% (normal).

Commentary

Differential Diagnosis

This girl had a clear background history of learning difficulties and epilepsy. She was known to the paediatric services from early infancy although had not had recent contact with the children's hospital. She was not taking anti-convulsant medication at the time of presentation as this had been discontinued 18 months previously.

She was referred with a history of vomiting and lethargy. She had had contact with a child with a gastrointestinal upset on the Saturday and had loose bowel motions herself on the Sunday (the day prior to admission). She began vomiting on the Monday (the day of admission). It would appear very likely from this history that she had contacted an enteroviral infection and this would have accounted for her gastrointestinal symptoms. Her mildly elevated white cell count would also support this observation.

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What created concern, was her neurological status on admission to hospital. She was clearly more lethargic and less interactive than one would have expected from a gastrointestinal infection. The clinical observations of her were that she had slurred speech, was staring vacantly at times and was ataxic (unsteady). There was also some variation in her performance and on the morning following admission for example the nursing staff commented that she was "more alert and bright when awake". In addition the nursing and medical staff reported that she had improved after receiving a dose of Diazepam (an anti-convulsant) at midday, two hours before I first met her.

When I met Claire first on Tuesday October 22nd 1986 I noted that she was indeed poorly responsive but did sit up when spoken to, opened her eyes and appeared vacant. She was responding to pain. On examining her arms and legs I noted that she had some stiffness and that her reflexes were abnormal. It would appear that I did not have access to her previous hospital chart as I made the comment that these findings "needed to be checked with her clinical notes". It was certainly possible in a child with known learning difficulties and epilepsy that the findings in her limbs could have been long standing. The other possibility was that some of the findings at least were a post-ictal manifestation. I would specifically have checked her for evidence of raised intracranial pressure by examining the back of her eye with an ophthalmoscope for papilloedema (swelling of the optic nerve head). I documented that this was not present. I would routinely have checked her gag response. I would also have checked her blood pressure (from the nursing records) and pulse rate (myself).

I was uncertain after speaking to her Grandmother whether there had been definite seizure activity witnessed on the day of admission. However when I spoke to Claire's mother later on that afternoon I obtained a history of a definite seizure affecting Claire's right side the previous day and I was in no doubt that she had indeed had a convulsive seizure on Monday the day of admission

I believe my impression was that this girl who had an undoubted epileptic tendency, and had had a witnessed seizure on the day prior to admission, was having subtle non-convulsant seizure activity triggered by a recent inter current viral infection. Most children will have obvious convulsive seizures when their epilepsy is triggered but it is well recognised that some children will have more subtle activity that will present with vacant staring, slurred speech and unsteadiness. In this condition the child will appear "encephalopathic". That is to say they will appear confused and poorly alert and may have occasional more overt seizures. This condition is referred to as non-convulsant status epilepticus.

The other possible trigger for her non-convulsant status epilepticus was that the infection in her bowel had actually spread to involve her brain causing meningo-encephalitis (inflammation of the lining of the brain and the brain itself). Enteroviral infection (a common cause of gastroenteritis) is one of the commonest causes of childhood meningitis and encephalitis and is usually a mild and self limiting illness.

I considered this possibility less likely as Claire had not mounted a fever and did not have evidence on examination of photophobia (disliking bright lights) or meningism (a stiff neck - usually seen with meningitis). With the possibility of meningitis and encephalitis in mind I recommended covering her with anti-biotic and anti-viral therapy (Cefotaxime and Acyclovir).

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Other explanations for her presentation included the possibility that the recent exposure to a viral illness had triggered an immune mediated inflammation in Claire's brain (acute disseminated encephalomyelitis) which can be associated with altered consciousness, seizures and abnormalities in limb tone. And finally Claire Roberts could have had an undiagnosed inborn error of metabolism that might explain her learning difficulties, epilepsy and sudden deterioration. There are a large number of potential metabolic disorders that might produce this clinical picture and require specialist investigation for diagnosis.

I commented on Claire's initial biochemistry profile (blood results) as being normal. Clearly measurement of her sodium levels was less than the normal 135 mmol/l, but my thinking would have been that a Sodium value of 132mmol/L was likely to be due to her recent vomiting and diarrhoea and could not on its own have explained her current encephalopathy or seizures.

Anti-Convulsant Therapy

I recommended treating Claire with anti-convulsant therapy as I felt she was in this state of non-convulsant status epilepticus. I used intravenous phenytoin to begin with as this is usually a non-sedating anti-convulsant and provides an anti-convulsant effect for several hours. When she failed to respond to phenytoin I suggested midazolam next because while this has a sedating effect it is short acting and has been shown to be an effective anti-convulsant in resistant seizure activity. I suggested sodium valproate as a 3rd line agent as this has been shown to be effective in status epilepticus and had clearly benefited Claire in the past. If she required on-going therapy which seemed likely this would be a reasonable choice as she had already demonstrated that she had tolerated and had benefit from this drug. My plan had been to organise brain imaging and EEG (electroencephalography) the following morning.

Fluid Therapy

The management of Claire's fluid therapy is clearly an important aspect of her care. It would be routine for children who are admitted with altered consciousness not to be offered oral fluids and therefore to require intravenous fluid replacement. The prescribing of fluids for children admitted acutely to hospital under a General Paediatrician is dealt with by the Paediatric Medical Team on call and is supervised by the Paediatric Medical Registrar on that team. Since being appointed as a Consultant Paediatric Neurologist 10 years ago I cannot recall writing a prescription for intravenous fluids and during this period have never written a fluid prescription for another Consultants patient. I would therefore not have had input into the choice of fluids in Claire's case.

It would be routine for children on intravenous fluids to have their urea and electrolytes measured on a daily basis or more frequently if necessary to facilitate adjustments to the fluids. Blood testing in hospital is routinely undertaken first thing in the morning and I believe I erroneously understood the urea and electrolyte result reported on Claire to have been that morning's result. My entry in the notes referring to her urea and electrolyte results was effectively a memo to myself that they could not have explained her clinical state that day. I believe that if I had understood the results to have been from the previous evening I would have requested an urgent repeat sample. In fact Claire's bloods were not repeated the morning after admission and the next urea and electrolyte measurement was on a blood sample obtained at 9.30pm on October 22nd. This result returned to the ward at 11.30pm that evening.

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The Syndrome of Inappropriate Anti-Diuretic Hormone secretion.

Claire developed a sudden severe hyponatremia during her stay in hospital. This is most likely to have occurred because of inappropriate secretion of anti-diuretic hormone (SIADH). The SIADH syndrome was first described in 1957. The role of anti-diuretic hormone is to hold onto fluids that the body might otherwise excrete through the kidney. If this hormone is secreted inappropriately the body may retain excess fluid (water). This can have the effect of diluting the blood contents and in particular it's content of sodium. A diluted content of sodium in the blood stream can act as a trigger for fluid (water) to enter the brain tissue and cause brain oedema (swelling).

The syndrome of SIADH can complicate a number of clinical conditions. It has been described to occur with the use of several drugs, and in the context of malignancy and several lung and brain disorders. The most likely explanation in Claire's case was the presence of meningitis (inflammation of the lining of the brain). The treatment of this condition is to restrict fluid intake until the inappropriate secretion resolves. The indication to restrict fluids would be a low sodium value in the presence of a raised urine osmolarity (measure of urine concentration).

There is evidence that severe hyponatremia is a poor prognostic factor in childhood neurological disorders. In a study of 72 children with acute neurological disorders admitted to hospital 31/35 with mild hyponatremia recovered fully while 37/37 children with moderate or severe hyponatremia either had residual deficits or died (1).

Claire's hyponatremia led to her developing cerebral oedema (swelling) and then brain herniation. The swollen brain will herniate downwards resulting in brain stem compression and cardio-respiratory arrest.

Although I did not seek an Intensive Care placement for Claire before I left the hospital on the evening of October 22nd, I am not sure whether she would have met the criteria for admission to Paediatric Intensive Care as there was no problem with her airway or breathing at that point and no supportive signs of raised intra-cranial pressure such as papilloedema, hypertension or bradycardia.

Comment on Choice of Fluid Therapy

The basic principles for prescribing maintenance intravenous fluids in children were identified in a landmark paper by Holiday and Segar in 1957 describing a simple formula for determining the maintenance water needs and recommending the use of a hypotonic saline solution equivalent to 0.2% saline and 5% dextrose (the contents of Solution 18). It is true to say that there have been concerns raised in recent years about the potential for Solution 18 to aggravate hyponatremia in acutely ill children. However it is also true that Solution 18 continued to be widely used into the late 1990s as an intravenous fluid source for children in Canada, the UK and Ireland. The 9th Edition of the "**Handbook of Pediatrics**", from The Hospital for Sick Children Toronto in 1997 identifies "Dextrose 5% + Sodium Chloride 0.2%" as a "useful Sodium maintenance fluid" in children (2). The 5th Edition of "**Forfar and Arniel's Textbook of Pediatrics**" published in 1998 and widely used in Britain and Ireland lists Dextrose 5% and Sodium Chloride 0.2% as a suitable solution for fluid and electrolyte replacement in childhood (3). In an article published in 2003, making a case for the use of "isotonic saline" solutions to prevent hospital acquired hyponatremia in children the authors begin by stating that "the current standard of care" is to administer hypotonic saline in maintenance parenteral fluids" (4).

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